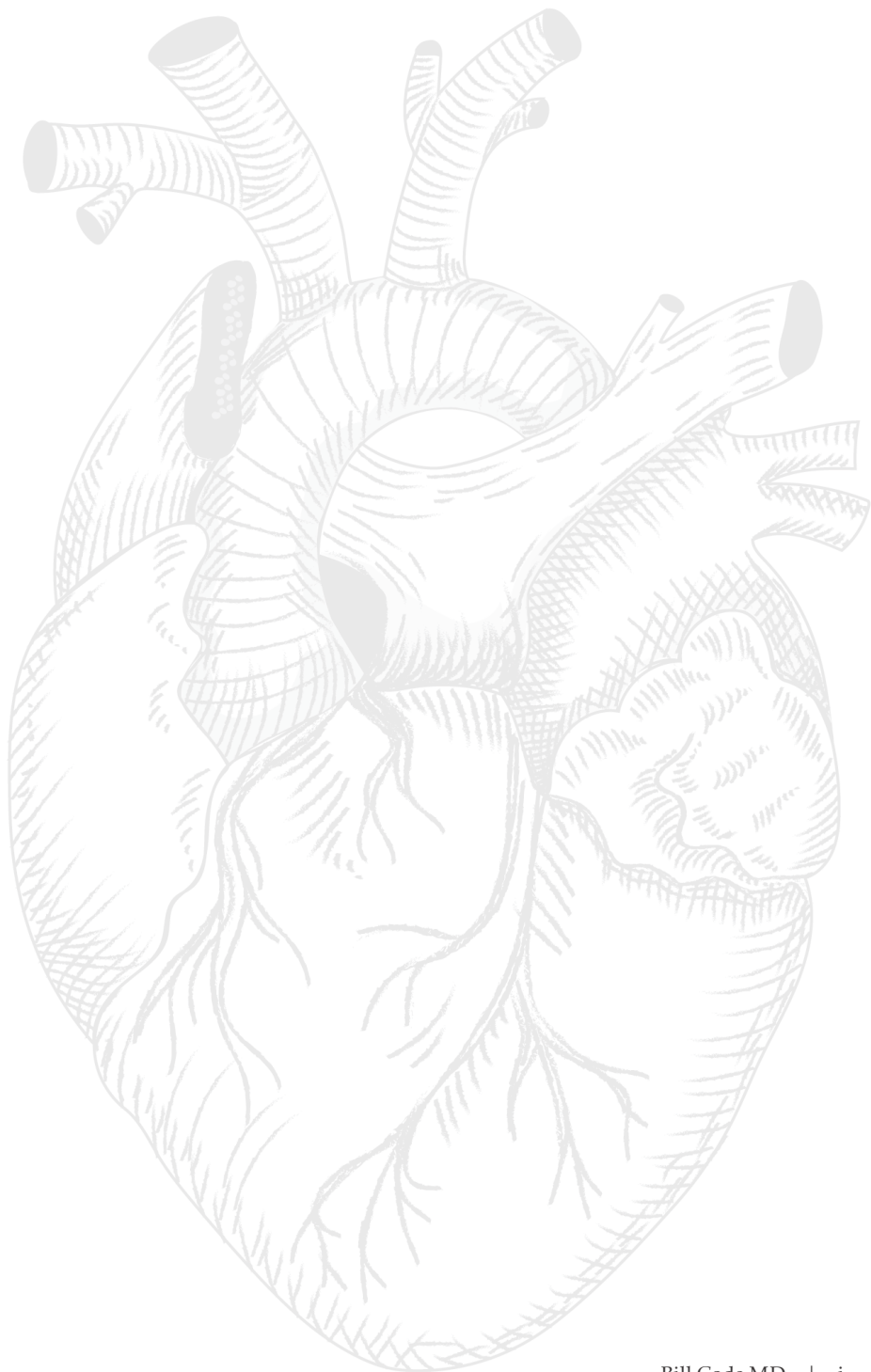




Solving the Heart Puzzle

An MD's Journey Through Oxygen, Omega-3s,
and the Science of Healing

— Bill Code MD —

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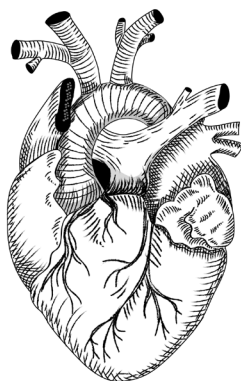
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CHAPTER 9

Oxygen: the God Molecule

Oxygen is, I believe, the God molecule. Of all the chemical gifts embedded in the universe, it is the one the creator — or nature, depending on your philosophy — chose as the primary engine of life for nearly every animal and plant on earth. That single fact should make us pay attention. And yet, in decades of medical practice, I watched patients and colleagues alike treat oxygen as a passive background gas rather than as the dynamic, life-sustaining force it truly is. This chapter is my attempt to correct that.

Let me start at the beginning — the very small beginning, inside each of your cells.

The Engine Room: Mitochondria, ATP, and the Oxygen Difference

Deep inside almost every cell in your body sits a tiny structure called the mitochondrion — often described as the cell's power plant. Its job is to convert the fuel you eat (carbohydrates and fats, themselves ultimately assembled from sunlight by plants) into a usable energy currency called ATP — adenosine triphosphate. Think of ATP as the rechargeable battery your body runs on. Every muscle contraction, every thought, every heartbeat costs ATP.

Here is where oxygen becomes heroic. When sufficient oxygen is present in the mitochondria, the cell can run a highly efficient process called oxidative phosphorylation. The yield: up to 34 molecules of ATP from a single glucose molecule. But strip that oxygen away — through blocked vessels, injury, inflammation, or poor lung function — and the cell is forced to fall back on an ancient, desperate fallback called fermentation. Fermentation produces only 4 ATP molecules from the same glucose. That is one-eighth the output. One-eighth.

For most tissues in the body, a temporary drop in energy production is uncomfortable but survivable. For the heart and brain, it is a crisis. These two organs live permanently at the very edge of their energy budget. They have almost no reserve. When oxygen supply falters, they falter — and they falter fast.

Why the Brain Feels it First

I want to focus on the brain first, because that is where most of us notice symptoms earliest: foggy thinking, poor memory, flat mood, and a grinding exhaustion that no amount of sleep seems to fix. Thinking is energetically expensive — brutally so. Anyone who has lived with chronic fatigue, for any reason, knows this in their bones. “I certainly do.”

When my multiple sclerosis peaked in the summer of 1996 — I was 43, newly diagnosed, and theoretically in the prime of my career as an anesthesiologist — getting out of bed in the morning felt like climbing a mountain without ropes. This was a man who had always loved mornings. The neurons responsible for planning, motivation, and movement were starved. Not dead, but starved. And starved neurons are quiet, idle or resting neurons.

The brain has three characteristics that make it uniquely vulnerable to oxygen shortfall, and uniquely rewarding when oxygen is restored.

First: The brain receives only about 2-3 percent of our resting cardiac output, yet it consumes 20 to 30 percent of the oxygen we inhale. That staggering mismatch is managed by an extraordinarily dense network of tiny blood vessels — especially microvessels smaller than the width of a human hair (around 50 microns, or 0.05 mm). Any insult to that microcirculation has an outsized effect.

Second: A neuron deprived of adequate oxygen does not immediately die — it simply goes quiet. It becomes a resting or idle neuron. Thinking becomes difficult, perhaps impossible. But the neuron is still there, still viable, waiting for oxygen providing energy to return. This is the key insight that has driven my clinical interest in oxygenation: what looks like permanent loss is often recoverable.

Third: Unlike almost every other organ, the brain can tolerate a temporary shutdown — coma, for example, is the brain's extreme energy-conservation mode. The heart cannot do this. The heart must beat continuously from birth to death. It cannot rest. As we explore ways to support both organs, we must never lose sight of that asymmetry.

Blood Flow: Why Even Small Changes Matter Enormously

Oxygen reaches your cells via the bloodstream. So before we talk about adding more oxygen, we need to understand blood flow — specifically, why even modest changes in vessel diameter produce dramatic changes in delivery.

This is where I need to introduce you to a beautiful and important piece of physics: Poiseuille's Law. The mathematician Jean Léonard Marie Poiseuille worked out in the nineteenth century that the flow of fluid through a tube is proportional to the fourth power of the tube's radius. Let that sink in for a moment. Not the radius. The fourth power of the radius. All anesthesiologists know this.

Here is what that means in practice: if a blood vessel's radius is reduced by half — say from 2 mm down to 1 mm, which is entirely possible when plaque builds up, or when a vessel goes into spasm — the blood flow through that vessel drops not by half, but to one-sixteenth of what it was. Conversely, if we can coax that vessel to dilate, to open up even modestly — through nutrition, medication, exercise, or supplemental oxygen — the gains are equally dramatic in the positive direction. Doubling the radius increases flow sixteen-fold. This is why every millimetre or even micron of the microcirculation or vessel health matters.

The Forgotten Frontier: Microcirculation

Until recently, the cardiovascular conversation has been dominated by the big vessels: the five major coronary arteries, two carotid arteries and the aorta. Cardiologists and surgeons have become extraordinarily skilled at stenting and bypassing these large vessels, and those interventions save lives every day. I have enormous respect for that work.

But the conversation has been incomplete. We now know that 40 to 60 percent of heart attacks occur not in the large coronary arteries but at the level of the microcirculation — the tiny vessels ranging from 5 to 100 microns in diameter. To put that in perspective: a red blood cell is approximately 7 microns wide and must squeeze through single-file. You cannot stent

a 10-micron vessel. You cannot bypass it. No surgeon's hands are that small.

This matters profoundly. It means that a significant portion of cardiovascular and neurological disease is occurring in territory that conventional procedures cannot reach. And it means that the path to healing these patients runs through a different kind of medicine — one focused on the fundamental health of the vessel wall, on reducing inflammation, on optimising blood viscosity and flow through nutrition, lifestyle, and yes, targeted oxygen therapy.

My goal in this book is to hand you practical tools to improve your own microcirculation. Most of these tools are within your reach today.

A Note About the Heart and Angina

Angina — that pressing, heavy chest discomfort that comes on with exertion and eases with rest — is the heart muscle's cry for more oxygen. It occurs when blood flow through the coronary arteries is insufficient to meet the heart's demand. What many people do not know is that the blood supply to the heart muscle itself arrives primarily during diastole, the resting phase between beats. The contraction phase (systole) is actually relatively constant in duration across a wide range of heart rates. But when the heart slows down, diastole (the resting phase) lengthens, and the coronary vessels have more time to fill and perfuse the muscle. This is why heart rate control is such an important part of managing coronary artery disease — a slower heart rate is literally a more nourishing one.

When two or more major vessels are significantly blocked (typically 80 percent or more), we now recognise that coronary artery bypass grafting offers better long-term outcomes than multiple stents. But as I just discussed, neither option touches the microcirculation to any degree. That requires a different approach entirely — and it is the approach this book is built around.

Your Home Oxygen Toolkit

Here is where things get practical — and where I hope you feel genuinely empowered.

My primary recommendation is a home oxygen concentrator, paired with a non-rebreather mask for higher-flow administration and nasal cannula for lower-flow, longer-duration use. A pulse oximeter — the small clip-on device for your fingertip — and a basic blood pressure cuff round out the toolkit. These four items together give you the ability to monitor and support both brain and heart recovery in real time, in your own home.

This setup is particularly valuable for any of what I call the five neurological microcirculation targets: traumatic brain injury (which is the same pathological process as concussion, whatever the cause), multiple sclerosis, stroke and shorter ones--transient ischaemic attacks (TIAs), peripheral neuropathy, and headache. I spoke on exactly these five conditions at the International Society of Orthomolecular Medicine in Toronto on October 4, 2025, and the evidence base for oxygen therapy in each of them continues to grow. It is this direct association between each of these and microcirculation that I have included these entities in a Solving the Heart Puzzle book. I want you to understand how each of these is intimately related.

Consider a concrete example. You hammer your thumb. Immediately you notice pain, redness, heat, and swelling — the four classic signs of inflammation. What is actually happening at the cellular level? The impact has bruised or broken tiny blood vessels in your thumb. Those damaged vessels can no longer carry their full load of blood. Perfusion to the local tissue drops. Oxygen delivery plummets. Within minutes, hypoxia triggers an inflammatory cascade — the body's emergency response to cellular energy crisis. The inflammation is not the injury; it is the reaction to oxygen starvation (hypoxia).

Now here is the key insight: if you applied supplemental oxygen via a non-rebreather mask within the first 60 to 90 minutes of that trauma, you could significantly blunt the inflammatory response, accelerate tissue repair, and reduce pain. I call this the Golden 60 to 90 Minutes — the critical window during which extra oxygen can redirect the tissue's trajectory from injury toward healing. Unfortunately, if you go to the emergency room with a bruised thumb, triage will rightly make you wait. By the time anyone sees you, that window of opportunity has passed. With a home oxygen concentrator in your toolkit, it need not.

Addressing the Three Fears About Supplemental Oxygen

Every time I discuss home oxygen therapy, I encounter resistance — not usually from patients, who are often eager and pragmatic, but from healthcare colleagues. The resistance clusters into three predictable fears. Let me address each one directly. These are addressed in more depth in the subsequent Myths chapter with great input by Professor Philip James of Dundee, Scotland.

Fear #1: Extra oxygen causes dangerous free radicals.

This concern has been around for decades and has been thoroughly studied. The worry is that breathing higher concentrations of oxygen will flood the body with reactive oxygen species (free radicals) that damage cells and DNA. The evidence does not support this. In fact, the opposite is true: the cell's own antioxidant systems — the enzymes and compounds that neutralise free radicals — function optimally when local oxygen levels are adequate. Hypoxia, not hyperoxia, impairs antioxidant production. When you restore normal or slightly supranormal oxygen levels to a tissue that has been starved, you are enabling it to protect itself, not attacking it.

Fear #2: Supplemental oxygen causes lung damage (oxygen toxicity).

Oxygen toxicity to the lungs is a real phenomenon — but it occurs at concentrations (typically above 60 percent oxygen) and durations (many continuous hours to days) far beyond what home oxygen therapy involves. A home concentrator delivers 90 to 95 percent oxygen at flow rates of 5 to 10 litres per minute, used intermittently for once or twice daily sessions of 30 to 60 minutes. Occasionally 4-6 hours is suggested as in acute concussion or stroke. At these parameters, lung toxicity is not a meaningful risk. The relevant literature on hyperbaric oxygen therapy — which involves much higher partial pressures of oxygen than a concentrator provides — confirms that short therapeutic exposures are safe for virtually all of patients. Context and dose matter enormously in medicine, and oxygen is no exception. If in doubt apply a pulse oximeter and have a caring individual sit with the person to reassure everyone.

Fear #3: Extra oxygen will suppress breathing in COPD patients — the “hypoxic drive” concern.

This fear is perhaps the most entrenched in clinical medicine, and also the most misunderstood. The concern is that patients with chronic obstructive pulmonary disease (COPD) rely on low oxygen levels to trigger their urge to breathe — and that giving them supplemental oxygen will remove that drive and cause them to stop breathing. The truth is that breath-to-breath respiratory drive in virtually all humans, including most COPD patients, is controlled primarily by carbon dioxide, not oxygen. If you hold your breath right now, it is rising carbon dioxide levels — detected by chemoreceptors — that eventually force you to exhale and then inhale. Oxygen is a much weaker driver of that reflex. While there is a small subpopulation of very severe, end-stage COPD patients in whom the hypoxic drive plays a larger role, careful titration of supplemental oxygen with pulse oximetry monitoring manages this risk safely. Withholding oxygen from an acutely hypoxic patient out of this fear is far more dangerous than the fear itself.

For further understanding I refer you to Chapter 11.

Clinician Note: The Hypoxia–Inflammation Axis

The link between hypoxia and inflammation is now well established. Hypoxia-inducible factor-1 α (HIF-1 α) is a transcription factor that responds within minutes to cellular oxygen deficit, upregulating pro-inflammatory cytokines (including IL-1 β , IL-6, and TNF- α) and promoting vasodilation and vascular permeability. This mechanism, originally evolved to recruit immune cells to hypoxic injury sites, becomes maladaptive in chronic low-grade tissue hypoxia — as seen in atherosclerosis and neurodegeneration including multiple sclerosis. Restoring local oxygen tension with supplemental oxygen therapy has been shown in multiple studies to modulate HIF-1 α activity and reduce the associated inflammatory burden. Understanding this axis reframes oxygen not merely as a supportive measure but as a mechanistic intervention.

Not Just the Brain and Heart

I have focused primarily on the heart and brain because they are the most oxygen-dependent organs, and the ones most likely to send you to the emergency room. But every organ system in the body — liver, kidney, gut, bone, peripheral nerves, immune cells — requires adequate oxygen for normal function, repair, and longevity. The mitochondria in a liver cell face the same physics as the mitochondria in a neuron: without oxygen, fermentation, 4 ATP, dysfunction. With oxygen, oxidative phosphorylation, 34 ATP, restoration.

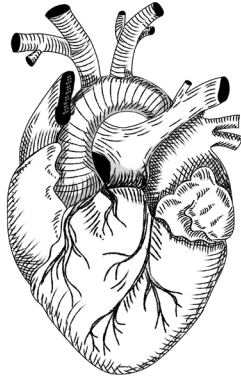
For 30 years, I have used this understanding as the foundation of my own health. I was 43 when I was diagnosed with MS. I am almost 73 now, recently retired, and I spend time in the garden, special times with grandchildren, and my evenings engaged with the community I love. I am not offering you a miracle. I am offering you the physics and the biology — and the practical tools that follow from them. The body wants to heal. Our job is to give it what it needs.

You need not wait for an enlightened specialist to come along. The tools are here. The evidence is here. Let us use them.

Action Steps

Practical takeaways from this chapter:

1. Assemble your home oxygen toolkit: an oxygen concentrator (5–10 L/min capacity), a non-rebreather mask, nasal cannula, a fingertip pulse oximeter, and a blood pressure cuff. These four items together provide real-time monitoring and extra oxygen delivery.
2. Remember the Golden 60 to 90 Minutes. For any acute injury, concussion, headache onset, or neurological event, early supplemental oxygen during this window can meaningfully shift the trajectory of recovery. Do not wait for a clinical setting to prepare your toolkit. It will be too late.
3. Target a resting oxygen saturation of 95–99 percent as measured by pulse oximeter. If you are consistently reading 90–93 percent at rest, discuss this with your physician — you are likely chronically under-oxygenated.
4. Prioritise microcirculation health through nutrition. Reduce processed sugars and seed oils, which drive endothelial inflammation. Increase Omega-3 fatty acids, polyphenol-rich vegetables, and adequate protein — all of which support vessel wall integrity and reduce blood viscosity. Add to this fish oil/olive oil blend daily.
5. Do not let Fear #1, #2, or #3 stop you from exploring therapeutic oxygen. Discuss the evidence in this chapter with your healthcare provider. If they are unfamiliar with the supporting literature, this book — and its references can serve as a starting point for that conversation.
6. If you have been told “there is nothing more we can do,” consider whether microcirculation health and oxygen optimisation have been systematically addressed. In most cases, they have not. That is not the end of the road. It is the beginning of a different one.



Acknowledgements

Just as it takes a village to raise a child, I have needed a village to write this book. I am grateful to everyone who have contributed to this end result. Inevitably, I will miss someone, and I sincerely apologize for that.

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Any errors you note in this book are my responsibility. Please forgive me and let me know for the next printing.