

2010

Ischemic Dormancy and Rehabilitation

A basis for integrated energetic intervention in ischemic pathology

Ischemic trauma creates reversible and irreversible symptoms resulting in both tissue dormancy and necrosis. PEMF protocols often evidence immediate durable neurological responses. Immediate PEMF responses suggest non-necrotic damage reversal happens almost immediately. The parallel tendencies for near complete recovery with prompt treatment, and significant recovery in much delayed intervention evidences reversible ischemic dormancy is a dominant factor in stroke pathology.



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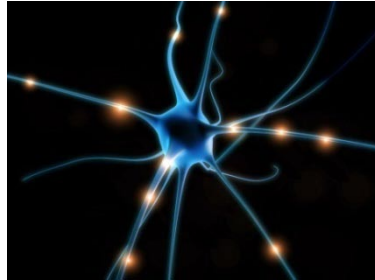
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Introduction

Ischemia occurs when blood flow is blocked. Flow blockage suffocates and starves cells downstream.

Sludge, usually resulting from energetic and chemical reductions zeta potential trigger clumps which clog blood flow.

Even though stroke is the most commonly recognized condition – ischemia happens everywhere. Any time blood flow to tissues is inhibited damage happens.



Ischemic damage is common, and often recoverable.

Damage is durable (permanent) when the lacks the resources to restore blood flow, oxygen and nutrients to the region. This occurs when multiple problems create collateral limits on healing

- Clumps clog bloods supplies the healing systems that enable recovery;
- Healing is disabled by other factors, toxicity, etc.

This document has three goals:

1. Describe the physiology of stroke as an actionable condition;
2. Recommend a therapy model;

Language

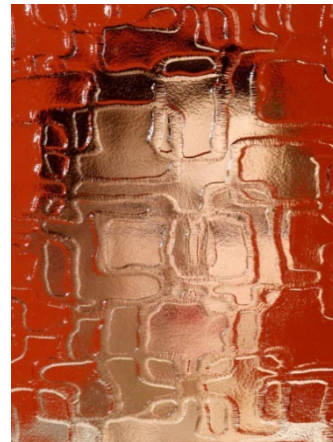
We tried use relatively simple language where we could. Otherwise, we used links to help you look up the definitions of the physiology terms.

Resources & Support

If you feel that the disease model present here may be applicable, we invite you to contact us through one of our websites:

- <http://www.dshedu.com>
- <http://www.wholehealthnetwork.com>
- <http://www.rejuvicell.com>

- Call 970 372 4274
- Email contact@wholehealthnetwork.com



Ischemia Basics

There are three main causes for ischemia:

1. [Vascular Occlusion](#) – A blockage clogs the blood supply;
2. [Endothelium Inflammation](#) – Narrowed [Capillary](#) network limit blood flow to a brain region. This is usually triggered by a toxic event or primary ischemic episode.
3. [Aneurysm](#) – The artery that carries blood failed.

There are three primary vulnerabilities which lead to ischemic incidents:

1. Loss of [electrostatic blood separation](#). [Blood sludge](#) causes blockage, which stops blood flow to downstream tissue;
2. Weakness in arteries. [Artery](#) degeneration weakens in the vessels that carry blood. It results from long-term depletion of nutrients/oxygen that maintain [blood vessel](#) tissue integrity, [chronic toxicity](#) in the blood, or both;
3. Tissue oxygen depletion. [Hypoxia](#) triggers [Capillary Inflammation](#) that creates a long-term limit blood flow to tissues. The inflammation persists until “reversal conditions are met” -- for more information see: [Oxygen Multistep Therapy – Chapter 1](#). The effect causes a temporary loss of blood flow to an area to become permanent, unless very specific reversal conditions occur.

Background

The [medical standard of care](#) for circulatory system dysfunction uses [anti-coagulants](#) to inhibit blood clotting.

This model disregards the core issue. Electrostatic potential governs particle dispersion in a fluid. Blood is a [colloidal](#) system. [Electrostatic](#) charge holds blood cells apart.

When this charge is lost, due to toxins, or electron donor depletion, particles clump together. In blood, these clumps clog the vascular system.

These circulating clumps are the dominant cause of occlusion.

Blood clumps which occur due to depletion the electrostatic charges which hold particles apart, is the dominant cause of ischemic episodes.

The following reference is of supreme importance because they describe the very real role of electrodynamics of fluids:

- [Thomas Riddick: Control of colloid stability through Zeta Potential](#)
- [TC McDaniel – Disease Reprive](#)
- [Book Excerpts online](#)

The alert reader will notice that the subject matter of these references is very different from the [blood clot process](#), which is the governing basis for post-ischemic event care.

Treating the wrong problem

[Medical Standard of Care](#) stroke care protocols do not address blood energetics.

They rely on inhibiting blood clotting under the curious belief that the dominant cause of occlusion is errant coagulation. In most cases, this is simply untrue:

- Clotting is normal cascade response which prevents bleeding from injury.
- Clumping or agglutination that results from loss of zeta potential is a disease condition. It is a result of toxic stress and/or nutrient depletion that cause electron deficiency in the blood which reduces [colloid stability](#) in the blood.

While inhibiting blood clotting may have some benefit, it's far better to target the cause of the problem – clumping from errant blood energetics.

Anti-coagulant agents that disrupt the clot mechanism don't contribute electrons that increase blood's

electrostatic potential – and usually contribute to further depletion.

As a result, therapy with pharmaceutical anti-coagulants presents a delicate and sometimes dangerous balance:

- Reduced clotting does reduce the tendency for clumps to become clots by inhibiting the Vitamin K clot mechanism, which often reduces the tendency for clumps to become clots;
- But they deplete Vitamin K which is an [antioxidant](#), and plays multiple other metabolic roles, and lead to other metabolic dysfunctions, which in turn increase adverse event risks;
- Anti-coagulants contribute to the “toxic load” and exacerbate toxin related dysfunctions and eventually reduce zeta potential;
- Interference with clotting increases the [hemorrhage](#) vulnerability, so internal bleeding is a serious concern with anti-coagulant medication.

The benefits are at best are limited, and generally come at the expense of hemorrhage vulnerability, and additional toxin load.

An antioxidant is an electron donor. An alert reader, who has studied [zeta potential](#), will recognize that electron depletion causes occlusion in the first place.

Rehabilitation and recovery are limited by the extent to which therapy resolves the core issue. If the primary problem is unresolved, healing doesn't happen.

Occlusion Triggers Aneurism

In most cases, an occlusion triggers an aneurysm. A downstream blockage dams blood flow. Extra pressure causes the weakened feed artery to blow out.

So while it may not seem obvious, avoiding occlusion, by maintaining blood zeta potential, is a critical step to avoiding catastrophic aneurysm.

The Irreversibility Concept

Most people that “stroke” symptoms are permanent when they utilize standard of care therapy.

This author asserts that this remains true only while the oxygen and nutrient supply to the affected tissue is not restored.

Ischemic Dormancy

Several anomalous recovery responses suggest that cells have a “survival mode” which enables them to remain alive for long periods of time after an ischemic trauma.

We refer to this survival mode as Ischemic Dormancy. The observed rate of recovery suggests that brain cell is much longer than widespread belief.

These cases suggest that ischemic symptoms are substantially reversible with early intervention and encouragingly reversible with delayed intervention.

This author asserts that the meager energetic requirements for cells in ischemic dormancy come from [anaerobic respiration](#).

While operating in anaerobic mode, cells have about 5% energy available, with 2 units of [ATP](#) per [glucose](#), compared to 40 during the [Oxidative Krebs Cycle](#).

While occlusions and inflammation limit or prevent blood flow, [glucose](#) can often reach the tissues.

This “backup” supply often enables cells to survive a long time.

Anomalous Recovery Explanation

Our hypothesis is that the anomalous rapid recoveries occurred because of restoration of normal cellular metabolism to dormant cells.

The care model, below, targets restoration of normal metabolism by multiple means.

Ischemic Dormancy Principle

Brain tissue from stroke dormancy appears to be the prevailing cause of debilitating stroke symptoms. As the condition matures dormant tissues die, and reversible damage becomes permanent.

The surprising success of hybrid interventions delayed up to ten years suggests cerebro-ischemic protective dormancy mechanism is durable significantly beyond documentation in previous medical literature.

The delay between ischemic event and therapy strongly affect both the response degree and rate, likely due to progressive cerebral necrosis. The transition from reversible to irreversible symptoms is much slower.

Ischemic dormancy is a survival response which enables long-term preservation brain tissue after a trauma event.

Stroke symptoms are the combined of dysfunction caused by ischemic dormancy and irreversible ischemic necrosis.

This paper explores the tendency for the body to preserve life-essential tissue, and the surprising reversibility of stroke symptoms even with late intervention using a combination of nutritional and energetic methods.

Clumps and Clogs

Zeta potential is the primary factor in ischemic event risk. Allopathic interpretation blames errant blood clotting for ischemic disorders. Colloidal stability is not considered a factor.

Zeta potential is a dominant factor because it reflects the dynamic balance that becomes fragile under stress and toxic load.

Surprise occlusion events, reflect a alignments in a matrix of cofactors that determine zeta potential or clumping in a fluid.

If the fluid is blood, then a clump is a clot. Clumps which clog vessels stop flow and trigger cellular

damage. When too many clumps clog too many vessels, unrecoverable damage happens.

Clumping incidence is driven by low energetic potential which reflects the same factors in other diseases.

Blowouts/Aneurysms

Ballooned arteries, [aneurysms](#), happen because the tiny vessels that feed arteries and bigger vessels clog and starve them.

Starvation weakens the tissue in the vessel. Starvation eventually weakens the collagen and muscle that give the artery strength. Finally, artery fails by creating a balloon or bursting.

Burst/aneurysm failure is caused by starvation of oxygen and food which prevent the cells in the artery from healing.

Tiny clumps that block the blood supply to the arteries are the cause of the arterial failure. So... Clogging the blood supply to the blood supply causes a different kind of failure, or aneurysm.

When these balloons burst, blood leaks out and the downstream tissue starves. Both effects can cause considerable, often lethal damage.

The hidden majority cause of aneurysm is that tiny clots that block the blood flow to the artery itself. When arterial structure fails, disaster happens.

These tiny clots reflect unresolved sludge which lasts long enough to undermine vascular integrity. Vascular degeneration happens everywhere.

Usually the burst event is triggered by a stress event, normally a downstream plug causes pressure to build up and the weak spot to burst.

Disease Cofactors

Vascular disease is chronic zeta potential deficiency. It is an energetic disease.

Heart disease is an energetic disorder – the blood lacks sufficient energetic and ionic potential to maintain fluidity and prevent clumps which cause a continuous process of micro-damage throughout the body.

Clumps that clog tiny vessels contributing to virtually every symptom associated with vascular disease.

Macular degeneration – is when the tiny vessels that feed the retina clog and try to heal without oxygen;

Vascular weakening – occurs when blood supply to the blood supply chokes;

Vascular Occlusion happens when clumps are so big they shut down a major blood supply;

Stroke – is when a rupture or occlusion happens in the brain;

And so on.

Vascular Lesions

We conveniently omitted atherosclerotic lesions which cause vascular narrowing.

Consider that even a narrowed artery requires a plug – without the plug to clog the narrowed channel disaster merely waits.

The issue of course, is why doesn't the lesion heal? Is it partly because the blood supply required to heal the lesion could be itself plugged?

Swelling and plaque accumulation which narrow the arteries, are injuries which cannot heal.

It seems annoying to point out that if the vascular system is so sludged up that the big pipes are about to clog -- that the small ones clogged long ago.

Micro-vessel clogs make it impossible to heal the lesions which are shutting down the big ones.

Persistent lesions evidence an absence of healing likely due to an absence of resources to do the healing.

As a result, plugged micro-vessels are guaranteed to be a factor in vascular degeneration.

The alert reader will recognize that if big arterial lesions heal normally and completely, then plaque would not accumulate – setting the stage for major occlusion later.

Vascular De/Regeneration

Vascular regeneration is a survival response – without it, life would be very short. Many miles of capillary, vessels and arteries are damaged every day as part of wear and tear of life.

Vascular systems regenerate.

- So the question becomes when and why do they stop regenerating?
- Why do certain symptom sets, called diseases always seem to come together?
- What do all these processes seem to have in common? Why are they different at all?
- Why do certain substances seem to help?
- Why do energetic therapies like PEMF always seem to produce such rapid improvements?
- Why are stroke victims who use Hyperbaric Therapy tend to recover more?
- Why is there such a strong correlation between heart disease risk and stroke risk?
- What is the relationship of toxins?
- What is the relationship of infectious agents?

Downhill Journey

A degenerate mess happens after the body loses the ability to keep up with the repair load.

When body can keep up with the damage, there's no real problem.

The critical balance is healing rate versus damage rate.

When damage exceeds healing, downhill happens.

Common downhill triggers are:

- building material deficiency;
- bug bloom;
- sludge slam from toxins;
- stress.

When downhill happens, it lasts forever or until it's fixed, whichever comes first.

Explanation of weak results

Medical science is nominally able to prevent and support recovery in ischemic syndromes.

Here are some principles:

- Clumping and clotting are very different ;
- Only clotting is managed, clumping is not;
- Dormancy preserves life challenged tissue;
- Dormancy and Necrosis, death, look alike;
- Tissues survive dormant for a long time.

Better results are possible and often rapid:

- Integrated energetic nutrient care seems to quickly restore dormant tissue;
- Toxin and nutrient flow are unmanaged;
- Event avoidance totally misses the major risk factor – clumping, which is effectively and safely managed with nutrients and energetics.

More Toxins Hurt

Causal factors resulting in ischemia tend to be lipo-suppressive and do not address toxin and pathogenic aspects of vascular degeneration.

Most drugs don't touch the pathogenic foundation or interfere with the mechanisms that pathogens use to damage the host.

Most pharmaceutical agents tend to interfere with healing by adding toxins to an already toxic metabolism. This additional damage means that most interventions make the problems worse.

Punch line

PEMF is an excellent tool in ischemic conditions for several reasons:

1. It immediately improves zeta potential which helps restore blood flow;
2. It opens the vascular system so more blood can flow;
3. It provides usable energy to dormant cells often restoring functions in dormant tissue.

These three performance aspects make PEMF a huge tool in the entire spectrum of ischemic conditions.

**PEMF supports ischemic tissues and vascular recovery
*at the same time.***

Primary hypothesis PEMF provides a life support for ischemic cells. It also aids collateral healing which reduces the tendency for long-term ischemia.

The tendency to produce immediate partial recovery from cognitive limits which accompany “stroke” suggests that PEMF has core value with all ischemic pathology.

Misdirection & Misinterpretation

While the social interpretation of disease is outside our scope, it may be useful to articulate reasons why this seemingly simple model has failed to emerge earlier.

Tendency not to recognize that PEMF exposure provides a backup cellular energetics which limit terminal ischemia.

Also a tendency to overlook oxygenation enhancement therapy:

- This protocol uses exploits Plasma oxygen transport in addition to improve cell oxygenation instead of RBC;
- Uses PEMF as a life support energy supply to curb final necrosis;
- Uses PEMF as a tool to catalyze vascular healing;
- Integrates functional detoxification which tends to limit the degree and rate of vascular recovery;
- Exploits the body's ability to cause ischemic cells enter a dormant state as an opportunity to restore blood flow.-

Cure (noun) versus Cure (verb)

A major challenge is medical tendency to interpret the word "cure" as a noun implying single cause, and hence a single act to restore health.

To cure (verb) is an act or process of health restoration, involving as many or as much intervention needed to get the job done.

The difference the noun and verb forms of the same word in different ears, inhibits the ability to see relationships, and coordinate intervention accordingly.

In other words, the notion that each disease has one cause and one cure is terribly misleading.

Conditions with multiple causes tend to defy cure (verb), because products that **cure (noun)** don't do enough to resolve conditions caused by a set of interrelated problems.

Stroke Care Model

There are several goals in stroke support:

1. **Cellular Life Support.** Keep as many cells alive as possible with energetic therapy PEMF to inhibit cell death;
2. **Optimize Blood Viscosity.** Manage Zeta Potential with biochemical agents including antioxidants and anionic surfactants to prevent clumps. Use ePad to deliver systemic electrons.
3. **Restore Brain Oxygen.** Oxygen Multistep Therapy (superior to Hyperbaric Therapy) to maximize systemic oxygen availability to the brain;
4. **Electrons to Brain.** Utilize energetic electron donors, ePads, to minimize inflammation and increase serum free-electron concentration to aid systemic and local zeta potential;
5. **Copious Antioxidants.** Provide large quantities of targeted antioxidants to minimize oxidative stress;
6. **Clean out the pipes.** Use fibrin enzymes to aid breakdown of clumps which interfere with blood flow.
7. **Neurological Detoxification.** Use Heavy Metals detoxification agents, phospholipids and nutrient detox agents to enable disposal of toxins that reduce zeta potential and interfere with neurological performance;
8. **Raw Materials.** Use targeted nutrients to support mobilization of stem cells to aid regeneration of damaged tissues.

Intervention Design

The diagram below pictorially represents the environment which set the stage for stroke.

1. Vascular degeneration driven by vascular disease risk factors
2. Blood sludge
3. Clot causes rupture or pressure spike causes rupture.
4. Clot shuts off blood flow to brain tissue.

