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[Hypotension](#) or low blood pressure is caused by loss of [vascular tone](#) and usually results from one or more of the following conditions:

- Insufficient [oxygen](#) to arterial smooth muscle to maintain tone
- Dysfunction of [brain area that controls Blood Pressure](#)
- Inhibition of [vagus nerve](#)
- Toxic [Shock](#) where one or more toxins disrupt tissue oxygen delivery
- Traumatic [Shock](#) where one or more events disable metabolism
- Acute dehydration which results in blood volume loss
- Severe blood loss

The body uses two mechanical processes to control blood pressure:

- Heart Rate and Force to force blood to move blood;
- [Vascular tone](#) to direct blood where needed through vascular system.

Hypotension from a weak heart is rare because usually diagnosed as [heart disease](#). Low blood pressure usually results from failure in vascular tone maintenance.

Stress and Flow

Arterial smooth muscle tension limits blood flow, and preserves pressure. Squeezing arteries directs blood where needed by restricting flow to areas where it is not needed.

Weak arterial tone inhibits the body's ability to regulate blood flow. Likewise, systemic [hypoxia](#), that triggers an entire-body [vasodilator](#) reflex, can also result in hypotension.

Breakdown in the vascular tone is the dominant underlying cause of low blood pressure. Loss of vascular tone causes limits blood and oxygen delivery to high demand areas in the body.

Flow-control failure causes poorly supplied tissues under-perform, exhibit functional weakness produce excess lactic acid. This transient under-performance results in a wide range of syndromes and symptoms ranging from benign to severe and degeneration.

Hypotension is both a cause and an effect of vascular tone loss. When tissues that control oxygen delivery do not get enough oxygen. This is [positive feedback](#).

It evidences a durable and recurring pattern which limits stress adaptive responses.

The Migraine Connection

Migraine-like symptoms nearly always present with reduced systolic blood pressure (below 105), or with a sudden relative drop in blood pressure prior to the migraine onset. Although this connection is weakly documented in medical literature, it is easily verified.

Several theories describe migraine cause, Depolarization, Vascular, Neural and Unifying. Curiously, none of these theories suggests that tissue oxygen deprivation as a trigger or cause for migraine. Hypoxia conditions, relating to capillary performance, and functional oxygen delivery, are fully hidden in medical evaluation methods, except in advanced cases where the arteries are sufficiently degenerate and show occlusion or aneurysm.

A French study in 2007, using the Positron Emission Tomography (PET) technique identified the hypothalamus as being critically involved in the early migraine stages. A disabled hypothalamus, controls blood flow, both victim and cause of poor oxygen during a migraine. The victim/cause pattern makes complicates recovery and explains why migraines tend to last a long time 4-72 hours.

The depression wave model results from the spreading hypoxic distress of brain tissue. We assert that the hypoxic (stress) triggers a portion of the brain to enter anaerobic glycolysis which causes local acidosis, which further inhibits the aerobic metabolism of nearby brain areas, which causes them to enter anaerobic glycolysis. The effect is a "wave" which puts a large region of the brain into distress.

We assert that migraine attacks are triggered by a blood desaturation failure from a stress event. This failure causes a drop in usable oxygen delivery to brain, directly or by triggering capillary edema. The drop below the migraine-trigger-threshold causes a cascade effect of distress processes including potentially neurotransmitters, hormones, inflammation and so on.

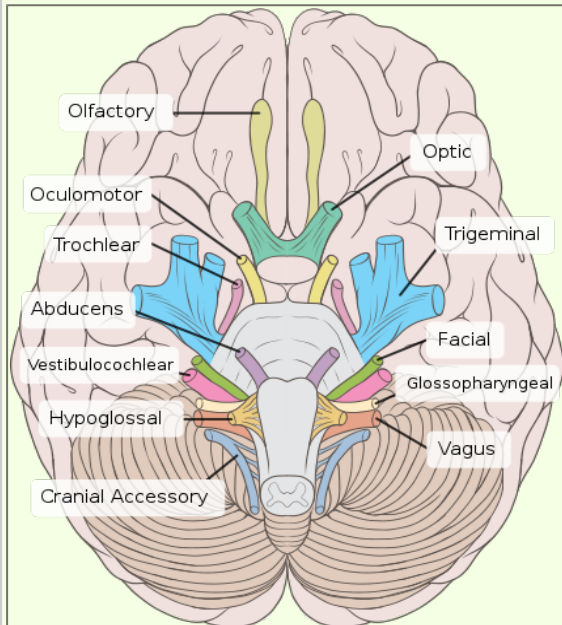
Both Manfred von Ardenne and Dr. Emanuel Revici developed methods that reduce the severity and incidence of migraines, though different, but complementary mechanisms:

- Manfred von Ardenne documented Oxygen Multistep Therapy, p- 251, 259, 282, which reduced migraine incidence and severity by restoring capillary blood flow;
- Dr. Revici and associates found that n-Butyl alcohol was sufficient to control migraines a strong majority of cases. The author asserts that this effect resulted from an unknown role as a Vasoregulator which aids restoration of normal blood flow to the brain after a migraine trigger.

Physiology Models

Hypotension is weakly defined in most medical literature. It generally reflects the inability of the body to regulate blood flow due to an absence of vascular tone. [Cardiac insufficiency](#) is outside this description.

Lack of oxygen to Brain Controls



Damage or trauma to the back of the head can establish conditions which inhibit signal generation that prevents proper blood flow.

Hypoxic trauma establishes durable blood flow reduction because of capillary swelling at the root of the vagus nerve. [See von Ardenne.](#)

Inhibited blood flow prevents normal regulation of sympathetic nervous system, including blood pressure. Leads to sympathetic/parasympathetic imbalances.

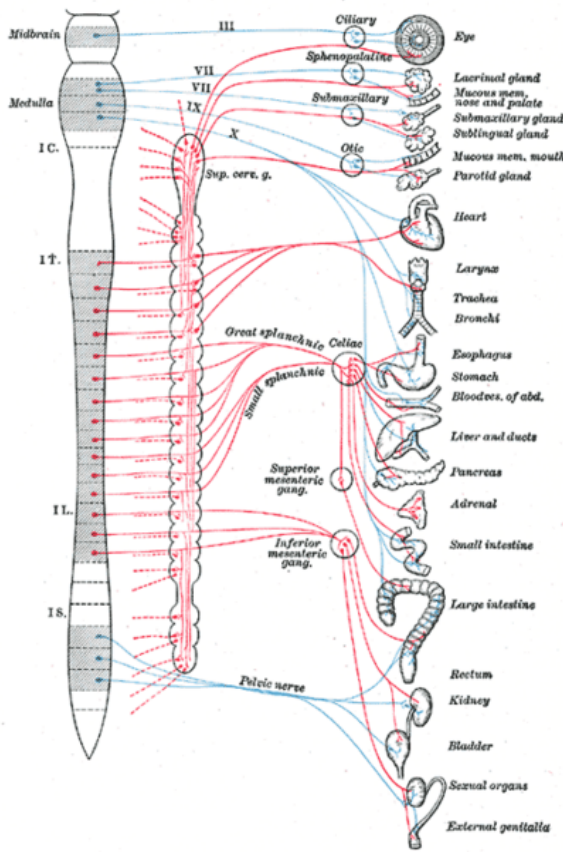
Autonomic Nervous System Imbalance

See [Vasovagal Syncope.](#)

Trauma or stress that exceeds the current adaptive range of the [autonomic nervous system](#) causes an imbalance where either the [sympathetic](#) or [parasympathetic](#) branch of the autonomic nervous system dominates.

Sympathetic dominance produces [hypertension](#), high blood pressure, while parasympathetic produces [hypotension](#), low blood pressure.

Chronic stress tends to create a durable and usually recurrent pattern of sympathetic or parasympathetic dominance. Episode recurrence reflects the normally progressive depletion metabolic agents which enable balance.



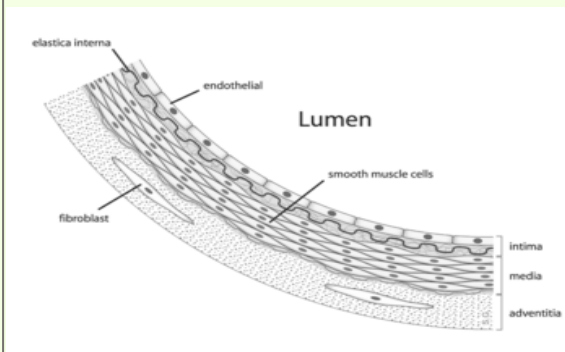
Principle agents which support autonomic balance:

- All B vitamins;
- Group 16 elements, Oxygen, Sulfur, Selenium

Agents which inhibit autonomic balance:

- All toxins which inhibit tissue oxygen delivery (cause **hypoxia**);
- **Stress** of any sort which exceeds current autoregulatory performance of the **ANS** which create conditions which lock **metabolism** into dysregulation.

Lack of oxygen to artery smooth muscles



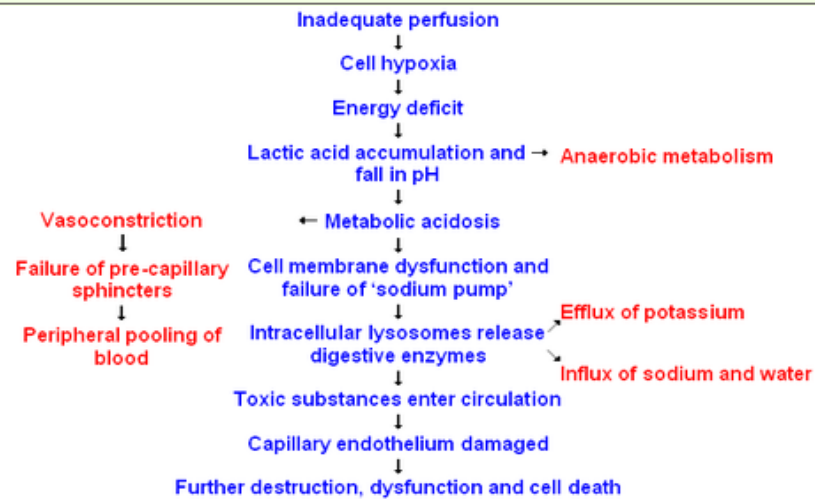
Secondary **hypoxia** is a medically unrecognized condition. While **vascularized** tissue receives oxygen from capillary networks, non-vascularized tissue is supported by **solubility** and **diffusion** processes in **blood plasma**.

Secondary hypoxia occurs when nutrient and oxygen delivery from plasma fails to meet the demands from non-vascularized tissue.

Smooth muscle cells in arteries are non-vascular and receive oxygen from plasma. When plasma oxygen concentration decreases below the vascular tone threshold, the smooth muscles cannot squeeze, resulting in hypotension.

Vascular dilation of a known effect of **hypoxia** except in the lungs, which respond with vasoconstriction, which further limits oxygen absorption.

Shock Cascade



Shock is the sequence of events which leads to systemic failure.

It reflects the process of metabolic breakdown resulting from progressive hypoxia occurring when the effects described above exceed durability.

Hypoxic Degeneration

Loss of oxygen to non-vascularized tissue enables degeneration.

Degeneration non-vascular tissue normally indicates durable decrease in secondary oxygen delivery. oxygen and likely nutrients to the degenerate tissue. Degeneration of non vascular tissue at soluble oxygen transport is degenerate to. This is a typical cause of many sorts of degeneration:

- Decreasing Vision (lens of eye)
- Vascular (arteriosclerosis)
- Connective tissue (cartilage, ligaments & tendons)

Protocol Model

Since the several models of hypotension reflect chronic stress effects, hypotension can be viewed as stress response pattern with dominant prevalence of the parasympathetic branch of the ANS. This means that the sympathetic response is inhibited or parasympathetic activation is elevated, or both.

Stress compensation performance reflects collateral performance in body systems:

1. Systemic and local oxygen delivery performance
2. Stress tolerance cofactors (neutralization & elimination)

Oxygen performance is a result of several factors (von Ardenne):

1. Adaptive delivery to demand variant tissues;
2. Unimpeded blood flow to vascularized tissue;
3. Plasma saturation for delivery to non-vascularized tissue;
4. Available reserves of oxygen delivery nutrient substrates;

Stress Tolerance Cofactors

1. Availability of [Group 6 Chalcogen Nutrients Sulfur & Selenium](#) / (Revisi) / [Guided Chemotherapy](#)
2. Oxygen Transport Cofactors, including [B Vitamins](#) & Oxygenic [Minerals, Mg](#)
3. Vascular Tone Modulators, [n-Butyl](#) & [glycerol](#)

The goal of this protocol is to optimize support of underlying compensation systems. There are several functional methods:

1. [Optimize oxygen availability to regulatory structures / Hypotension](#) (Requires strong heart)
2. Nutrients that support Oxygen Transport
3. Detoxification of agents which inhibit oxygen transport (Primary and Secondary)
4. [Optimize oxygen availability to vascular structures / 36h](#)
5. Supplement Nutrients that support Vascular Tone

Therapeutic Agent Overview

This provides concurrent maintenance of primary systems which result in failure to maintain vascular tone:

- Oxygen to brain to support areas that control blood pressure with Oxygen Multistep Therapy and Mitochondria Nutrients;
- Nutrients which aid in transfer of oxygen from blood to tissue;

Oxygen Transport Nutrients

Component	Role
B Vitamins	Vitamin Cofactors for oxygen delivery
Magnesium Orotate / Chloride	Magnesium aids oxygen use by cells and desaturation
Thiamine (Vitamin B3)	Mobilizes bile and activates liver detoxification
Arginine Alphaketoglutarate	Vasoregulator aids dilation response. Provides NO substrate for vasoregulation and CN detoxification pathways.

Toxins which bind to [hemoglobin](#) sites on red blood cells limit oxygen transport.

- [Glycated Hemoglobin](#) from chronic elevated glucose reduces oxygen binding sites on blood cells;
- Elevated plasma [ureas](#) result from under-performance in the [urea cycle](#), reduces plasma oxygen availability in blood below optimal levels. Elevated ureas decrease oxygen available to non-vascularized tissues by limiting [plasma oxygen solubility](#) or plasma oxygen desaturation or both (author). This effect increases hypoxic vulnerability to non-vascularized tissues;
- [Cyanide](#) binds hemoglobin receptors reducing oxygen transport. This can deplete [Hydroxocobalamin, B-12](#), which has stronger affinity for CN than hemoglobin. Secondly, [elevated CN can deplete Nitrite \(arginine alphaketoglutarate\) and thiosulfates substrate reserves.](#)

Detoxification

Component	Role
Magnesium Thiosulfate	Improves chloride oxidation of stress toxins. Supplies ionic magnesium and sulfur. Aids elimination Nitrate and Ammonium ureas.
Butyrate	Aids reduction of Ammonium ureas. Aids elimination of toxic lipids which accumulate with prolonged stress.
Sulfur & Selenium	Aids cellular neutralization of stress toxin antibodies that develop in response to toxic exposure. Can replace oxygen as a metabolite during acute stress because of chemical reactive similarity , hence aid acute stress tolerance when reserves are sufficient.

Vascular tone is limited by inhibition of the following control systems:

1. Inhibited signals from the brain to vagus nerve to the organ systems which govern blood pressure;
2. Reduced production of vascular neurotransmitters which govern vascular tone, [NO donors](#) and alcohols;
3. Oxygen deprivation arterial muscles which constrict to maintain vascular tone.

Vascular Tone

Component	Role
n-Butyl Alcohol	Vascular neurotransmitter which enables vascular constriction reflex (Revici) that apparently depletes under catabolic stress.
Glycerol	Probable Vascular neurotransmitter which enables vascular constriction reflex (Revici) that apparently depletes under catabolic stress.
Magnesium Chloride	Salt that aids body compensation for electrolyte disturbances and electrolyte deficiency typical with hypotension.

Protocol

This protocol provides four recommendations:

1. Restore systemic oxygen availability with Oxygen Multistep Therapy
2. Support nutrient metabolism with Myers Cocktails and OMST Nutrients
3. Support secondary detox metabolism with sulfur and selenium preparations
4. Titrate vascular tone with vasoregulatory alcohols (n-Butyl & glycerol) and magnesium rich electrolytes

There are multiple effects which result in hypotension. This protocol model divided into levels, based on therapy/nutrient combinations most likely restore normal regulation.

Level	Primary Mechanism	Therapy Model	Product

<u>1</u>	Vasoregulator/ Electrolyte Deficiency	Nutrients system which restore regulation and electrolytes	Flow EC
<u>2</u>	Oxygen Nutrient Transport Deficiency	Nutrient package to support tissue oxygen delivery	Oxygen Nutrient Transport Blend (OMST)
<u>3</u>	Capillary edema in Brain and Body	Therapy to reset capillary edema switch. See Oxygen Multistep Therapy	Oxygen Multistep Therapy System
<u>4</u>	Toxin Disrupted Oxygen Delivery	Advanced Plan Required	Physiology Assessment

If you do not achieve normal blood pressure on one level, it means that dysfunction from one or more of the other levels is likely preventing recovery.

Level 1 / Vasoregulator & Electrolyte Restoration

This titration provides a ramped support for vascular cofactors which depend on quantitative dysregulation level.

Supplement	Purpose
Flow EC	Aid kidneys and fluid distribution Magnesium salts & Glycerin. Support electrolytes to maintain fluid
Electrolyte Deficiency	Adds working salts and adrenal agents to improve ionized mineral availability. Important with Betaine-HCL to improve digestion.

Recommend experiment using Flow EC to manage blood pressure during day. Low BP is likely some combination of hypoxia. Flow C and Flow E tend to push metabolism toward Anabolic and will aid sleep.

1. Use BP Cuff to take BP
2. Use it to manage Dosage by table below
3. And take the following number of droppers

Suggested Usage Table:

Systolic BP	Droppers of Flow EC	Capsules of Electrolyte Deficiency
< 115	1	1
< 110	2	1
< 105	3	2
< 100	3	2
< 95	4	3

Dropper: 1 mL is approximately 1 dropper full or about 10 drops.

Level 2 / Oxygen Transport Nutrient Protocol

Oxygen Transport Nutrients

Component	Dosage	Role
OMST Nutrients	1-3x daily as needed	Vitamin Cofactors for oxygen delivery
Oral Myers Cocktail	1-3x weekly	Liposomal supplement delivers intracellular nutrients to support oxygen performance.
Oxyoil Sulfur/Selenium	1-3 ml/day	Provides secondary oxygenic, 6 valence, nutrients for collateral oxygen support.

Level 3 / Oxygen Multistep Therapy

Please see our [OMST protocol support](#) page for more information.

This protocol supports hypotension which results from:

- Systemic hypoxia - blood pressure is low because the whole body lacks oxygen and the vascular system does not constrict;
- [Endothelial Dysfunction](#) - where oxygen delivery to parts of the body which control blood pressure is inhibited limited because blood flow is limited by inflammation of the [endothelium](#). (documented by [Manfred von Ardenne](#))

For Hypotension, consider these options:

1. If you do not exercise regularly use [OMST 36h](#) to build durability;
2. After restoring durability use [OMST Hypotension](#);
3. User [OMST 15 min quick procedure](#), or [OMST Maintenance](#) to maintain optimal vascular performance.

Please visit our [product site for OMST systems and nutrients](#). You will need to join the site to access pricing information.

Level 4 / Detailed Analysis / Assessment

Limited response to the protocol above suggests oxygen transport and/or desaturation are acutely limited by toxic factors.

We offer advanced metabolic with our physiology assessment system. These effects likely reflect multi-system dysfunction and often require detailed intervention.

Please consider these additional approaches:

- [Get a Physiology Assessment to create a care plan](#)
- Evaluate Dysaerobic Imbalance. Use [Anabolic Repair Protocol](#)
- Test for nitrate ureas. If elevated Use [Nitrate Urea Detox](#).
- If elevated [HbA1c](#). If elevated use [PC Detox](#) to aid glucose metabolism.

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