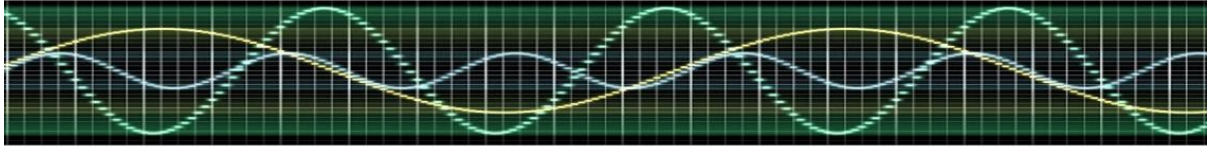




# Redland Reflexology

BODY OPTIMISER - SOUND WAVE TECHNOLOGY



Diminishing Function Risk			Optimal and Balanced			Increasing Inflammation Risk		
1	2	3	4	5	6	7	8	9
Critical	Severe	Moderate	Mild	Normal	Mild	Moderate	Severe	Critical

## ATHEROSCLEROSIS

### Some Associated References re GROK response (to check for imbalances)

To check for imbalances you can use the search function on each of the two spreadsheets of results to search for items mentioned within the GROK response.

Note: GROK warns that it's responses to queries may not be correct.

### GROK Query: JAN 2026

What functionality imbalances in the body should we look for to identify atherosclerosis using the following Body Chemistry Functionality list?

### GROK Response: JAN 2026

#### Blood 1 - Bloods relating to body chemistry interaction - Blood-Lipids

- HDL-C: Low levels indicate reduced reverse cholesterol transport, contributing to plaque buildup.
- LDL-C Direct: Elevated levels promote cholesterol deposition in arterial walls.
- Neutral Fat (MB): High levels associated with increased atherogenic risk.
- Non-HDL-C: Elevated as a comprehensive marker of atherogenic lipoproteins.
- Total Cholesterol: High levels correlate with increased atherosclerosis risk.
- Triglycerides: Elevated levels linked to atherogenic dyslipidemia.

#### Blood-Lipids2

- Apo-A-1: Low levels reduce protective effects against plaque formation.
- Apo-B: Elevated levels indicate higher numbers of atherogenic particles.
- Apo-B: Apo A-1 Ratio: High ratio signals imbalance favoring atherosclerosis.
- HDL2-C: Low levels of this subfraction reduce anti-atherogenic protection.
- LDL-P: Elevated particle number increases plaque vulnerability.
- Lp(A) Mass: High levels promote thrombosis and plaque progression.
- sdLDL-C: Elevated small dense LDL is highly atherogenic.
- HDL-P: Low particle number diminishes protective HDL function.

#### Blood-Other

- Ferritin: Elevated levels may indicate iron overload contributing to oxidative stress in arteries.
- Circulating Immune Complex (CIC): High levels suggest immune-mediated vascular inflammation.

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#### Blood 2 - Bloods relating to body chemistry interaction

- Neutrophil: Elevated counts indicate chronic inflammation promoting atherosclerosis.
- Platelets: High counts or activation contribute to thrombosis in plaques.
- Monocytes: Increased levels involved in foam cell formation in arterial walls.
- Lymphocytes: Imbalances may reflect immune dysregulation in plaque progression.
- Eosinophils: Elevated in some allergic responses linked to vascular inflammation.
- Basophils: High levels occasionally associated with inflammatory vascular conditions.
- WBC: Elevated total count signals systemic inflammation accelerating atherosclerosis.
- RDW: Increased variability associated with cardiovascular risk and plaque instability.

#### Fatty Acids, Omega-3

- Alpha-Linoleic (ALA): Low levels reduce anti-inflammatory effects on vessels.
- Docosahexaenoic (DHA): Deficiency impairs endothelial function and increases plaque risk.
- Docosapentaenoic (DPA): Low levels linked to reduced vascular protection.
- Eicosapentaenoic (EPA): Low intake or levels promote inflammation and thrombosis.
- HS-Omega-3 Index (RBC EPA+DHA): Low index is a strong predictor of atherosclerotic events.
- Omega-3 Total: Overall deficiency exacerbates omega-6/3 imbalance favoring atherosclerosis.

#### Fatty Acids, Omega-6

- Arachidonic (AA): Elevated levels promote pro-inflammatory eicosanoids in plaques.
- Gamma Linoleic Acid (GLA): Imbalances can contribute to inflammatory pathways.
- Linoleic (LA): High levels without omega-3 balance increase atherogenic risk.

#### Fatty Acids, Other

- Cis-Monounsaturated Total: Imbalances may affect lipid metabolism in arteries.
- Saturated Total: High saturated fats contribute to elevated LDL and plaque formation.
- Trans Total: Elevated trans fats directly promote endothelial dysfunction and atherosclerosis.

#### Gastrointestinal Functions 1 - Inflammation/Oxidation

- Cytokine Activity: Elevated pro-inflammatory cytokines drive atherosclerotic plaque formation.
- Fibrinogen: High levels promote clotting and plaque stability issues.
- Hs-CRP: Elevated as a key marker of vascular inflammation and atherosclerosis risk.
- Homocysteine: High levels cause endothelial damage and promote plaque buildup.
- Histamine: Imbalances may contribute to inflammatory responses in vessels.
- Lp-PLA2: Elevated indicates plaque inflammation and rupture risk.
- Myeloperoxidase: High levels promote oxidation of LDL in arterial walls.
- nf-Kappa b: Activated pathways lead to chronic inflammation in atherosclerosis.
- SedimentationRate: Elevated ESR reflects systemic inflammation associated with vascular disease.

#### Gastrointestinal Functions 2 - Insulin Resistance

- Ferritin: Elevated linked to insulin resistance and accelerated atherosclerosis.
- Free Fatty Acids (FFA): High levels contribute to lipotoxicity and endothelial dysfunction.
- HOMA-IR: Elevated score indicates insulin resistance, a major driver of atherosclerosis.
- Leptin: High levels in resistance states promote vascular inflammation.
- IRi score: High scores signal metabolic imbalance favoring plaque development.
- Oleic acid: Imbalances in fatty acids can exacerbate insulin-related vascular damage.

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#### Pancreas- Glycemic Control

- HbA1c: Elevated indicates poor glycemic control, accelerating atherosclerosis.
- Blood glucose: Chronic hyperglycemia damages endothelium and promotes plaques.
- Glycation Gap: Imbalances reflect advanced glycation end-products in vessels.

#### Nutritional Analysis 1 Amino Acids

- Arginine: Low levels impair nitric oxide production, leading to endothelial dysfunction.
- Cysteine: Imbalances affect glutathione and oxidative stress in arteries.
- Glutamine: Deficiencies may impact immune and vascular health.
- Glycine: Low levels linked to reduced anti-inflammatory effects.
- Methionine: High levels can elevate homocysteine, promoting atherosclerosis.
- Homocysteine-related amino acids (e.g., Methionine, Cysteine): Imbalances lead to hyperhomocysteinemia.

#### Nutritional Analysis 2 Macrominerals

- Calcium: Imbalances contribute to vascular calcification in atherosclerosis.
- Magnesium: Deficiency promotes endothelial dysfunction and plaque progression.
- Phosphorus: High levels with calcium imbalance lead to calcification.
- Sulphur: Imbalances affect detoxification and oxidative stress.

#### Microminerals

- Selenium: Deficiency increases oxidative damage to vessels.
- Manganese: Low levels impair antioxidant defenses.
- Iron: Excess promotes lipid peroxidation in plaques.
- Chromium: Deficiency linked to insulin resistance and atherosclerosis.
- Copper: Imbalances affect ceruloplasmin and oxidative stress.
- Zinc: Low levels impair endothelial function and increase inflammation.

#### Nutritional Analysis 3 Vitamins/CoEnzymes

- Eicosapentaenoic (EPA): Low levels reduce anti-inflammatory protection.
- B3, Niacin: Deficiency or therapeutic imbalances affect lipid profiles.
- B6, Pyridoxine: Low levels elevate homocysteine.
- B9, Folate: Deficiency causes hyperhomocysteinemia.
- B12, Cobalamin: Low levels increase homocysteine risk.
- CoEnzyme Q10: Deficiency impairs mitochondrial function in vascular cells.
- Vitamin A: Imbalances affect retinal and vascular health.
- Vitamin C: Low antioxidant levels promote oxidized LDL.
- Vitamin D, 25-Hydroxy: Deficiency strongly linked to atherosclerosis progression.
- Vitamin E: Low levels allow lipid peroxidation in plaques.
- Vitamin K: Deficiency promotes vascular calcification.
- Beta Carotene: Low antioxidant protection against oxidative stress.

#### Physical Functionality 1 Basic Physical

- Hypoxia: Chronic low oxygen promotes endothelial injury.
- pH Balance: Acidosis can exacerbate vascular inflammation.
- Glutathione: Low levels increase oxidative stress in arteries.

#### Bone Mineral Density/Disease

- Calcification, Cervical: Indicates ectopic calcification similar to atherosclerosis.
- Calcification, Lumbar: Reflects systemic calcification processes.
- Calcification, Sacrum: Part of vascular calcification patterns.
- Calcification, Thoracic: Associated with coronary artery calcification.

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- Calcium Loss: Imbalances lead to misdirected calcification in vessels.
- Rheumatism: Inflammatory conditions overlapping with atherosclerosis.

#### Physical Functionality 2 Brain Nerve

- Cerebral Arteriosclerosis: Direct indicator of atherosclerotic changes in brain vessels.
- Parasympathetic NS Function: Imbalances affect vascular tone.
- Sympathetic NS Function: Overactivity promotes hypertension and atherosclerosis.

#### Physical Functionality 3 Cardiovascular/Cerebrovascular

- Brain Tissue Blood Supply Status: Impaired supply indicates atherosclerosis.
- Cerebral Blood Vessel Elasticity: Reduced elasticity signals stiffening from plaques.
- Cerebral Blood Vessel Resistance: Increased resistance due to narrowing.
- Cerebrovascular Blood Oxygen Pressure (PaO<sub>2</sub>): Low levels from stenotic vessels.
- Cerebrovascular Blood Oxygen Saturation (Sa): Reduced in atherosclerotic disease.
- Cerebrovascular Blood Oxygen Volume (CaCO<sub>2</sub>): Imbalances reflect hypoxic risk.
- Left Ventricular Ejection Impedance: Increased in response to vascular resistance.
- Blood Fat: Elevated lipids directly contribute to plaques.
- Coronary Artery Elasticity: Reduced in atherosclerosis.
- Coronary Artery Resistance: Increased due to plaque buildup.
- Coronary Perfusion Pressure: Altered in coronary atherosclerosis.
- Galectin-3: Elevated indicates fibrosis in plaques.
- Heart Peripheral Resistance(TRR): High in systemic atherosclerosis.
- Left Ventricular Effective Pump Power: Reduced due to ischemic burden.
- Blood Viscosity: High viscosity promotes thrombosis in plaques.
- Cholesterol Crystal: Presence indicates advanced plaque formation.
- Myocardial Blood Perfusion Volume: Reduced in coronary atherosclerosis.
- Myocardial Oxygen Consumption: Imbalances from supply-demand mismatch.
- Vascular Resistance: Increased overall in atherosclerosis.
- Vascular Elasticity: Reduced arterial compliance.
- Stroke Volume (SV): Altered in advanced disease.
- Pulse Wave Coefficient K: Abnormalities indicate vascular stiffness.
- NT-proBNP: Elevated in heart strain from atherosclerosis.
- Myocardial Blood Demand: Increased unmet demand in ischemia.

#### Collagen Index

- Cartilage: Imbalances may parallel vascular matrix changes.
- Organs: Collagen alterations in vascular tissues.
- Nervous System: Affects perivascular support.
- Vessels: Reduced collagen integrity in atherosclerotic walls.

#### Physical Functionality 4 Endocrine Glands

- Adrenal cortex: Dysfunction affects cortisol and vascular inflammation.
- Adrenal medulla: Imbalances in catecholamines promote hypertension.

#### Eye Health

- Visual Fatigue: May indicate microvascular changes from atherosclerosis.

#### Physical Functionality 5 Hormones: Mood/Sex/Sleep/Stress

- Norepinephrine: Elevated promotes vasoconstriction.
- Epinephrine: High levels contribute to endothelial stress.
- Estrogen: Low levels in postmenopausal women increase risk.
- Testosterone: Low levels in men associated with higher risk.

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- Cortisol: Chronic elevation promotes inflammation and plaques.
- Dehydroepiandrosterone (DHEA): Low levels linked to vascular aging.
- Estradiol: Imbalances affect vascular protection.
- Human Growth Hormone (HGH): Deficiency accelerates vascular disease.
- Insulin-like Growth Factor 1: Low levels promote endothelial dysfunction.
- Melatonin: Low antioxidant effects increase oxidative stress.

#### Immune System

- Immunoglobulin, IgA: Imbalances in mucosal immunity may affect systemic inflammation.
- Immunoglobulin, IgG: Elevated in chronic infections linked to atherosclerosis.
- Immunoglobulin, IgM: High in acute responses contributing to plaques.

#### Kidney Function

- Creatinine, serum (mg/dL): Elevated indicates renal involvement in atherosclerosis.
- Cystatin C: High levels as a marker of vascular risk.
- Uric acid: Elevated promotes endothelial dysfunction.
- Blood urea nitrogen (BUN): High in renal atherosclerosis.

#### Physical Functionality 6 Liver and Gall Bladder Function

- ALP: Elevated in biliary or vascular issues.
- ALT: High indicates liver stress from dyslipidemia.
- AST: Elevated in metabolic syndrome with atherosclerosis.
- Bile Secretion Function: Imbalances affect lipid metabolism.
- Bilirubin (DBIL): Alterations in liver function impacting lipids.
- Detoxification, Phase I: Impaired clearance of toxins promoting oxidation.
- Detoxification, Phase II (all subtypes): Deficiencies allow accumulation of atherogenic compounds.
- Liver Fat Content: High in NAFLD, which parallels atherosclerosis.
- Protein Metabolism: Imbalances affect inflammatory proteins.
- Serum Globulin (A/G): Altered ratios in chronic inflammation.
- Serum Total Bile Acid (TBA): High in dyslipidemia.
- Total Bilirubin (TBIL): Imbalances reflect liver-vascular interplay.

#### Physical Functionality 7 Body Reproductive Function: Female

- Menopause: Associated with increased atherosclerosis risk due to estrogen decline.

#### Physical Functionality 8 Thyroid Function

- Anti-Thyroglobulin Antibody: Autoimmunity linked to vascular inflammation.
- Anti-Thyroid Peroxidase Antibody: High in thyroiditis overlapping with atherosclerosis.
- rT3, reverse T3: Imbalances in hypothyroidism promote dyslipidemia.
- T3, free: Low levels in hypothyroidism increase risk.
- T4, free: Deficiency affects lipid profiles.
- TSH: Elevated indicates hypothyroidism accelerating atherosclerosis.

#### Toxicities 3 Bacterial Diseases

- Bartonella genus (1-39): Infections promote endothelial inflammation.
- Borrelia burgdorferi: Lyme disease associated with vascular complications.
- Chlamydomphila psittaci: Related to chlamydia species linked to plaques.
- E. coli: Endotoxins contribute to inflammation.
- Helicobacter pylori: Infection correlates with increased atherosclerosis risk.
- Mycoplasma genus(1-5): Associated with chronic vascular inflammation.
- Mycobacterium tuberculosis: Latent infections may exacerbate plaques.

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#### Toxicities 4 Fungus

- Candida Albicans: Overgrowth linked to systemic inflammation.
- Cryptococcus gattli: Infections can affect vascular health.

#### Toxicities 5 Human Toxins

- Alcohol: Excessive promotes dyslipidemia and oxidative stress.
- Chemical toxicity: Exposure accelerates endothelial damage.
- Drugs, medication: Certain drugs induce atherogenic effects.
- Drugs, recreational: Stimulants increase vascular risk.
- Electromagnetic radiation: Potential oxidative stress contributor.
- Excitotoxins: Promote neurovascular inflammation.
- Herbicide toxicity: Environmental toxins linked to vascular disease.
- Pesticide toxicity: Accumulates and promotes oxidation.
- Smoke / Smog pollution: Particulates directly cause atherosclerosis.
- Stimulants: Elevate catecholamines and risk.
- Stress hormones: Chronic elevation drives inflammation.
- Tobacco/Nicotine: Major promoter of endothelial dysfunction and plaques.
- Metabolic waste: Accumulation in uremia affects vessels.

#### Toxicities 6 Minerals - Heavy Metals

- Aluminum: Accumulation promotes oxidative stress.
- Arsenic: Exposure linked to vascular calcification.
- Cadmium: High levels cause endothelial toxicity.
- Mercury: Promotes lipid peroxidation.
- Lead: Chronic exposure accelerates atherosclerosis.
- Nickel: Contributes to inflammatory responses.

#### Toxicities 7 Molds

- Stachybotrys (Black Mold): Mycotoxins promote inflammation.
- Aspergillus: Infections or toxins affect vascular integrity.

#### Toxicities 8 Parasites

- Babesiosis: Vector-borne, linked to vascular complications.
- Chagas disease: Directly causes cardiomyopathy and vascular damage.
- Malaria: Chronic infection promotes endothelial activation.
- Toxoplasmosis: Latent infection associated with increased risk.

#### Toxicities 9 Viruses

- Coxsackie Virus: Associated with myocarditis and vascular inflammation.
- Cytomegalovirus: Strongly linked to accelerated atherosclerosis.
- Epstein Barr Virus (EBV): Chronic activation promotes plaques.
- Herpes Simplex 1 (HSV-1): Infection correlates with coronary disease.
- Herpes Simplex 2 (HSV-2): Similar vascular inflammatory effects.
- Human Herpes Virus-6 (HHV-6): Linked to endothelial dysfunction.
- Influenza A: Acute infections trigger plaque rupture.
- Influenza B: Similar acute cardiovascular risks.
- Parainfluenza (1-4): Respiratory viruses exacerbate inflammation.
- Respiratory Syncytial Virus (RSV): Associated with vascular events.
- Varicella-zoster (Chickenpox): Reactivation linked to vascular disease.

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## GROK Query: JAN 2026

What functionality imbalances in the body should we look for to identify **atherosclerosis** using the following Body Components Organs list? Please identify the primary and secondary items in each section that are related to the disorder that could become imbalanced in the early stages and latter stages of the disorder.

## GROK Response: JAN 2026

### Eye Left Primary

- L Ophthalmic Artery: Endothelial dysfunction and initial plaque buildup in early stages; significant narrowing or occlusion leading to reduced blood flow in latter stages.
- L Retinal Arteries: Mild thickening and lipid deposition in early stages; complete blockage causing ischemic events in latter stages. Secondary
- L Retina: Subtle visual field changes or microvascular alterations in early stages; retinal ischemia, vision loss, or infarction in latter stages.
- L Ciliary Body: Minor perfusion deficits in early stages; chronic hypoxia leading to structural damage in latter stages.

### Eye Right Primary

- R Ophthalmic Artery: Endothelial dysfunction and initial plaque buildup in early stages; significant narrowing or occlusion leading to reduced blood flow in latter stages.
- R Retinal Arteries: Mild thickening and lipid deposition in early stages; complete blockage causing ischemic events in latter stages. Secondary
- R Retina: Subtle visual field changes or microvascular alterations in early stages; retinal ischemia, vision loss, or infarction in latter stages.
- R Ciliary Body: Minor perfusion deficits in early stages; chronic hypoxia leading to structural damage in latter stages.

### Heart Heart Primary

- Brachiocephalic trunk: Early endothelial injury and fatty streak formation; advanced plaque rupture and thrombosis in latter stages.
- L Common Carotid Artery: Initial intimal thickening in early stages; severe stenosis causing turbulent flow in latter stages.
- Pulmonary Trunk: Mild wall stiffening in early stages; extensive calcification and reduced elasticity in latter stages.
- R Pulmonary Artery: Subclinical plaque accumulation in early stages; obstructive lesions impairing pulmonary circulation in latter stages. Secondary
- R Auricle: Reduced filling due to upstream flow issues in early stages; atrial enlargement or arrhythmias from chronic ischemia in latter stages.
- Chordae Tendineae: Minor tensile stress changes in early stages; rupture or dysfunction from ventricular remodeling in latter stages.
- Posterior Papillary Muscle: Subtle contractile impairment in early stages; infarction leading to mitral regurgitation in latter stages.
- Superior Vena Cava: Flow disturbances from adjacent arterial issues in early stages; congestion from right heart failure in latter stages.
- Inferior Vena Cava: Mild venous return alterations in early stages; severe congestion and edema in latter stages.

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#### Heart Anterior Cut Primary

- Pulmonary Arteries: Early lipid infiltration in vessel walls; critical narrowing or embolism in latter stages.
- Aorta: Intimal damage and cholesterol deposits in early stages; aneurysm formation or dissection in latter stages.
- Pulmonary Trunk: Subtle hardening in early stages; significant lumen reduction affecting right ventricular output in latter stages. Secondary
- Right Atrium: Mild pressure elevations in early stages; dilation and failure from chronic overload in latter stages.
- Right Ventricle: Reduced ejection fraction subtly in early stages; hypertrophy and failure in latter stages.
- Left Atrium: Flow imbalances from pulmonary issues in early stages; fibrillation or thrombosis in latter stages.
- Left Ventricle: Early wall stress increases; myocardial infarction or cardiomyopathy in latter stages.
- Papillary Muscle: Contractile inefficiency in early stages; rupture causing valve incompetence in latter stages.

#### Heart Top Cut Primary

- Pulmonary Trunk: Early endothelial dysfunction; advanced occlusion impacting pulmonary flow in latter stages.
- Aorta: Fatty plaque initiation in early stages; rupture or severe stenosis in latter stages. Secondary
- Left Ventricle: Mild hypertrophic changes in early stages; dilation and systolic dysfunction in latter stages.
- Right Ventricle: Subclinical strain in early stages; cor pulmonale in latter stages.
- Papillary Muscle: Minor ischemic changes in early stages; necrosis leading to valvular issues in latter stages.

#### Organs Adrenal Cross Section Primary

- Blood Vessels: Early plaque formation in supplying arteries; complete blockage causing glandular hypoperfusion in latter stages. Secondary
- Zona Fasciculata: Subtle cortisol production fluctuations in early stages; atrophy and adrenal insufficiency in latter stages.
- Zona Reticularis: Mild androgen imbalances in early stages; significant hormonal disruptions in latter stages.
- Zona Glomerulosa: Early aldosterone secretion changes; chronic hypoaldosteronism from ischemia in latter stages.
- Medulla: Minor catecholamine release alterations in early stages; severe dysfunction leading to autonomic instability in latter stages.

#### Organs Brain Sagittal Primary

- Great Cerebral Vein (of Galen): Secondary flow restrictions from arterial issues in early stages; thrombosis or congestion in latter stages. Secondary
- Thalamus (3rd ventricle): Mild sensory processing deficits in early stages; infarction causing thalamic syndrome in latter stages.
- Cerebral Peduncle: Subtle motor pathway disruptions in early stages; hemiplegia from stroke in latter stages.
- Cerebellum: Coordination imbalances subtly in early stages; ataxia from chronic hypoperfusion in latter stages.
- Medulla Oblongata: Early autonomic fluctuations; respiratory or cardiovascular failure in latter stages.

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#### Organs Kidney Cross Section L Primary

- L Renal Artery: Initial intimal thickening in early stages; stenosis leading to renovascular hypertension in latter stages.
- L Arcuate Arteries: Mild luminal narrowing in early stages; occlusion causing cortical infarcts in latter stages.
- L Interlobular Arteries: Endothelial damage in early stages; thrombotic events in latter stages. Secondary
- L Cortex: Subclinical filtration reductions in early stages; atrophy and chronic kidney disease in latter stages.
- L Medulla: Minor tubular function changes in early stages; ischemia leading to acute kidney injury in latter stages.
- L Renal Pelvis: Flow dynamics alterations in early stages; hydronephrosis from secondary hypertension in latter stages.

#### Organs Kidney Cross Section R Primary

- R Renal Artery: Initial intimal thickening in early stages; stenosis leading to renovascular hypertension in latter stages.
- R Arcuate Arteries: Mild luminal narrowing in early stages; occlusion causing cortical infarcts in latter stages.
- R Interlobular Arteries: Endothelial damage in early stages; thrombotic events in latter stages. Secondary
- R Cortex: Subclinical filtration reductions in early stages; atrophy and chronic kidney disease in latter stages.
- R Medulla: Minor tubular function changes in early stages; ischemia leading to acute kidney injury in latter stages.
- R Renal Pelvis: Flow dynamics alterations in early stages; hydronephrosis from secondary hypertension in latter stages.

#### Organs Liver Detail Primary

- Intralobular Arteriole: Early plaque buildup; occlusion reducing hepatic blood supply in latter stages. Secondary
- Sinusoids: Mild flow impairments in early stages; congestion and liver dysfunction in latter stages.
- Hepatocyte: Subtle metabolic changes in early stages; necrosis or cirrhosis from ischemia in latter stages.
- Kupffer Cells: Inflammatory activation in early stages; overwhelmed immune response in latter stages.

#### Organs Liver Vessels Primary

- Hepatic Artery: Endothelial dysfunction in early stages; severe narrowing causing hepatic ischemia in latter stages. Secondary
- Portal Vein: Secondary pressure increases in early stages; portal hypertension in latter stages.
- Inferior Vena Cava: Flow disturbances in early stages; Budd-Chiari syndrome-like issues in latter stages.

#### Organs Lung Lobes Alveoli Primary

- Branch Of pulmonary Artery: Mild stiffening in early stages; embolism or occlusion in latter stages. Secondary
- Aveolar Sac: Subtle gas exchange inefficiencies in early stages; hypoxia and respiratory failure in latter stages.

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- Capillaries: Microvascular changes in early stages; pulmonary hypertension in latter stages.
- Type I Pneumocyte: Minor diffusion barriers in early stages; alveolar damage in latter stages.

#### Organs Meninges Cross Section Primary

- Blood Vessel: Early arterial plaque; rupture causing subarachnoid hemorrhage in latter stages. Secondary
- Subarachnoid Space: Mild pressure changes in early stages; hematoma formation in latter stages.
- Pia Matter: Subtle nutrient delivery issues in early stages; inflammation or adhesions in latter stages.

#### Organs Prostate Cross Section Primary

- Vasculature: Initial arterial narrowing in early stages; ischemia from occlusion in latter stages. Secondary
- Urethra: Flow restrictions in early stages; obstruction from tissue changes in latter stages.
- Smooth Muscle Cells: Contractile imbalances in early stages; atrophy in latter stages.

#### Organs Skin Cross Section Primary

- Artery: Endothelial damage in early stages; peripheral occlusion in latter stages. Secondary
- Dermis: Mild perfusion deficits causing delayed healing in early stages; ulceration or gangrene in latter stages.
- Blood Capillary: Microcirculatory slowdown in early stages; capillary rarefaction in latter stages.

#### Organs Small Intestine Wall Primary

- Submucosal Artery: Plaque initiation in early stages; mesenteric ischemia in latter stages. Secondary
- Villi: Nutrient absorption reductions in early stages; malabsorption syndrome in latter stages.
- Lacteal: Lymphatic drainage issues secondary to vascular problems in early stages; edema in latter stages.

#### Organs Stomach Cross Section Primary

- Submucosal Artery: Early luminal narrowing; occlusion leading to gastric ischemia in latter stages. Secondary
- Gastric Gland: Secretory imbalances in early stages; ulceration or atrophy in latter stages.
- Parietal Cells: Acid production fluctuations in early stages; cell death in latter stages.

#### Organs Teeth Cross Section Primary

- Blood Vessels: Vascular thickening in early stages; pulpal ischemia in latter stages. Secondary
- Pulp: Inflammatory changes in early stages; necrosis in latter stages.
- Dentin: Nutrient supply deficits in early stages; structural weakening in latter stages.

#### Organs Thyroid Cross Section Primary

- Blood Vessels: Arterial plaque buildup in early stages; reduced glandular perfusion in latter stages. Secondary

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- Thyroid Follicle: Hormone synthesis mild disruptions in early stages; hypothyroidism from ischemia in latter stages.
- Follicular Cells: Cellular stress in early stages; apoptosis in latter stages.

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