Phonocardiography and Optimising Human Performance

Topics to cover today
- Phonocardiography review
- Conscious / Subconscious balance
- Genotype and Phenotype meridians
- Eyes into distortion
- ATP and Hypoxia challenges
- Nutrition for energy
- Hypoxia
- Toxicity
- Infection
- Energy requirements in sport

Phonocardiography diagnostic techniques can be performed by using a digital stethoscope but similar results can be achieved by using a standard stethoscope but you just will not be able to see the cardiograph.
RED constitution people tend to have high Homocysteine levels and have APOE4 expressions. GREEN constitutional people tend to have arteriosclerosis, angina pectoris, intermittent claudication and valvular stenosis. BLUE constitution people tend to have arrhythmias and cardiac genetic defects and valvular regurgitation.

Normal Heart Sounds

The first sound is 2 to 3 times louder than the second. The period between the second sound and the next first sound is twice as long as the period of time between the first sound and the second. This is normal. Anything different is abnormal.
Both auricular / ventricular valves must close at the same time. That closure is the first heart sound (LUB).

Pulmonary and aortic valves are closed by the blood pressure pushing back creating the second sound (DUB).

Rest period is longer as this is the period that the ventricles are opening again and should be twice as long as the closing period.
Phonocardiography

Optimising human performance depends upon optimal ATP mitochondrial production requiring
1. Optimal nutritional
2. Optimal oxygen delivery
3. Absence of toxins
4. Absence of infections
5. Positive emotional state
LIFE and HEALTH ARE DEPENDANT UPON ADEQUATE NUTRITIONAL INTAKE

AMINO ACIDS
1. BUILD TISSUES
2. TRANSPORT MOLECULES
3. FORM ANTIBODIES
4. FORM ENZYMES
5. BUILD CHEMICAL MESSENGERS i.e. HORMONES AND NEUROTRANSMITTERS

FATTY ACIDS
1. FORM CELL MEMBRANES
2. ARE SOURCES OF ENERGY
3. ARE STORES OF ENERGY
4. PROTECT ORGANS
5. ACT AS ELECTRICAL AND THERMAL INSULATORS
6. BUILD STEROID HORMONES
CARBOHYDRATES

1. Are a source of energy
2. Link with amino acids to form glycoproteins
3. Link with fatty acids to form glycolipids.

VITAMINS

1. ACT AS CO-ENZYMES IN SPECIFIC ENZYME PATHWAYS
2. ACT AS ANTIOXIDANTS
3. INVOLVED WITH BLOOD CLOTTING
4. PART OF CELL MEMBRANES

Co-Enzymes

Thiamine, Thiamine phosphate, FMN, FAD, NAD, NADP, CoA, Pyridoxal-5-phosphate, H4Folate, Methenyl H4 Folate

Methylene H4 Folate, Methyl H4 Folate, Adenosylcobalamin, Methylocobalamin, Biotin, Vitamin C, Alpha Lipoic acid, SAM, CoQ10
MINERALS ACT TO
1. Supply major elements and trace elements that may be lacking in the diet.
2. Act as catalysts, thus playing a major role in metabolism and cell building.
3. Regulate the permeability of cell membranes.
4. Maintain water balance and osmotic pressure between the inside and outside environment.
5. Influence the contractility of muscles.
6. Regulate the response of nerves to stimuli.

LIFE DEPENDS UPON IONIC BALANCE TO MAINTAIN HOMEOSTASIS

IONIC BALANCE DEPEND UPON ADEQUATE NUTRIENT UPTAKE FROM IONIZED MINERALS
The daily 1500–2000 Calories recommended for a human adult are taken as a combination of oxygen and food molecules, the latter mostly carbohydrates and fats, of which glucose \((C_6H_{12}O_6)\) and stearic acid \((C_{57}H_{110}O_6)\) are convenient examples.

The food molecules are oxidised to carbon dioxide and water in the mitochondria

\[
C_6H_{12}O_6 + 6O_2 \rightarrow 6CO_2 + 6H_2O
\]
\[
C_{57}H_{110}O_6 + 81.5O_2 \rightarrow 57CO_2 + 55H_2O
\]

and some of the energy is used to convert ADP into ATP

\[
ADP + HPO_4^{2-} \rightarrow ATP + H_2O
\]

The rest of the chemical energy in the carbohydrate or fat is converted into heat: the ATP is used as “energy currency”, and some of the chemical energy it contains when split and reacted with water, is used for other metabolism.
(At each stage of a metabolic pathway, some chemical energy is converted into heat).

Only a tiny fraction of the original chemical energy is used for work.
Understanding energy production is as easy as 1,2,3

1

2

3

1. Glycolysis
2. Kreb’s cycle
3. Electron transport

Glycolysis

Kreb’s cycle

Electron transport

For every 1 molecule of Glucose, 38 molecules of ATP are formed.

- 8 ATP by Glycolysis
- 2 ATP in the Kreb’s cycle
- 28 ATP by Electron transport

Glucose

Glycolysis

Pyruvate

Acetyl CoA

Kreb’s cycle

ATP

Electron transport

ADP
Glycolysis

Pyruvate

Acetyl CoA

Kreb's cycle

ATP Electron transport ADP

Alternative sources of fuel

Fatty acids can be oxidized as Acetyl CoA

Amino acids can be oxidized in the Kreb's Cycle

Magnesium

Zinc

Potassium

NAD

Vit B1 (TP)

Vit B2 (R5P)

Vit B3 (NAD)

α-Lipoic acid

Magnesium

Vit B1 (TP)

Vit B2 (R5P)

Vit B3 (NAD)

Vit B5 (CoA)

α-Lipoic acid

Magnesium

Manganese

CoQ10

Iron

Sulfur

Phosphorus

O2

Energy pathway
The 10 most common medical symptoms
- Fatigue
- Back ache
- Colds
- Respiratory
- Abdominal pains
- Anxiety / Depression
- Memory loss / Vision dysfunction
- Arthritis
- Skin
- Chest pains
The 10 most common diseases/causes of death in 2012 per 100,000 population in the UK were:
1. Coronary ischaemic heart disease
2. Cerebrovascular disease
3. Malignant neoplasm of trachea
4. Pneumonia
5. Diseases of pulmonary circulation
6. Bronchitis, emphysema and COPD
7. Malignant neoplasm of breast
8. Chronic liver disease and cirrhosis
9. Diabetes mellitus
10. Hypertensive disease

Hypoxia is a condition in which the body or a region of the body is deprived of adequate oxygen supply.

Hypoxia may be classified as either *generalized*, affecting the whole body, or *local*, affecting a region of the body.

Symptoms
Gradual onset - Light-headedness
Numbness / tingling of extremities, Nausea and anorexia.
Tiredness
Visual deterioration
Memory loss
Feeling the cold
Degenerative changes
Symptoms
Rapid onset - ataxia, confusion / disorientation / hallucinations / behavioural change, severe headaches / reduced level of consciousness, papilloedema, breathlessness, pallor, tachycardia and pulmonary hypertension.

If hypoxia is very severe, a tissue may eventually gangrene. Extreme pain may also be felt at or around the site. Eventually leading to the late signs cyanosis, bradycardia / cor pulmonale and hypotension followed by death.

Because haemoglobin is a darker red when it is not bound to oxygen (deoxyhaemoglobin), as opposed to the rich red colour that it has when bound to oxygen (oxyhaemoglobin), when seen through the skin it has an increased tendency to reflect blue light back to the eye.
Hypoxia can result from a failure at any stage in the delivery of oxygen to cells. This can include decreased partial pressures of oxygen, problems with diffusion of oxygen in the lungs, insufficient available haemoglobin, problems with blood flow to the end tissue, and problems with breathing rhythm.

Functional Testing for Hypoxia

Phonocardiography
80% of patients show a deceased first sound with most of them due to Hypoxia.

Oxygen saturation is a term referring to the concentration of oxygen in the blood. The human body requires and regulates a very precise and specific balance of oxygen in the blood. Normal blood oxygen levels in humans are considered 95-100 percent. If the level is below 90 percent, it is considered hypoxia.

Blood oxygen levels below 80 percent may compromise organ function, such as the brain and heart, and should be promptly addressed. Continued low oxygen levels may lead to respiratory or cardiac arrest.
In medicine, oxygen saturation (SO$_2$), commonly referred to as "sats", measures the percentage of hemoglobin binding sites in the bloodstream occupied by oxygen. At low partial pressures of oxygen, most hemoglobin is deoxygenated.

At around 90% (the value varies according to the clinical context) oxygen saturation increases according to an oxygen-hemoglobin dissociation curve and approaches 100% at partial oxygen pressures of >10 kPa.
A pulse oximeter relies on the light absorption characteristics of saturated hemoglobin to give an indication of oxygen saturation.

Functional Biochemistry Testing
i) All muscles weak on testing
ii) Single muscle weakens on repeated muscle testing (aerobic challenge)
iii) Positive eyes into distortion up and down
iv) Weak muscle strengthens to Oxygen
v) Strong muscle weakens to CO2 and / or Xanthine oxidase

How to start examining a patient
Balancing the Conscious to the Subconscious
1. Therapy localise the Conscious ESR on the frontal bones.
2. Then Therapy localise the Subconscious ESR on the greater wings of the sphenoid bone.

Or
1. Therapy localise the Subconscious ESR on the greater wings of the sphenoid bone.
2. Then Therapy localise the Conscious ESR on the frontal bones.
3. If weakness occurs then give MIRON light therapy to the umbilicus for one minute.

Challenge for YANG and YIN positive B & E Points. There will always be at least one of each.

Cross Therapy Localise to find which one negates the other. The one that negates the other is the genotype. The other is the phenotype.
The genotype of a person is the inherited instructions it carries within its genetic code. The phenotype is the composite of an person’s observable characteristics such as biochemical or physiological properties resulting from the expression of the genes as well as the influence of environmental factors and the interactions between the two.

Eyes into Distortion (EID)

4. Dehydration
7. Infection
1. Nutrition
5. Exercise
2. Toxicity
6. Allergy
3. Mechanics

8. Hypoxia

Patient Protocol for Hypoxia
From weakness patient strengthens to HYPOXIC eye position
Confirm using OXYGEN vial to strengthen
Challenge using following vials

PHOSPHOLIPIDS
EPO, BSO, Borage
Black cumin
Flax, Chia
Grape seed
Hazelnut, Hemp
Macadamia
Olive, Coconut
Peanut
Pumpkin
Super Omega 3
Walnut
WGO

HEMOGLOBIN
ALA
PBG
UPG III
CPG III
PP IX

Co-ENZYME Q10
Co-Q10 in oil
Oxygen in the air

Red blood cell

Tissue cell

Tissue cell mitochondria

Reactive Oxygen Species

Normal mitochondrial oxidation
Respiratory burst
Phase 1 detoxification
Hypoxia / Hyperoxia

SOD
Fe - Cu

Catalase
Glutathione peroxidase
NADH peroxidase
Other Peroxidases

Hydroxyl radical

Water + O2

Arginine

NADPH

NADP+ Citrulline

Nitric oxide

Peroxynitrite

Hydrogen peroxide

Singlet oxygen
Reactive Oxygen Species

1. Normal mitochondrial oxidation
2. Respiratory burst
3. Phase I detoxification
4. Hypoxia / Hyperoxia
5. (Xanthine oxidase)

SOD
- Fe
- Zn/Cu

DOD
- Mn

Catalase

Glutathione peroxidase

NADH
Peroxidase

Other Peroxidases

KILLS
+ve BACTERIA, VIRUSES

KILLS
-ve BACTERIA, VIRUSES, FUNGI

KILLS PARASITES

KILLS VIRUSES, FUNGI, PARASITES

Oxygen transport
Oxygen into the Lungs
By volume, dry air contains
78.09% nitrogen
20.95% oxygen
0.93% argon
0.039% carbon dioxide
and small amounts of other gases.
Air also contains a variable
amount of water vapor, on average
around 1%.

At sea level the partial pressure of
oxygen (pO2) in the lungs = 21%
of atmospheric pressure 760mm
Hg = 160mm Hg.
At 16000ft with atmospheric
pressure at 400mm Hg pO2 =
82mm Hg

Henry’s Law of Solution states
that the quantity of a gas going
into simple solution at constant
temperature is proportional to the
pressure. The solubilities of
oxygen, carbon dioxide and
nitrogen are in the ratio of
2:50:1
Movement of gases is always from the region of high tension to a region of low tension. Oxygen will thus pass from the lung alveoli to the blood and then to the tissues. CO2 tension is higher in the blood so passes from the blood to the alveoli.

Oxygen is transported in the blood in 2 ways
1. Dissolved in the plasma = 0.3 volume %. Small but important in determining the oxygen tension gradient from the plasma to the tissues.
2. Combined with haemoglobin in the red cell.

Almost all the oxygen in the blood is bound to hemoglobin, so interfering with this carrier molecule limits oxygen delivery to the periphery.

Hemoglobin increases the oxygen-carrying capacity of blood by about 40-fold,
with the ability of hemoglobin to carry oxygen influenced by the partial pressure of oxygen in the environment, a relationship described in the oxygen–haemoglobin dissociation curve. When the ability of hemoglobin to carry oxygen is interfered with, a hypoxic state can result.

At tensions above 100mm Hg the haemoglobin is fully saturated with oxygen and the dissociation curve is plotted as a percentage saturation against tension.

The Bohr Effect
In addition to tension and haemoglobin content, the oxygen content of the blood depends upon the CO2 being carried simultaneously. An increase in pCO2 from the normal value of 40mm Hg shifts the oxygen dissociation curve thus less oxygen is carried at a given tension.
Markers for Hypoxia

Strong muscle weakens to CO2 and / or Xanthine oxidase

Oxygen transport

Blood leaves the lungs at an oxygen tension of 100mm Hg and returns at 40mm Hg.

Carbon dioxide transport

Only 4ml% is given off in the passage through the lungs which equals the amount taken up by the tissues.
Mechanics of Breathing

Lung volume

3400ml
3000ml
0ml

Tidal volume

Resting respiratory level

time

Forced expiration

Residual volume

time
Lung volume: 5000ml

- Forced inspiration followed by forced expiration
- Vital Capacity
- Residual Volume
- Tidal volume = 400ml
- Only 250ml of this air reaches the alveoli, the last 150ml remains in the bronchial tubes and is called dead space air.

Chief muscles of breathing are the Diaphragm and the Intercostals.
Inspiration is an active process of depressing the diaphragm down wards and contracting the intercostal muscles \ moving the chest wall upwards and outwards.

Expiration is brought about by passive elastic recoil of the lungs and relaxation of the inspiratory muscles.

Mechanical Faults

1. Cranial
2. Cervical spine
3. Thoracic spine
4. Diaphragm
5. M/S joint
6. Sternoclavicular joint
7. Acromioclavicular joint
8. Ribs
9. Lumbar spine
Oxygen into the Blood

The alveoli are located in the respiratory zone of the lungs, at the distal termination of the alveolar ducts and atria. These air sacs are the forming and termination point of the respiratory tract. They provide total surface area of about 100 m².

The alveoli consist of an epithelial layer and extracellular matrix surrounded by capillaries. The alveoli contain some collagen and elastin fibres. The elastic fibres allow the alveoli to stretch as they are filled with air during inhalation. They then spring back during exhalation in order to expel the carbon dioxide-rich air.
There are three major cell types in the alveolar wall
1. Type I (Squamous Alveolar) cells that form the structure of an alveolar wall

2. Type II (Great Alveolar) cells that secrete pulmonary surfactant to lower the surface tension of water and allows the membrane to separate, therefore increasing its capability to exchange gases.

3. Macrophages that destroy foreign material, such as bacteria.

Re-inflation of the alveoli following exhalation is made easier by pulmonary surfactant, which is a phospholipid and protein mixture that reduces surface tension in the thin fluid coating within all alveoli. The fluid coating is produced by the body in order to facilitate the transfer of gases between blood and alveolar air.
Plasma membranes consist of both lipids and proteins. The fundamental structure of the membrane is the phospholipid bilayer, which forms a stable barrier between two aqueous compartments. In the case of the plasma membrane, these compartments are the inside and the outside of the cell.

Plasma membranes of human cells contain four major phospholipids
1. Phosphatidylcholine,
2. Phosphatidylethanolamine
3. Phosphatidylserine,
4. Sphingomyelin
which together account for more than half of the lipid in most membranes.

These phospholipids in human red blood cells are asymmetrically distributed between the two halves of the membrane bilayer.
The outer leaflet consists mainly of phosphatidylcholine, sphingomyelin and glycolipids.

Where as phosphatidylethanolamine and phosphatidylyserine are the predominant phospholipids of the inner leaflet.

A fifth phospholipid, phosphatidylinositol, is also localized to the inner half of the plasma membrane.

Although phosphatidylinositol is a quantitatively minor membrane component, it plays an important role in cell signalling.

The head groups of both phosphatidylyserine and phosphatidylinositol are negatively charged, so their predominance in the inner leaflet results in a net negative charge on the cytosolic face of the plasma membrane.
In addition to the phospholipids, the plasma membranes of animal cells contain glycolipids and cholesterol. The glycolipids are found exclusively in the outer leaflet of the plasma membrane, with their carbohydrate portions exposed on the cell surface.

They are relatively minor membrane components, constituting only about 2% of the lipids of most plasma membranes. Cholesterol is a major membrane constituent of human cells, being present in about the same molar amounts as the phospholipids.
A Phospholipid

- Polar head group (hydrophilic)
- Apolar, hydrocarbon tails (hydrophobic)

The unsaturated fatty acid tails are kinked and lead to more spacing between the polar heads and hence more movement.

R may be Choline, Inositol, Ethanolamine, Serine, Sphingomyelin
Cell Membranes

Passive transport      Active transport

Key nutrients for synthesising the phospholipids
Acetyl CoA (Vit B5, Magnesium, P5P)
NAD, NADPH (Vit B3 complex)
Mg, Zn, SAM (Mg, P-5-P, Folates, B12)
Choline
Serine
Inositol
Saturated fatty acids C16-18
Unsaturated fatty acids C18-24
Lecithin
Lecithin is a generic term to designate any group of yellow-brownish fatty substances occurring in animal and plant tissues composed of phosphoric acid, choline, fatty acids, glycerol, glycolipids, triglycerides, and phospholipids (e.g., phosphatidylcholine, phosphatidylethanolamine, and phosphatidylinositol).

Soybean-derived Lecithin dietary supplements are composed of 19-21% Phosphatidylcholine, 8-20% Phosphatidylethanolamine, 20-21% Inositol phosphatides, 33-35% Soybean oil, 2-5% Sterols, 5% Carbohydrates/free, 1% Moisture, and 5-11% Other phosphatides.¹

Lecithin is only found natural in natural fats, and is not found in processed foods. Foods containing lecithin include: chia seeds, butter, eggs, soy, pumpkin seeds and beef. Lecithin helps break up fats (emulsifier), and helps the body to absorb and use vitamins and calcium.
Hemoglobin saturation
The quantity of oxygen carried by the saturated blood will depend upon the haemoglobin content of the red cells. With a normal haemoglobin of 14.5gm/100ml blood 20ml of oxygen will combine with the haemoglobin in every 100ml of blood (20 volume %).

The amount carried when fully saturated is called the oxygen capacity.

Hemoglobin is also found outside red blood cells in the A9 dopaminergic neurons in the substantianigra, macrophages, alveolar cells, and mesangial cells in the kidney. In these tissues, hemoglobin has a non-oxygen-carrying function as an antioxidant and a regulator of iron metabolism.
Anaemias

Anaemia is a decrease in number of red blood cells or less than the normal quantity of hemoglobin in the blood. Anaemia may also be diagnosed where there is decreased oxygen-binding ability of each hemoglobin molecule due to deformity or lack in numerical development as in some other types of hemoglobin deficiency.

1. Red cell aplasia
2. Aplastic anaemia
3. Microcytic anaemia – Iron deficiency
4. Macrocytic anaemia's – Vitamin B12, Folic acid
5. Hemolytic anaemia
6. Blood loss
7. Fluid overload
Iron deficiency maybe due to
1. Diet
2. Malabsorption
3. Parasites
4. Haemorrhage

Supplement with
Ferrous phosphate RED body types
Ferrous Chloride GREEN body types
Ferrous sulphate BLUE body types

Hemoglobin and Myoglobin contain heme, a cyclic tetrapyrrrole consisting of 4 molecules of pyrrole. One atom of ferrous iron resides at the centre.

Heme-dependent enzymes
Catalase
Various peroxidases
i-Nitric Oxide Synthase
Myeloperoxidase
Cystathione synthase
Cytochrome p450
Cytochromes for energy production
Sulfite oxidase
Thyroperoxidase
Succinyl CoA is formed in the Krebs’ cycle.

Magnesium seems to work as it helps in converting the 6-unit molecule (a hexamere) of the enzyme to the 8-unit being much more active.
PBG is shunted to HPL probably by a CYP 450 enzyme.

Always challenge with mauve acetate when high Uroproporphyrin III or Coproporphyrin III.
Hemoglobin is composed of heme with one Fe2+ and a globin protein composed of an alpha chain of 141 amino acids and one beta chain of 145 amino acids.

<table>
<thead>
<tr>
<th>Alpha chain</th>
<th>Beta chain</th>
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<tbody>
<tr>
<td>Phenylalanine 6</td>
<td>Phenylalanine 8</td>
</tr>
<tr>
<td>Lysine 11</td>
<td>Lysine 9</td>
</tr>
<tr>
<td>Threonine 9</td>
<td>Threonine 6</td>
</tr>
<tr>
<td>Valine 11</td>
<td>Valine 18</td>
</tr>
<tr>
<td>Methionine 2</td>
<td>Methionine 1</td>
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<tr>
<td>Leucine 17</td>
<td>Leucine 18</td>
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</table>
Myoglobin in muscle cells stores oxygen in the resting state as oxymyoglobin and on exercise releases oxygen.

It is composed of the same amino acids as in hemoglobin.

Oxygen (O2), nitric oxide (NO), carbon monoxide (CO) and hydrogen sulfide (H2S) bind to the iron atom in heme proteins. Once bound to the prosthetic heme groups, these molecules can modulate the activity/function of those hemeproteins, affording signal transduction.

Myeloperoxidase (MPO) is a peroxidase enzyme and is most abundantly expressed in neutrophil granulocytes. MPO has a heme pigment, which causes its green colour in secretions rich in neutrophils, such as pus and some forms of mucus.
Thyroid peroxidase or thyroperoxidase (TPO) is an enzyme expressed mainly in the thyroid that liberates iodine for addition onto tyrosine residues on thyroglobulin for the production of thyroxine (T₄) or triiodothyronine (T₃), the thyroid hormones.

Pyroluria is known by many different names including Pyrrole Disorder, Kryptopyrrole, Kryptopyrroluria, Pyrroluria, Pyrolle Disorder, Mauve Factor and Hemeprrole. Pyroluria can best be described as the abnormal synthesis and metabolism of the oxygen carrying molecule haemoglobin.
As with all cells there are waste or by-products produced and the by-product of haemoglobin is a metabolite called hydroxyhemopyrrolin-2-one (HPL) also known as Pyrrole. The metabolite was originally thought to be a Kryptopyrrole but further studies have proven this not to be the case.

The Mauve Factor

OHHPL (hydroxyhemopyrrolin-2-one)

OHHPL (Mauve Factor)

• In human urine, blood and CSF
• Mistakenly identified as kryptopyrrole, a persistent erroneous term
• Chemically similar to kryptopyrrole, which can be used for OHHPL assay
Mauve history
- Discovered in urine in 1957
- Named for lilac-coloured appearance on paper chromatograms developed with Erhlich’s reagent
- Labile and elusive
- Abram Hoffer is the father of Mauve

High-Mauve and behaviour
- Down syndrome 70%
- Schizophrenia 40-70%
- Autism 50%
- ADHD 30%
- Alcoholism 20-80%
<table>
<thead>
<tr>
<th>Pfeiffer correlates</th>
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<tbody>
<tr>
<td>• Nail spots</td>
</tr>
<tr>
<td>• Stretch marks</td>
</tr>
<tr>
<td>• Pale skin</td>
</tr>
<tr>
<td>• Poor tanning</td>
</tr>
<tr>
<td>• Knees and joints</td>
</tr>
<tr>
<td>• Constipation</td>
</tr>
<tr>
<td>• Dream recall</td>
</tr>
<tr>
<td>• Morning nausea</td>
</tr>
<tr>
<td>• Light and sound</td>
</tr>
<tr>
<td>• Odour intolerance</td>
</tr>
<tr>
<td>• Migraines</td>
</tr>
<tr>
<td>• Stitch-in-side</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Walsh</th>
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<tbody>
<tr>
<td>• Low stress tolerance</td>
</tr>
<tr>
<td>• Anxious, overly pessimistic</td>
</tr>
<tr>
<td>• Explosive anger</td>
</tr>
<tr>
<td>• Hyperactivity</td>
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<tr>
<th>Kruesi</th>
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<tbody>
<tr>
<td>• Social withdrawal</td>
</tr>
<tr>
<td>• Emotionally labile</td>
</tr>
<tr>
<td>• Loss of appetite</td>
</tr>
<tr>
<td>• Easily fatigued</td>
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</tbody>
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| • Abnormal fat distribution        |
| • Irritable bowel                  |
| • Delayed puberty                  |
| • Irregular periods                |
| • Overcrowded teeth                |
| • Joint pains                      |
| • Reading difficulties             |
| • Motion sickness                  |
| • Auditory processing disorder     |
| • Memory loss                      |
| • Insomnia                         |
• Sugar craving
• Poor morning appetite
• Frequent infections
• Allergies
• Impotence
• Sweet breath and body odour

Igor Bondarenko PhD
“It may well be a P450 enzyme that oxidises hemopyrrole and kryptopyrrole. 2-hydroxyhemopyrrole-2-one is either an intense chelator of Vit B6 and zinc, or it facilitates their urinary excretion, or both”.

Igor Bondarenko PhD
“Interestingly, PBG is broken down by a deaminase, and the release of ammonia from it may presume more P-5-P for utilising the formed ammonia in, for example, glutamine synthetase-catalysed reaction”.

• Paranoia
• Seizure
• Intolerance to bright light
Pyroluria and Gluten Sensitivity
It is not uncommon for those with this condition to have gluten and casein sensitivity. This condition is more prevalent in many of the same populations that we see increased prevalence of gluten sensitivity. It can cause wide ranging symptoms.

Vitamin B12
1. Hydroxycobalamin
2. Adenosylcobalamin
3. Methylcobalamin
Vitamin B₁₂ is a water soluble vitamin with a key role in the normal functioning of the brain and nervous system, and for the formation and maturation of red blood cells.

It is normally involved in the metabolism of every cell of the human body, especially affecting DNA synthesis and regulation, but also fatty acid synthesis (especially odd chain fatty acids) and energy production.
Only bacteria have the enzymes required for its synthesis, although many foods are a natural source of B\textsubscript{12} because of bacterial symbiosis and usually produce hydroxocobalamin, but conversion between different forms of the vitamin can be accomplished in the human body.

Vitamin B\textsubscript{12} was discovered from its relationship to the disease pernicious anemia, which is an autoimmune disease in which parietal cells of the stomach responsible for secreting intrinsic factor are destroyed, the same cells responsible for secreting acid in the stomach.

Intrinsic factor is crucial for the normal absorption of B\textsubscript{12}, so a lack of intrinsic factor, as seen in pernicious anemia, causes a vitamin B\textsubscript{12} deficiency. Many other subtler kinds of vitamin B\textsubscript{12} deficiency and their biochemical effects have since been elucidated.
Methionine synthase, an enzyme that uses MeB₁₂ to catalyze the conversion of homocysteine back into methionine. This functionality is lost in vitamin B₁₂ deficiency, and can be measured clinically as an increased homocysteine level.

Myelin damage resulting from B₁₂ deficiency, even in the presence of adequate folate and methionine, is more specifically and clearly a vitamin deficiency problem. It has been connected to B₁₂ most directly by reactions related to MUT, which is required to convert methylmalonyl coenzyme A into succinyl CoA.
Failure of this second reaction to occur results in elevated levels of MMA, a myelin destabilizer. Excessive MMA will prevent normal fatty acid synthesis, or it will be incorporated into fatty acid itself rather than normal malonic acid.

If this abnormal fatty acid subsequently is incorporated into myelin, the resulting myelin will be too fragile, and demyelination will occur.

Methylmalonyl CoA is formed as an intermediate in the catabolism of valine and by the carboxylation of propionyl CoA arising in the catabolism of isoleucine, cholesterol and odd numbered fatty acids or directly from propionate a major product of microbial fermentation in the rumen.
Energy production
1. Glycolysis
2. Krebs’ Cycle
3. Electron transport

Modulated by T4 and T3

Intermediate of the Krebs’ cycle

Adenosylcobalamin (B12 coenzyme)

Methylmalonic acid

D-MethylCoA

L-MethylmalonylCoA

SuccinylCoA

Methylmalonic acid
Folate deficiency limits cell division, erythropoiesis, production of red blood cells, is hindered and leads to megaloblastic anemia, which is characterized by large immature red blood cells.

This pathology results from persistently thwarted attempts at normal DNA replication, DNA repair, and cell division, and produces abnormally large red cells called megaloblasts (and hypersegmented neutrophils) with abundant cytoplasm capable of RNA and protein synthesis, but with clumping and fragmentation of nuclear chromatin.

Folic acid is itself not biologically active, but its biological importance is due to tetrahydrofolate and other derivatives after its conversion to dihydrofolate in the liver.

H4Folate Tetrahydrofolate
CHH4Folate Methenyl tetrahydro folate
CH2H4Folate Methylene tetrahydro folate
CH3H4Folate Methyl tetrahydro folate
Heart Muscle Function

Cardiac muscle like skeletal is striated but exhibits intrinsic rhythmicity. In cardiac muscle the sarcoplasmic reticulum is less extensive and thus the intracellular supply of Ca++ for contraction is less, thus relying upon extracellular Ca++ for contraction. If deprived of extracellular Ca++ the heart ceases to beat within 1 minute.
Ca** enters muscle cells through voltage gated Ca** specific channels opening during depolarisation induced by spread of the cardiac action potential and closing when the action potential declines. Activation of protein kinase enzymes (Mg** dependant) modulate intracellular Ca** entry.

Ca** entry requires optimal cell membrane integrity and the presence of trans fatty acids or oxidised fatty acids will inhibit this. Thus the necessity for good organic cold pressed unsaturated oils such as flax seed etc. Pyridoxal-5-phosphate (Vitamin B6) is important in the stabilization of cell membranes.

Optimizing Cardiac Function
Magnesium –phosphate, chloride, sulphate, citrate
Calcium – lactate, chloride, sulphate, citrate
Pyridoxal-5-phosphate
Heart tissue extract, Hawthorn
Vitamin E – wheatgerm oil
Vitamin C – SMART C
Essential fatty acids / Lecithin
Oxygen into the Mitochondria and where it Functions

Energy pathway

Glycolysis

Citric Acid Cycle

Electron transport or Oxidative phosphorylation pathway
Lactic acid
Tissues that function under hypoxic conditions produce lactic acid.

D/L Lactic acid – RED body types
L.Lactic acid – GREEN body types
D.Lactic acid - BLUE body types
The Lactic Acid (Cori Cycle)

Under anaerobic conditions NADH cannot be reoxidized through the respiratory chain to oxygen. Pyruvate is reduced by NADH to lactate catalysed by lactate dehydrogenase. There are three different specific isoenzymes of lactate dehydrogenase that have clinical significance.

The re-oxidation of NADH via lactate formation allows glycolysis to proceed in the absence of oxygen by regenerating sufficient NAD for another cycle of the reaction catalysed by glyceraldehyde-3-phosphate dehydrogenase.
Some tissues derive much of their energy from glycolysis and produce lactate –
Erythrocytes  Brain
GI tract   Renal medulla
Retina  Skin
The liver, kidney and heart usually take up lactate and oxidize it but will produce it under hypoxic conditions

Coenzyme Q10

Co-enzyme Q10
Coenzyme Q10 (ubiquinone) is a lipid-soluble compound that occurs in all kinds of cell membranes in the human body. It has several biochemical functions:

- it is indispensable for producing energy in the cells in the form of ATP
- it is an essential fat soluble antioxidant
- it helps regenerate other antioxidants esp Vit E
- it stimulates cell growth and inhibits cell death
| It is beneficial for the prevention of cell damage in hypoxia, especially in the cardiac muscle. It has been used for the protection of myocardium in different cardiovascular disorders, such as angina pectoris, hypertension, arrhythmia and congestive heart failure. |
| It has been proven to have anti-tumour and immune system enhancing properties when tested in animals. |
| Genetic mutations, ageing, cancer and statin-type drugs can cause a decrease in the levels of coenzyme Q10 in tissues and blood. |
| Low ratio of coenzyme Q10 to low-density lipoprotein (LDL) cholesterol is a strong indicator of risk of atherosclerosis (clogging of the arteries) |
Best sources mg / Kg
- Beef, pork and chicken heart 113+
- Beef, pork and chicken liver 50+
- Sardines and red flesh fish 50+
- Soy, olive, grape seed oils 50+
- Peanuts, sesame, pistachio, hazelnuts 20+
- Parsley 20+
- Avocado 10+

Toxins

Toxic metals – Black walnut
- Coriander herb
- Coriander spice
- Lemon balm
- Yarrow
- Other spices
Toxins

Chemicals - Coriander spice
NAC
Lemon balm
Yarrow
Other spices

Toxins

Radiation - Coriander spice
Turmeric
Yarrow

Infectious Diseases
Bacteria –
Ginger
Ionic silver
Mannose
Thiamine / Silver

Virus –
Astragalus
Echinacea
Selenium

Parasites –
AP formula
Cloves
Coriander seed
RED, GREEN, BLUE
Spice mixes

Fungi –
Coconut oil
Coriander
Other spices
Pau D’arco
AF Creaqm locally

Probiotics
Bifidobacteria Bifidus
Lactobacillus Acidophilus
Lactobacillus Bulgaricus
Lactobacillus Casei
Lactobacillus Plantarium
Lactobacillus Rhamnosus
Smart Probiotic

Muscle Oxygen Requirements
during Exercise

A muscle requires approximately 50x more oxygen per minute when active than when at rest. This is achieved by
1. An increase in lung blood flow and cardiac output from 5 litres per minute to 50 litres per minute. This gives an increase of 6x.
2. Redistribution of the blood flow to the active muscles. This gives an increase of 3x.
3. More oxygen is extracted from every 100ml of blood passing through the muscle as a result of lowered oxygen tension in the muscles. This gives an increase of 3x.
Total increase is thus 6x3x3=54

ATP in muscle during exercise

- Glucose - gluconeogenesis
- Creatine phosphate
- Muscle glycogen - glycogenolysis
- Beta oxidation – burning fats
- Amino acids

<table>
<thead>
<tr>
<th></th>
<th>Type 1 Slow twitch</th>
<th>Type 2 Fast twitch</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myosin ATPase</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Energy utilization</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Mitochondria</td>
<td>Many</td>
<td>Low</td>
</tr>
<tr>
<td>Colour</td>
<td>Red</td>
<td>White</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Contraction rate</td>
<td>Slow</td>
<td>Fast</td>
</tr>
<tr>
<td>Duration</td>
<td>Prolonged</td>
<td>Short</td>
</tr>
</tbody>
</table>
### Anaerobic Sprinter vs. Aerobic Marathon

<table>
<thead>
<tr>
<th>Anaerobic Sprinter</th>
<th>Aerobic Marathon</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 2 (glycolytic) fibres are used predominantly</td>
<td>Type 1 (oxidative) fibres are used predominantly</td>
</tr>
<tr>
<td>Creatine phosphate is the major energy source during the first 4-5 seconds</td>
<td>ATP is the major energy source throughout</td>
</tr>
<tr>
<td>Glucose derived from muscle glycogen and metabolised by anaerobic glycolysis is the major fuel source</td>
<td>Blood glucose and free fatty acids are the major fuel sources</td>
</tr>
<tr>
<td>Muscle glycogen is rapidly depleted</td>
<td>Muscle glycogen is slowly depleted</td>
</tr>
</tbody>
</table>

### Sources of fuel during exercise

<table>
<thead>
<tr>
<th>ANAEROBIC</th>
<th>AEROBIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type II (glycolytic white) fibres are used predominantly</td>
<td>Type I (oxidative red) fibres are used predominantly</td>
</tr>
<tr>
<td>1-5 seconds: Creatine phosphate is the major energy source</td>
<td>First 4 minutes: blood glucose</td>
</tr>
<tr>
<td>5-10 seconds: Glucose derived from muscle glycogen is metabolised by anaerobic glycolysis leading to lactic acid formation</td>
<td>4-18 minutes: liver glycogen</td>
</tr>
<tr>
<td>Rapid depletion of muscle glycogen</td>
<td>18-70 minutes: muscle glycogen</td>
</tr>
</tbody>
</table>

### Glycolysis from Glucose

**AEROBIC**

<table>
<thead>
<tr>
<th>Glycolysis from Glucose ATP YIELD</th>
<th>Glycolysis from Glucose ATP YIELD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phosphofructokinase</td>
<td>2 ATP</td>
</tr>
<tr>
<td>Pyruvate kinase</td>
<td>2 ATP</td>
</tr>
<tr>
<td>Glyceraldehyde-3-phosphate dehydrogenase</td>
<td>1 ATP</td>
</tr>
<tr>
<td>Total 10</td>
<td>2 ATP</td>
</tr>
</tbody>
</table>

### Krebs Cycle

<table>
<thead>
<tr>
<th>Krebs Cycle ATP Yield</th>
<th>Krebs Cycle ATP Yield</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinyl CoA synthetase</td>
<td>2 ATP</td>
</tr>
<tr>
<td>Total 2</td>
<td>2 ATP</td>
</tr>
</tbody>
</table>

### Oxidative Phosphorylation

<table>
<thead>
<tr>
<th>Oxidative Phosphorylation ATP Yield</th>
</tr>
</thead>
<tbody>
<tr>
<td>NADH + H+</td>
</tr>
<tr>
<td>FADH2</td>
</tr>
<tr>
<td>Total 24</td>
</tr>
</tbody>
</table>

### Glycolysis from Glycogen

<table>
<thead>
<tr>
<th>Glycolysis from Glycogen ATP Yield</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plus 2 ATP to lactic acid</td>
</tr>
<tr>
<td>Total 2</td>
</tr>
</tbody>
</table>

### KREBS CYCLE ATP YIELD

<table>
<thead>
<tr>
<th>Total 2</th>
<th>2 ATP</th>
</tr>
</thead>
</table>

### OXIDATIVE PHOSPHORYLATION ATP YIELD

<table>
<thead>
<tr>
<th>Total 24</th>
</tr>
</thead>
</table>

### GLYCOLYSIS FROM GLYCOGEN ATP YIELD

<table>
<thead>
<tr>
<th>Total 2</th>
</tr>
</thead>
</table>
Anaerobic exercise
First fuel source is Creatine phosphate 4-5 seconds

Creatine phosphate
\[ \text{creatine phosphokinase} \rightarrow \text{ADP} \rightarrow \text{ATP} \]

Creatine phosphate (Arginine, Glycine, Methionine ATP)

Arginine

\[ \text{arginine transaminase} \rightarrow \text{Glycine} \]

Glycine

\[ \text{B6} \rightarrow \text{Ornithine} \]

Ornithine

Glycocyamine (guanidoacetate)

\[ \text{ATP} \rightarrow \text{methyltransferase} \rightarrow \text{SAM} \]

SAM

\[ \text{B3} \rightarrow \text{LIVER} \rightarrow \text{Homocysteine} + \text{Adenosine} \]

Creatinine in urine

Creatine phosphate

Second is anaerobic glycolysis using muscle glycogen

Muscle Glycogen

\[ \text{Adrenalin, B6, Ca, Mg} \]

Glucose-1-phosphate

\[ \text{Mg} \]

Glucose-6-phosphate

\[ \text{Mg, B3, Zn,} \]

1,3-Bisphosphoglycerate

\[ \text{Mg, K} \]

Phosphoenolpyruvate
Protocol for exercise testing
1. Anaerobic challenge contract muscle 2x second 10x. Muscle weakens.
2. Mg-ADP weakens. From weakness challenge
   Creatine phosphate
   1,3-Bisphosphoglycerate
   Phosphoenolpyruvate

Aerobic Exercise Fuel
1. Glucose – Blood, Liver Glycogen, Gluconeogenesis (of Amino acids and odd numbered Fatty acids)
2. Muscle Glycogen
3. Fatty acids

Aerobic exercise
First is blood glucose

Diet
Liver glycogen
Blood glucose
Lactate muscle - liver (Cori cycle)
Gluconeogenesis from amino acids
Protocol for exercise testing
1. Aerobic challenge contract a strong aerobic muscle 1x second 10x. Muscle weakens.
2. Mg-ADP weakens. From weakness challenge
   Glucose – Liver glycogen
   Gluconeogenesis
   Muscle glycogen
   Acetyl CoA – beta oxidation

Second is Muscle Glycogen

Muscle Glycogen
   Adrenalin, B6, Ca, Mg
Glucose-1-phosphate
   Mg
Glucose-6-phosphate
   Mg, B3, Zn, K
Lactate
   B3
Pyruvate

Third is Beta Oxidation
TL Pinch fat
Challenge against
   T4, T3, Adrenalin
   ATP, Mag, CoA
   Carnitine
   FAD, NAD, H2O
   O2 – Iron, B12
   Adenosylcobalamin
   CO2
Each molecule of fatty acid requires one molecule of FAD, one molecule of NAD+, one molecule of CoA, and one molecule of H₂O to oxidise each time to acetyl CoA.
Optimal products
Dosing
Timing

Optimal products
Must remain strong to cross therapy localisation to
1. CV22
2. GV21
3. GV28

Right brain activity – Humming
Left brain activity - Mathematics

Dosing
Amount of liquid or capsules that strengthen weak muscle(s)
Timing
Cross therapy localise to the alarm points for remaining strong. Those that remain strong are the optimal times of dosing.

Usually St, SI, Cx or TW

Alarm points

Timing
1. Amino acids 15 minutes before breakfast
2. Vitamins and Minerals with meals
3. Fatty acids with evening meal
4. Probiotics, CoQ10, Folic acid last thing at night.
5. Herbs and spices between or before meals.
Patient protocol
1. Test body type colour
2. Cross extensor reflexes
3. Conscious / subconscious emotion balance
4. Test B&E points
5. Assess genotype and phenotype meridians
6. Start with phenotype meridian and challenge with EID

7. Test and treat positive eye positions.
8. Tap B&E point to assess if anything more required
9. Using genotype meridian challenge with EID. Usually hypoxia. If positive challenge against O2. Then Phospholipid, Co-Q10 and Hemoglobin vials.

10. If phospholipid challenge against culinary oils.
11. If Co-enzyme Q10 challenge against oil based Co-Q10.
12. If Hemoglobin challenge
   ALA – Adenosylcobalamin, P5P
   PBG- CH2H4Folate
   UBG III – P5P, H4Biopterin
   CPG III – P5P
   PP IX-P5P
If only haemoglobin strengthen - Iron, Folate

13 Challenge with MAUVE acetate. Test for Lutein

Products
All products, laminates, test kits, biomarker, DVDs, Online video education available at www.epigenetics-international.com
sales@epigenetics-international.com