

ICAK Nutrition

Module 13

**Nutritional Management of
Common Cardiovascular
Conditions**

A Phonocardiogram is a recording of the sounds and murmurs made by the heart with the help of a digital stethoscope, of the sounds made by the heart during a cardiac cycle. The sounds are a result from vibrations created by closure of the heart valves.

There are at least two: the first when the **atrio-ventricular valves** close at the beginning of systole and the second when the **aortic and pulmonary valves** close at the end of systole. It allows the detection of sub-audible sounds and murmurs, and makes a permanent record of these events.

80% of all physicians die of heart disease.

80% of cardiac conditions are muscular – primarily hypoxia

10% valvular

6% nervous

4% coronary

More people are affected and die with heart disease than any other illness

Functional Testing

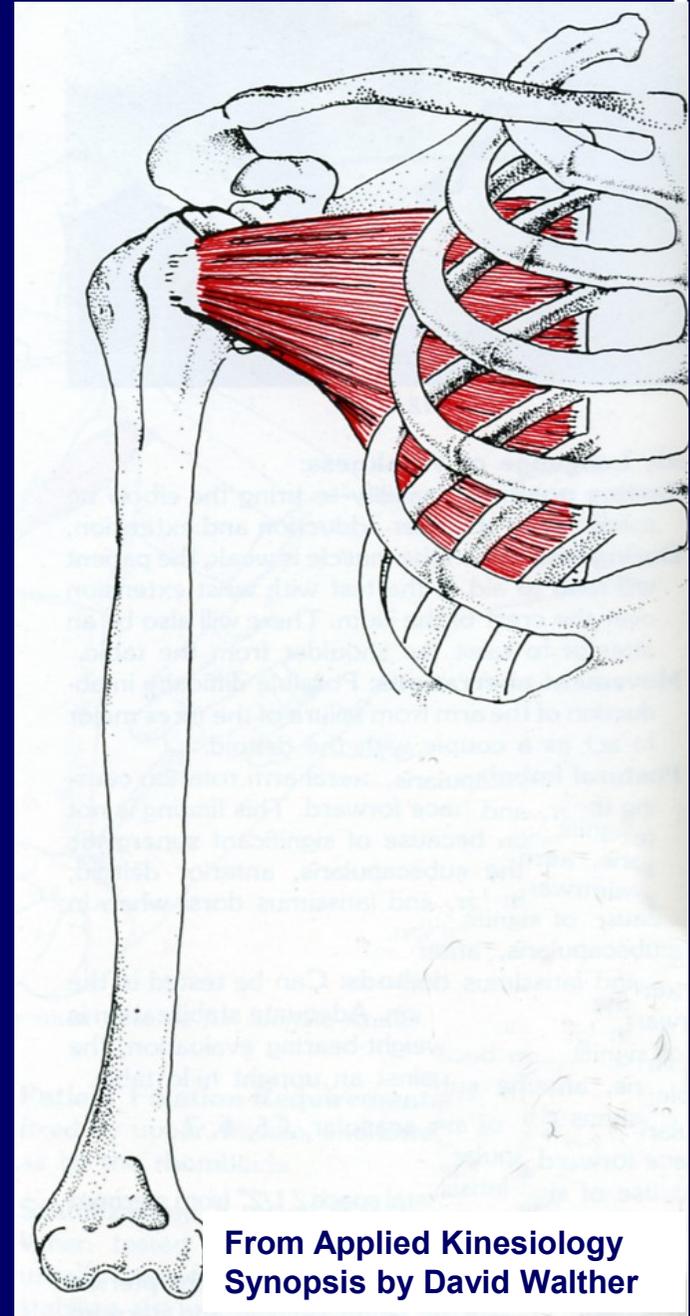
Only one muscle – the **SUBSCAPULARIS** is associated in AK with the heart

Subscapularis

Origin –

Subscapular fossa

**Insertion – Lesser
tuberosity of the
humerus and fibrous
capsule.**



From Applied Kinesiology
Synopsis by David Walther

Function – Medial rotation of the humerus

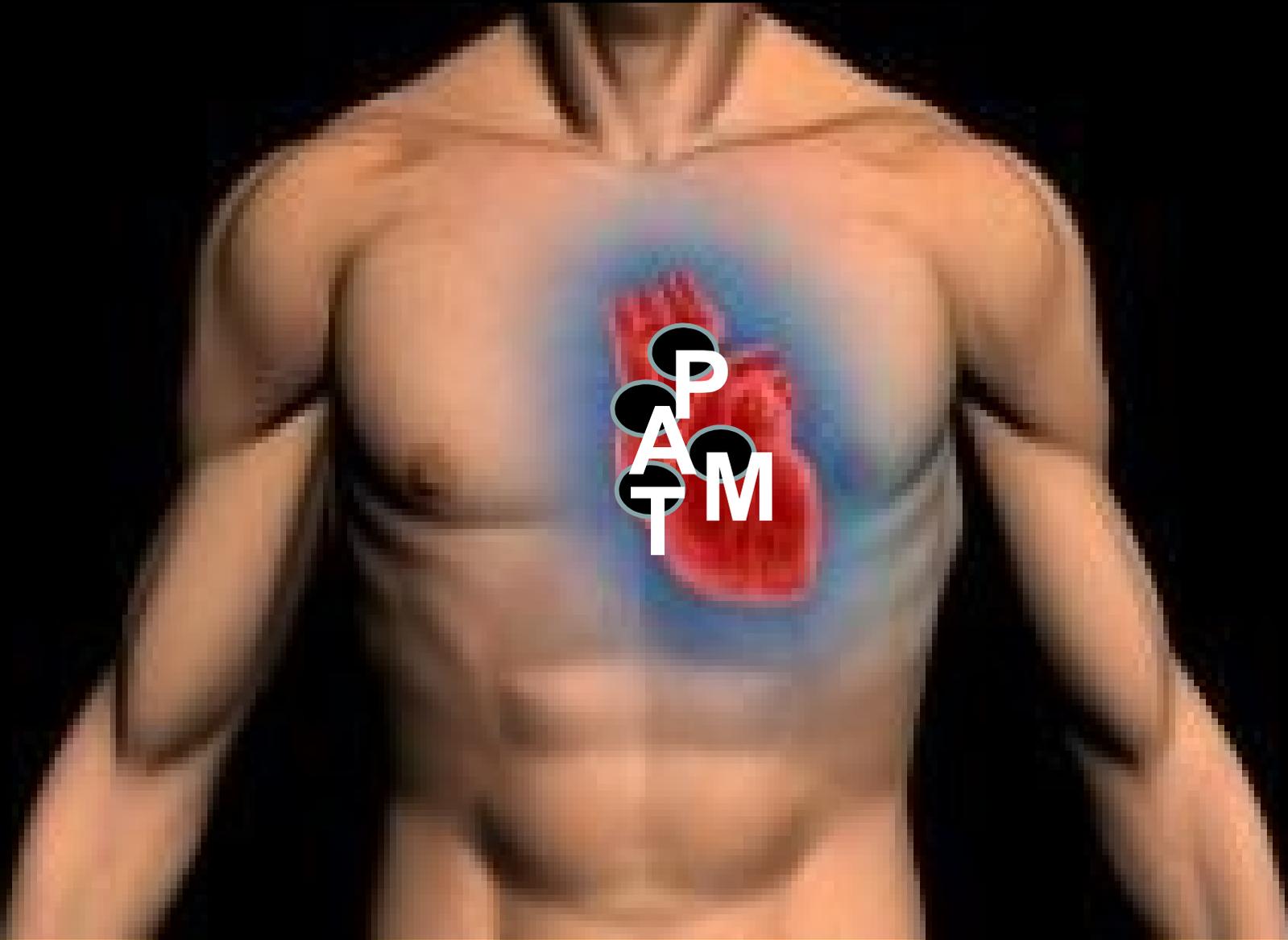
Nerve supply – Upper and lower subscapular nerve C5, 6

Meridian association – Heart

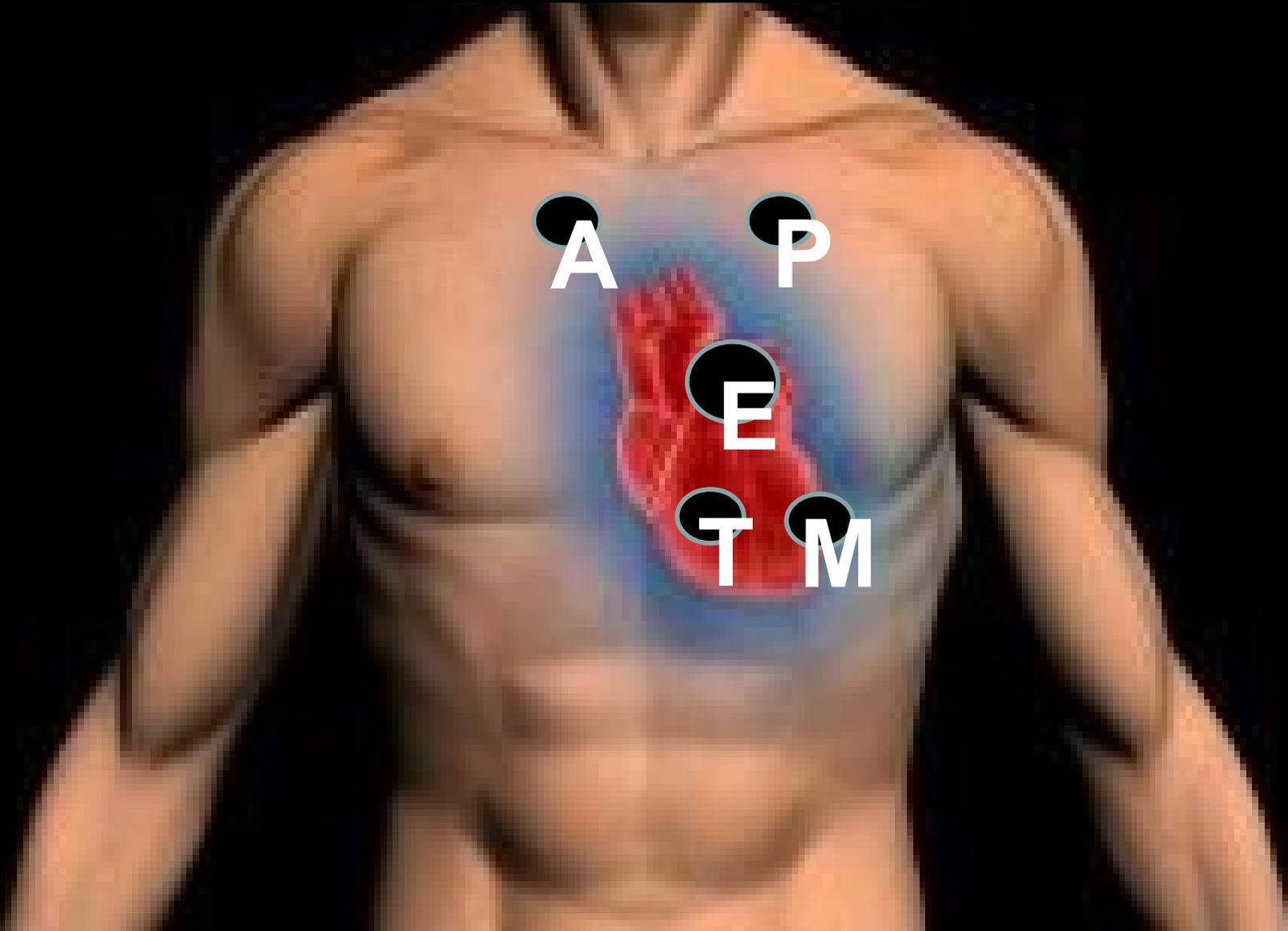
The aortic area, pulmonic area, tricuspid area and mitral area are areas on the surface of the chest where the heart is auscultated. Heart sounds result from reverberation within the blood associated with the sudden block of flow reversal by the valves closing.

Because of this, auscultation to determine function of a valve is usually **not performed** at the position of the valve, but at the position to where the sound waves reverberate.

Actual position of valves



Optimal auscultation areas



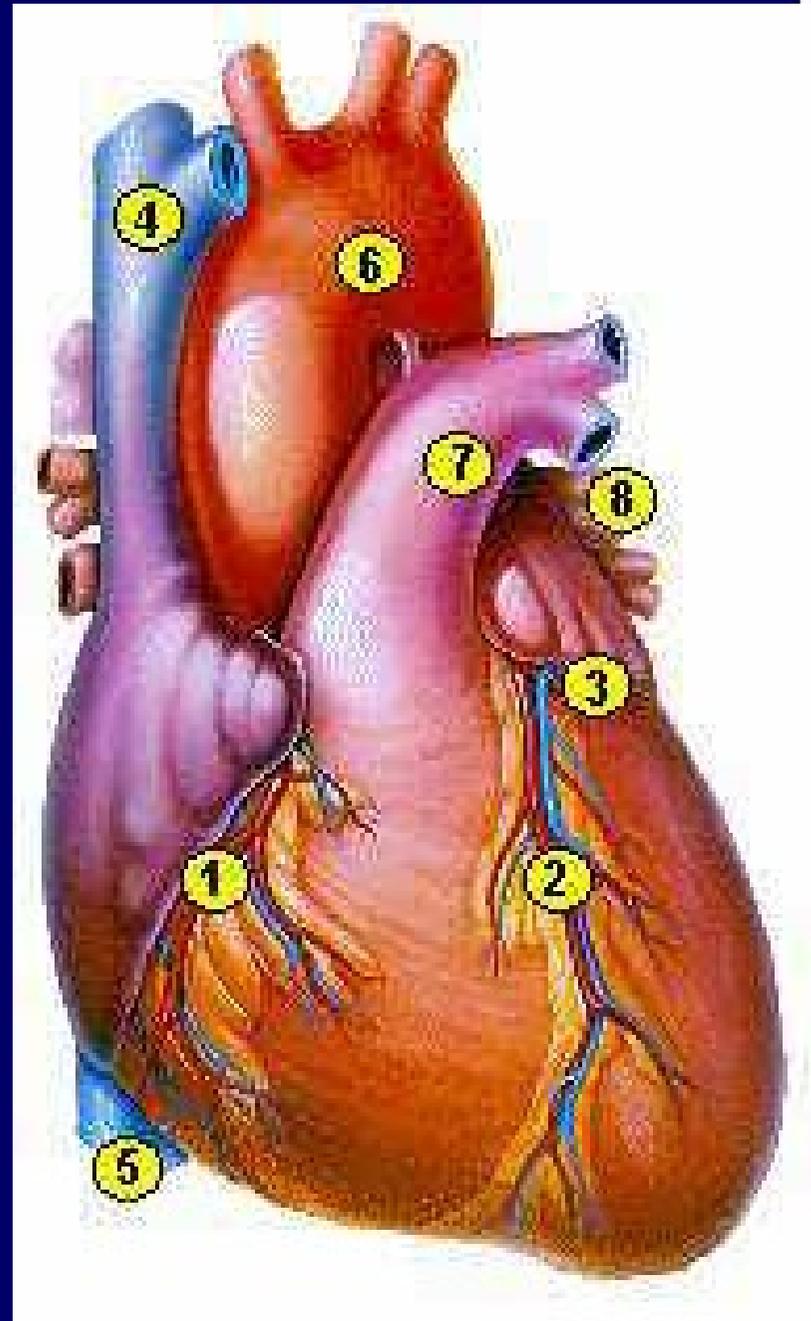
Heart valve auscultation points

Pulmonary valve (to pulmonary trunk)	left second intercostal space	left upper sternal border
Aortic valve (to aorta)	right second intercostal space	right upper sternal border
Erb's point	Left third intercostal space	medial left sternal border
Mitral valve (to left ventricle)	left fifth intercostal space	medial to left midclavicular line
Tricuspid valve (to right ventricle)	left fifth intercostal space	lower left sternal border

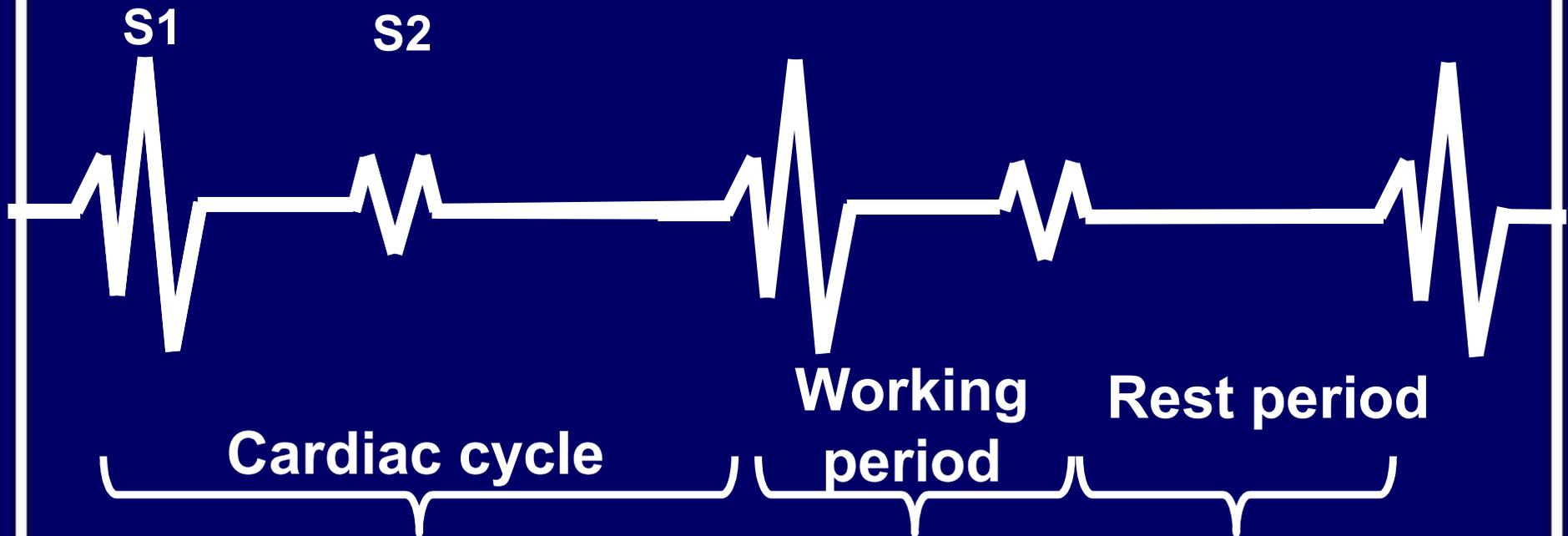
In cardiology, **Erb's point** refers to the third intercostal space on the left sternal border where **S2** is best auscultated. It is essentially the same location as what is referred to with left lower sternal border (LLSB).

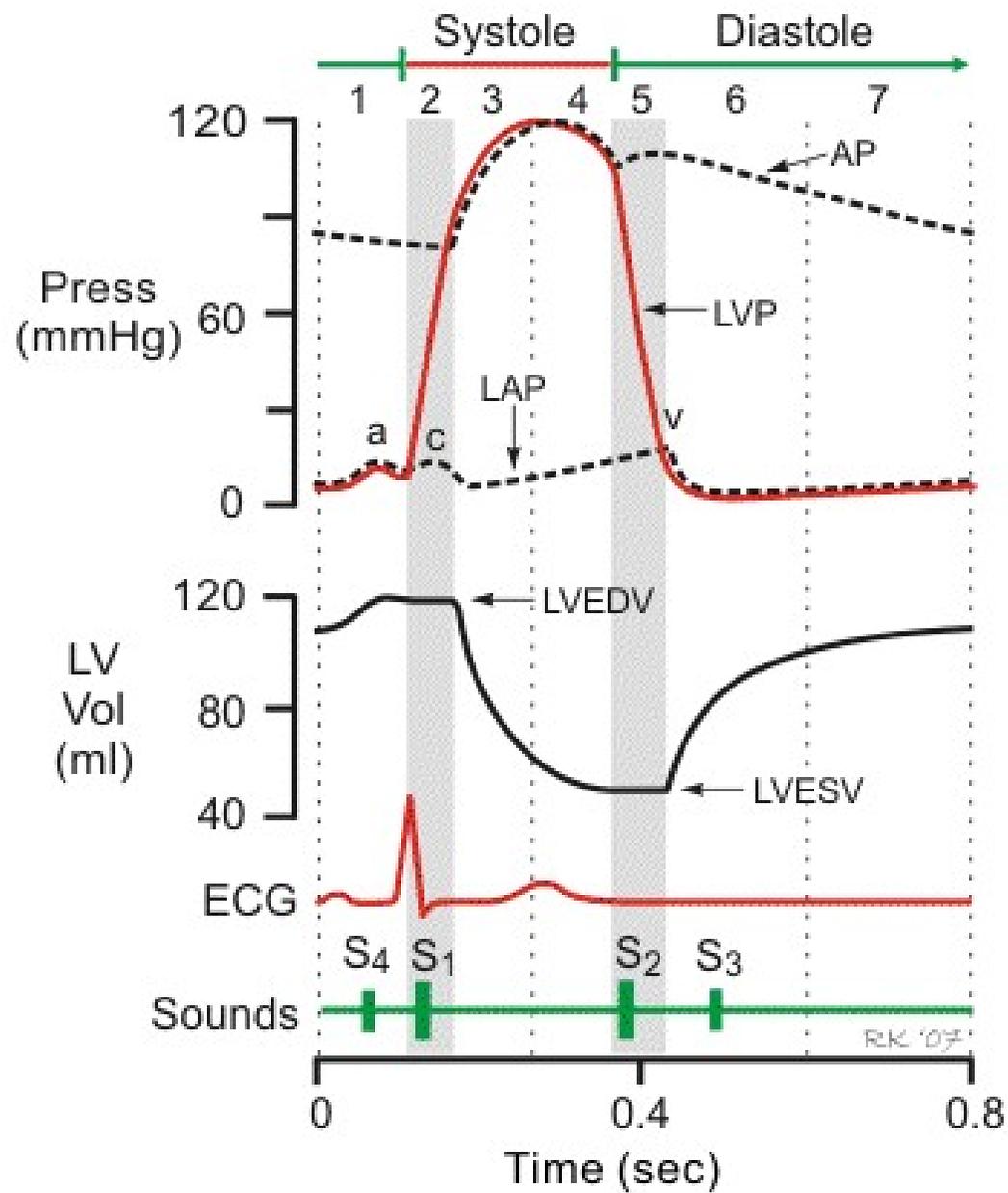
Coronary Arteries and Great Vessels

1. Right Coronary
2. Left Anterior Descending
3. Left Circumflex
4. Superior Vena Cava
5. Inferior Vena Cava
6. Aorta
7. Pulmonary Artery
8. Pulmonary Vein



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.

Trimethylamine

Trimethylamine (TMA) is an organic compound with the formula $\text{N}(\text{CH}_3)_3$. This colorless, hygroscopic, and flammable tertiary amine has a strong "fishy" odour in low concentrations and an ammonia-like odour at higher concentrations.

In humans it is synthesized exclusively by gut microbiota from dietary nutrients such as **choline and carnitine**.*

High levels of trimethylamine are associated with the development of fish odour syndrome, which arise from the foul, fishy odour of trimethylamine.*

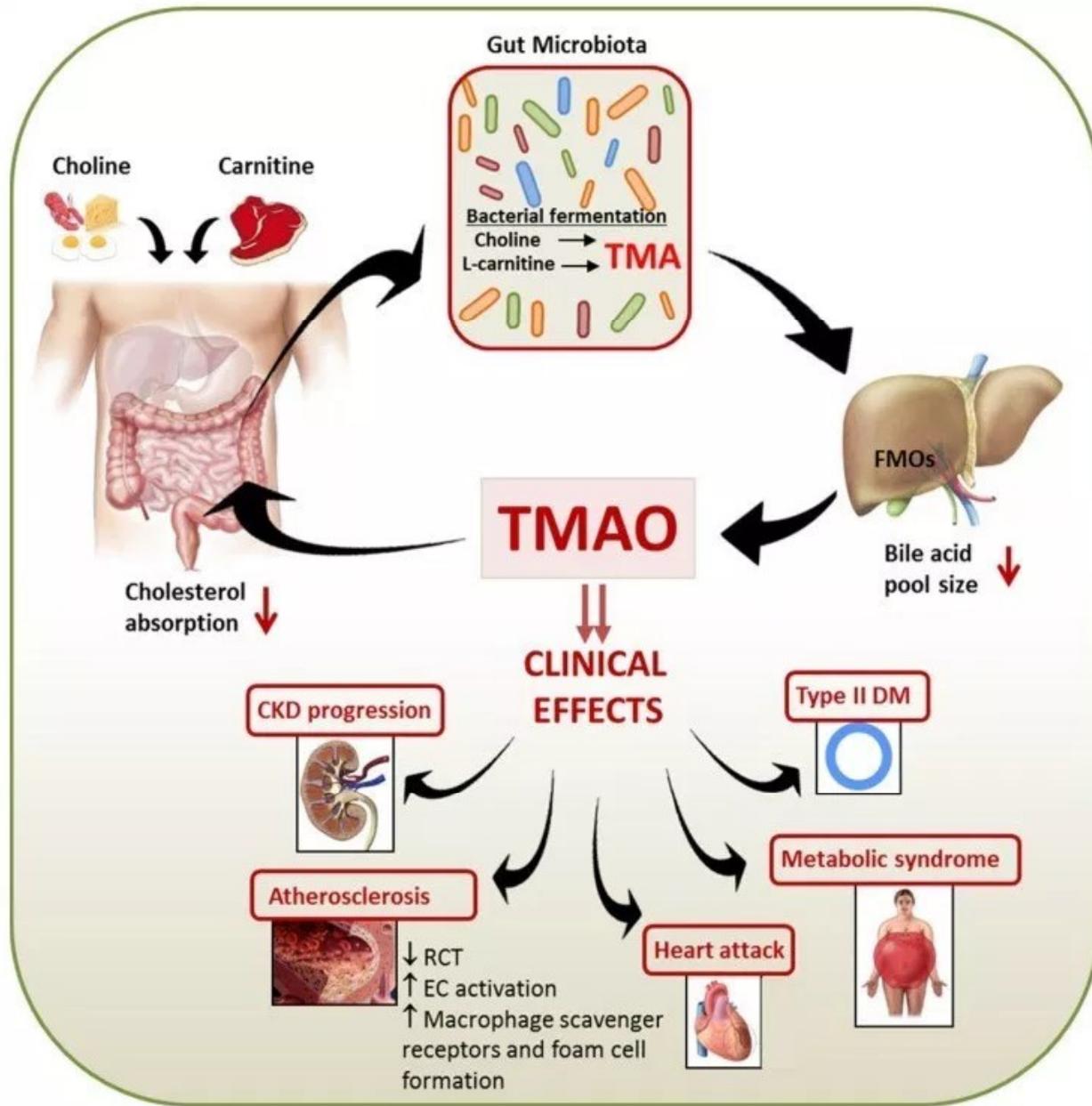
It is the substance mainly responsible for the odour often associated with **rotting fish, some infections, bad breath and can be a cause of vaginal odour due to bacterial vaginosis..**

*** Falony G, Vieira-Silva S, Raes J (2015). "Microbiology Meets Big Data: The Case of Gut Microbiota-Derived Trimethylamine". *Annu. Rev. Microbiol.* 69: 305–321.**

Trimethylaminuria is an autosomal recessive genetic disorder involving a defect in the function or expression of flavin-containing monooxygenase³ (trimethylamine monooxygenase) which results in poor trimethylamine metabolism.

Individuals with **trimethylaminuria** develop a characteristic fish odour—the smell of trimethylamine—in their sweat, urine, and breath after the consumption of choline-rich foods. (liver, eggs, wheatgerm, soybeans, scallops, salmon, chicken.) *

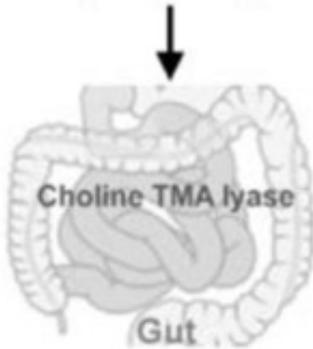
* Linus Pauling Institute » Micronutrient Information Center





Protein rich diet.

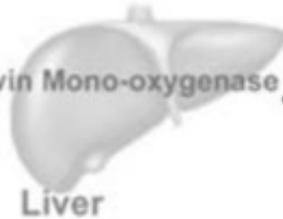
Choline, Carnitine, Betaine



Modulated by Resveratrol

Trimethylamine (TMA)

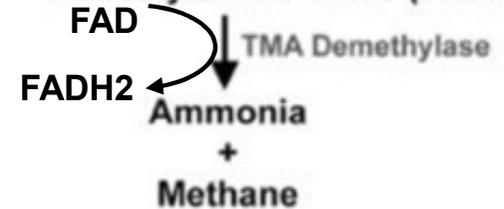
Flavin Mono-oxygenase 3



Trimethylamine oxide (TMAO)



Formaldehyde
+
Dimethylamine



Cardio-metabolic disorders

Renal Disorders

Cancer

Neurological disorders

Bacterial enzymes in red
Human enzymes in blue

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Research Article

Resveratrol Attenuates Trimethylamine-*N*-Oxide (TMAO)-Induced Atherosclerosis by Regulating TMAO Synthesis and Bile Acid Metabolism via Remodeling of the Gut Microbiota

Ming-liang Chen, Long Yi, Yong Zhang, Xi Zhou, Li Ran, Jining Yang, Jun-dong Zhu, Qian-yong Zhang, Man-tian Mi

Federico Rey, *Invited Editor*; Caroline S. Harwood, *Editor*

DOI: 10.1128/mBio.02240-15  Check for updates

The gut microbiota is found to be strongly associated with atherosclerosis (AS). Resveratrol (RSV) is a natural phytoalexin with anti-AS effects; however, its mechanisms of action remain unclear. Therefore, we sought to determine whether the anti-AS effects of RSV were related to changes in the gut microbiota. We found that RSV attenuated trimethylamine-*N*-oxide (TMAO)-induced AS in ApoE^{-/-} mice. Meanwhile, RSV decreased TMAO levels by inhibiting commensal microbial trimethylamine (TMA) production via gut microbiota remodeling in mice. Moreover, RSV increased levels of the genera *Lactobacillus* and *Bifidobacterium*, which increased the bile

Trimethylamine *N*-oxide (TMAO)
is a product of the oxidation
of trimethylamine.

**A study published in 2013,
assessing 513 adults with a
history of major adverse
cardiovascular events ---**

an average age of 68, and 69% of whom previously or currently smoke, may indicate that high levels of **TMAO** in the blood are associated with an increased risk of additional cardiovascular events.

*Tang, W.H. Wilson; Zeneng Wang; Bruce S. Levison; Robert A. Koeth; Earl B. Britt; Xiaoming Fu; Yuping Wu; Stanley L. Hazen (April 25, 2013).

The concentration of **TMAO** in the blood increases after consuming foods containing carnitine or lecithin if the bacteria that convert those substances to TMAO are present in the gut. *

*Gina Kolata (April 24, 2013). "Eggs, Too, May Provoke Bacteria to Raise Heart Risk". *The New York Times*. Retrieved April 25, 2013.

High concentrations of **carnitine** are found in red meat, some energy drinks, and some dietary supplements; **lecithin** is found in soy, eggs*, as an ingredient in processed food and is sold as a dietary supplement.

*Gina Kolata (April 24, 2013). "Eggs, Too, May Provoke Bacteria to Raise Heart Risk". *The New York Times*. Retrieved April 25, 2013.

Some types of normal gut bacteria (e.g. species of *Acinetobacter*) in the human microbiome convert dietary carnitine to TMAO. TMAO alters cholesterol metabolism in the intestines, in the liver, and in artery walls.*

*Hazen, Stanley. "New Research On Red Meat And Heart Disease". *The Diane Rehm Show (Transcript)*. WAMU 88.5 American University Radio. Retrieved 10 April 2013.

In the presence of **TMAO**, there is increased deposition of cholesterol in, and decreased removal of cholesterol from peripheral cells such as those in artery walls.*

*Hazen, Stanley. "New Research On Red Meat And Heart Disease". *The Diane Rehm Show (Transcript)*. WAMU 88.5 American University Radio. Retrieved 10 April 2013.

It has been suggested that **TMAO** may be involved in the regulation of arterial blood pressure and etiology of hypertension and thrombosis (blood clots) in atherosclerotic disease.*

*Tilg, Herbert (2016-06-22). "A Gut Feeling about Thrombosis". *New England Journal of Medicine*. 374 (25): 2494–2496.

A 2017 meta-analysis found higher circulating **TMAO was associated with 23% higher risk of cardiovascular events and a 55% higher risk of mortality.***

***Qi, Jiaqian; You, Tao; Li, Jing; Pan, Tingting; Xiang, Li; Han, Yue; Zhu, Li (2018). "Circulating trimethylamine N-oxide and the risk of cardiovascular diseases: a systematic review and meta-analysis of 11 prospective cohort studies". *Journal of Cellular and Molecular Medicine*. 22 (1): 185–194.**

Right common carotid artery

Right internal jugular vein

Right brachial artery

Right renal vein

Inferior vena cava

Right common iliac artery

Right external iliac vein

Right femoral artery

Left common carotid artery

Left internal jugular vein

Arch of aorta

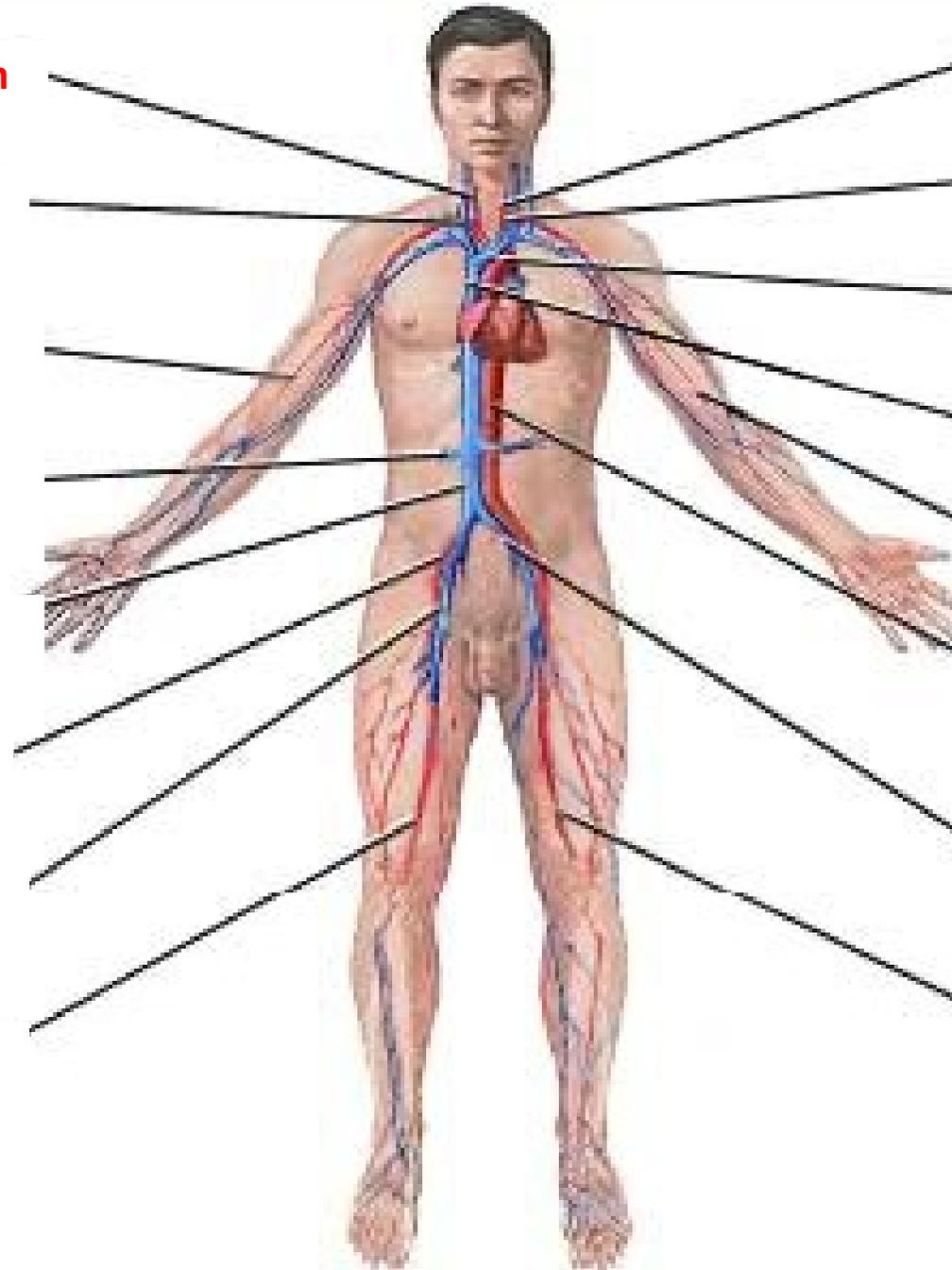
Superior vena cava

Left brachial artery

Abdominal aortic artery

Left common iliac artery

Left femoral artery

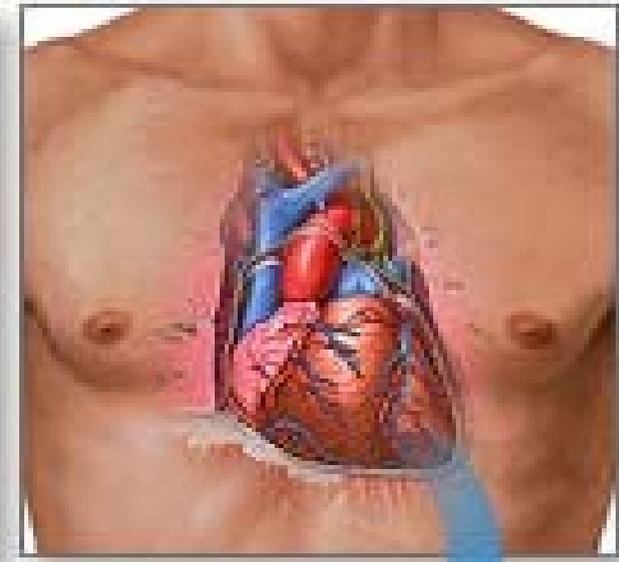




Urine test



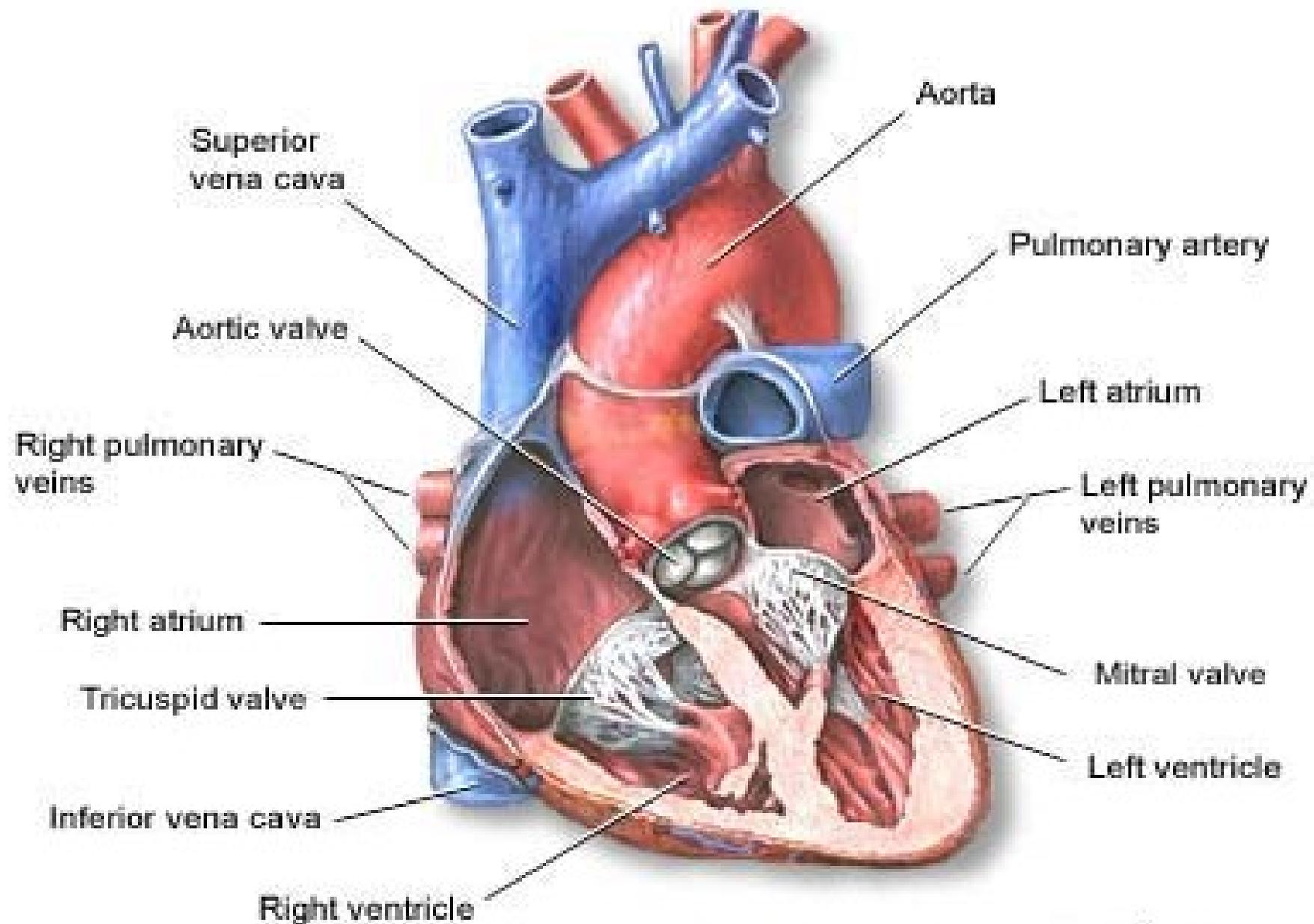
Blood test



ECG reading

BLOOD PRESSURE
and
HEART RATE

$$\text{Flow} = \frac{\text{Difference in pressure}}{\text{Resistance}}$$



Hypertensive Medications

Diuretics

Vasodilators

ACE inhibitors

Angiotensin antagonists

Beta Blockers

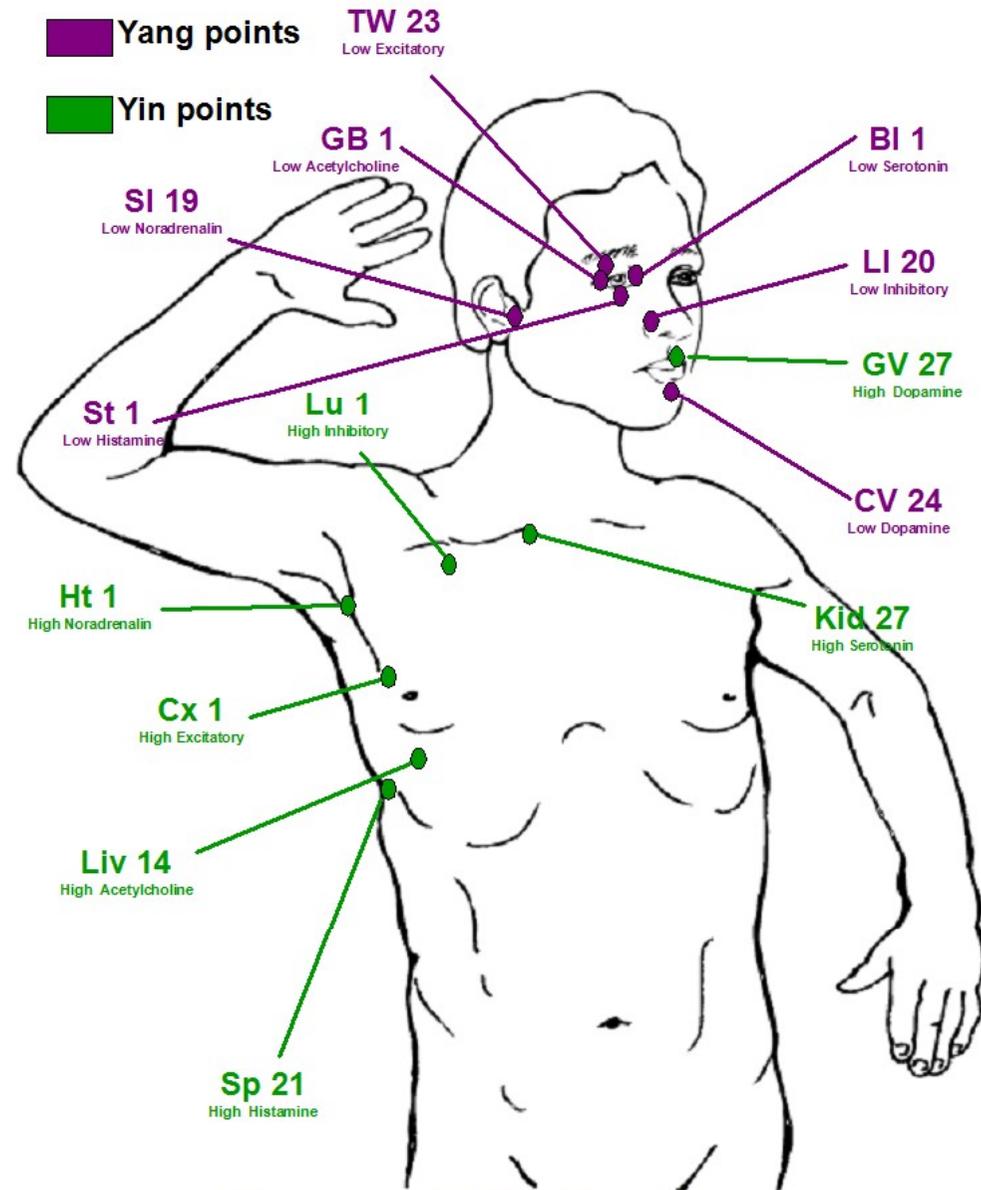
Alpha Blockers

Calcium channel blockers

Nervous system inhibitors

**Yang points
begin or end
on the face.**

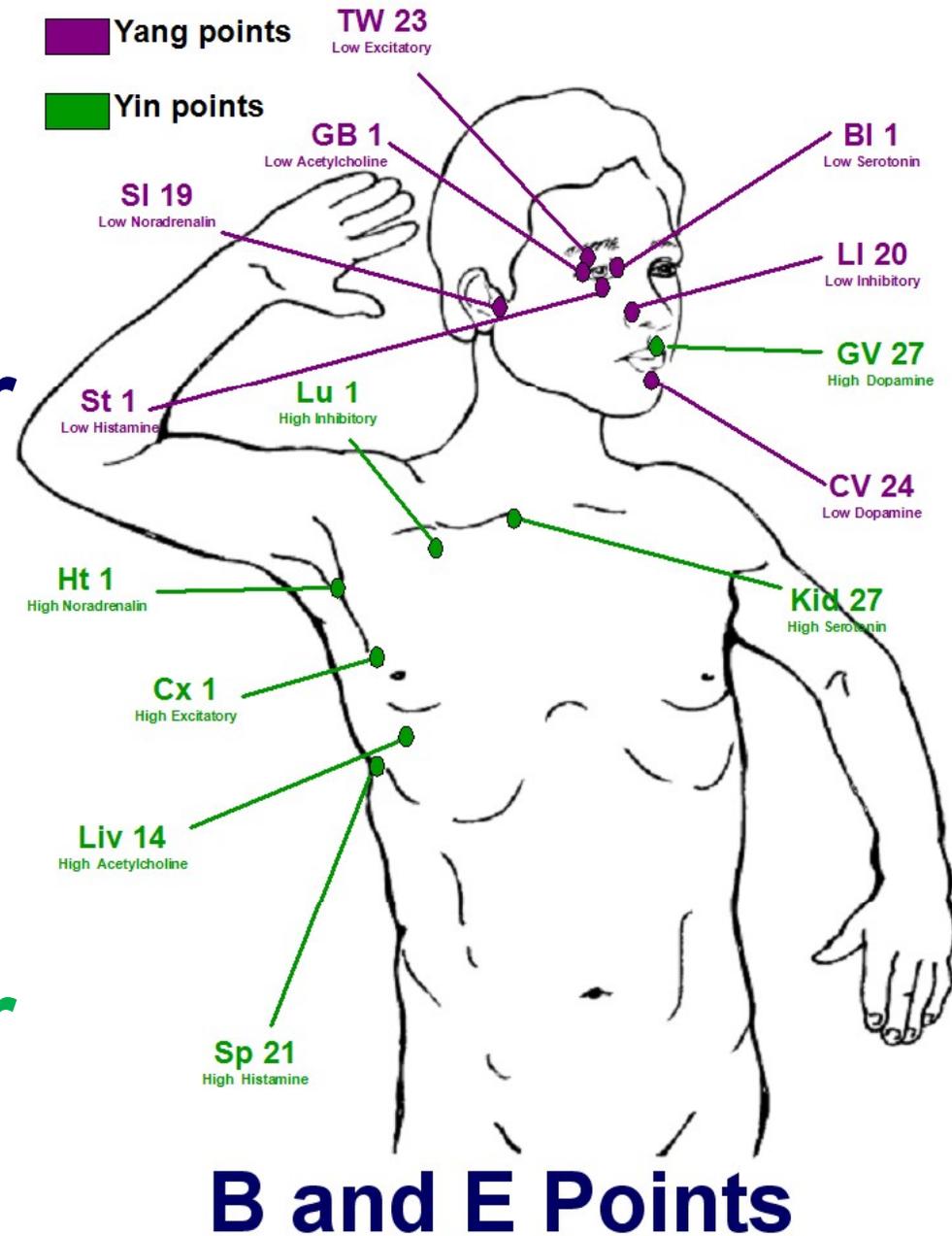
**Yin points
begin or end
on the trunk.**



B and E Points

**Yang points
indicate
neurotransmitter
deficiencies.**

**Yin points
indicate
neurotransmitter
excesses**



B and E Points

Acetylcholine

CHOLINE

Pyruvate

pyruvate dehydrogenase

3p 374nm

4q 376nm

23X 400nm

Vit B1

Vit B2

Vit B3

Vit B5

α -Lipoic acid

Acetyl CoA

choline acetyltransferase

10q 383nm

K, Br, Cl, I, NaSO4

Inhibited by
atropine, ethanol,
Cd, Hg,

CoA

ACETYLCHOLINE

Metabolic Pathways - <http://smpdb.ca/search>

BRENDA enzyme database -

<http://www.brenda-enzymes.org/enzyme>

ACETYLCHOLINE

Inhibited by

Chemicals – pesticides
solanine, sodium fluoride
thyme, galantamine,
huperzine
aspartame, aspartate,
phenylalanine lovastatin
melatonin , methotrexate,
phos serine, diazepam,
eugenol, insulin, limonene
Toxic metals Cd, Cu, Hg,
Sn, Radiation

Metabolic Pathways -
<http://smpdb.ca/search>
BRENDA enzyme database -
<http://www.brenda-enzymes.org/enzyme>

H₂O

acetylcholinesterase

7q 380nm

B2

B3

Mn⁺⁺

Zn⁺⁺ Cysteine Recycled

Acetate + Choline

GB – Low Acetylcholine

High heart rate (tachycardia)

High conduction rate

High atrial contractibility

High ventricular contractibility

High Blood pressure

Liver - High Acetylcholine

Slow heart rate (bradycardia)

Slow conduction rate

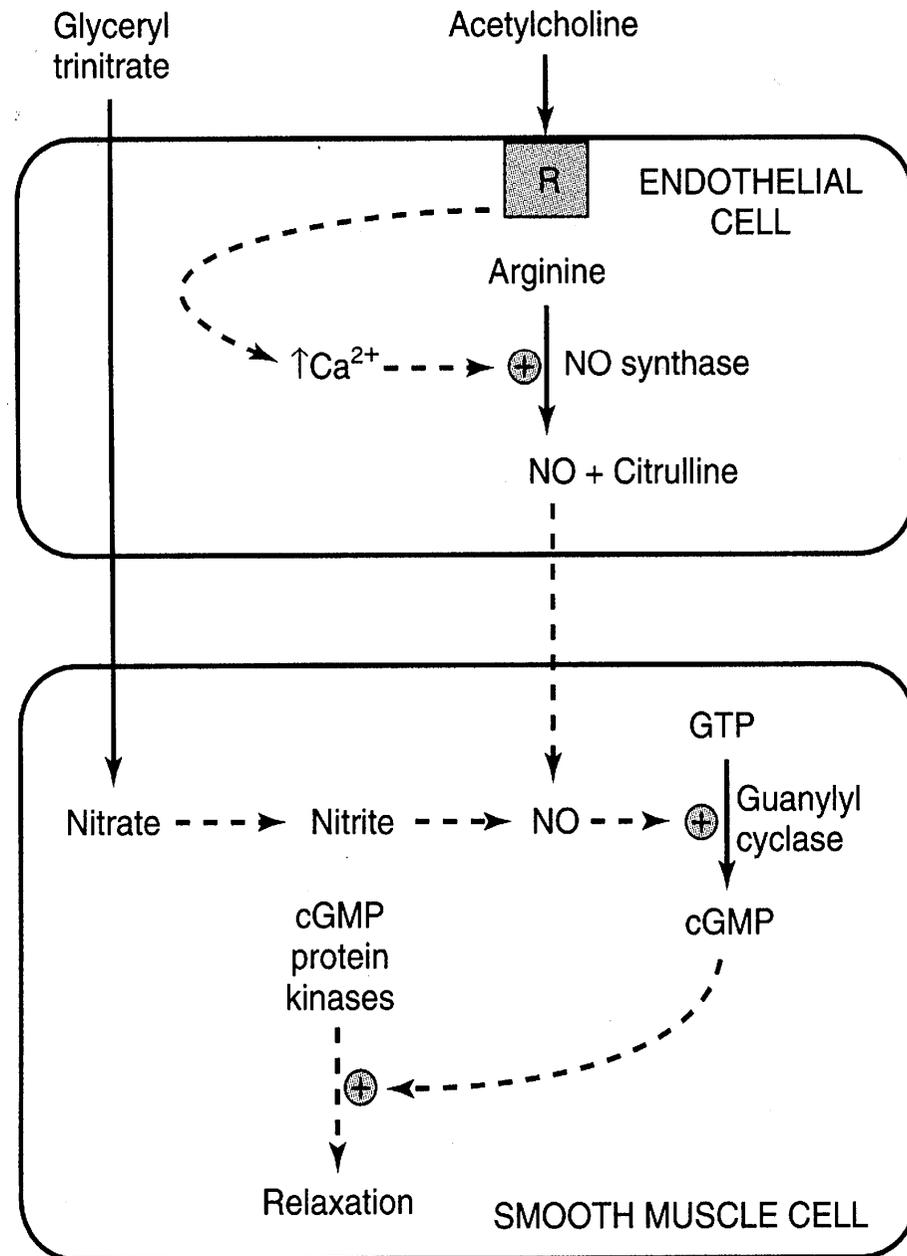
Slow atrial contractibility

Slow ventricular contractibility

Low blood pressure.

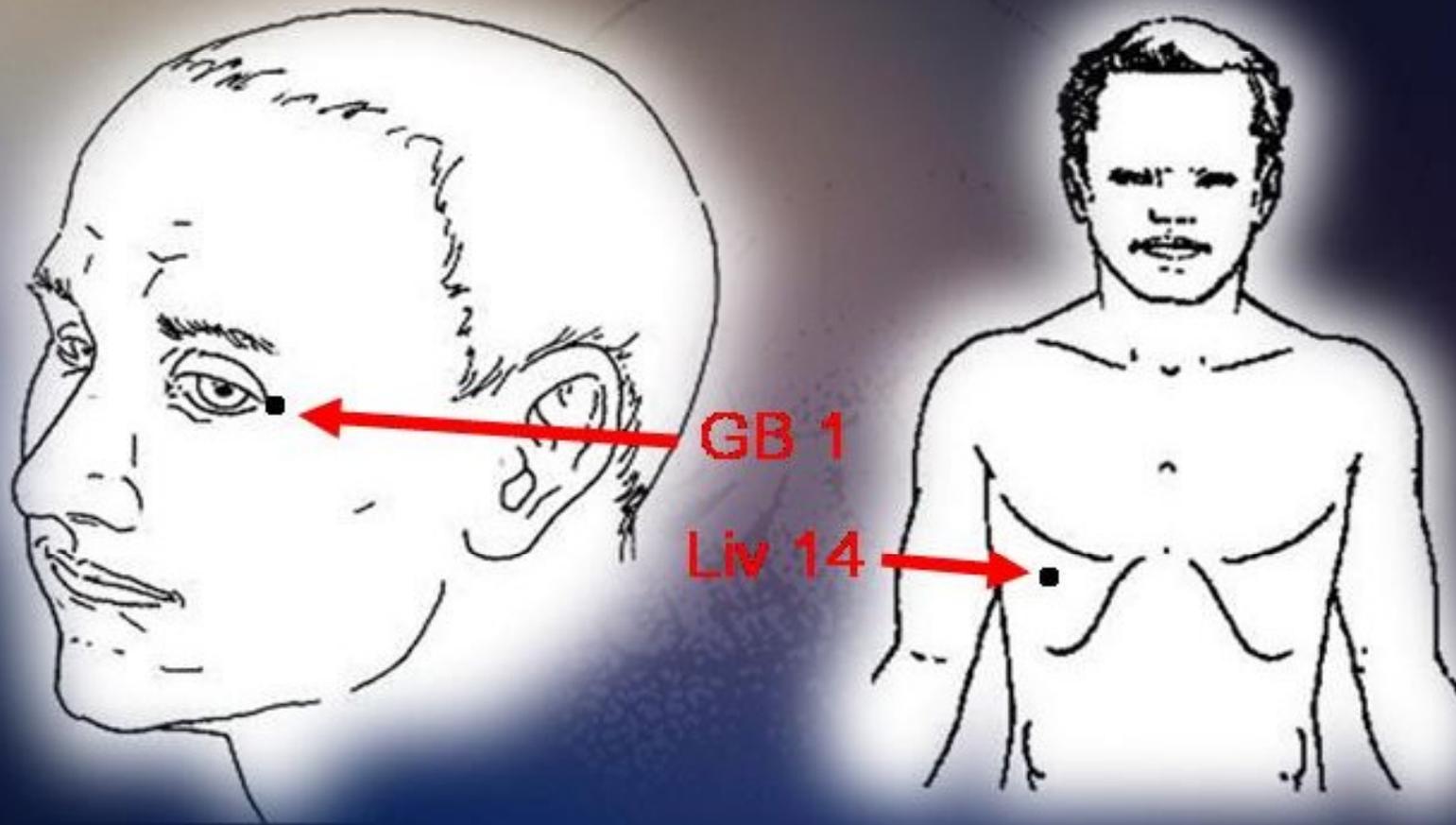
**Stimulates the release of NO in
blood vessels and so
vasodilates.**

Possible mechanism by which **Lemon Balm** may improve memory by increasing cerebral circulation.



ACETYLCHOLINE MERIDIAN DIAGNOSTIC POINTS

YANG POINTS (DEFICIENCY) YIN POINTS (EXCESS)

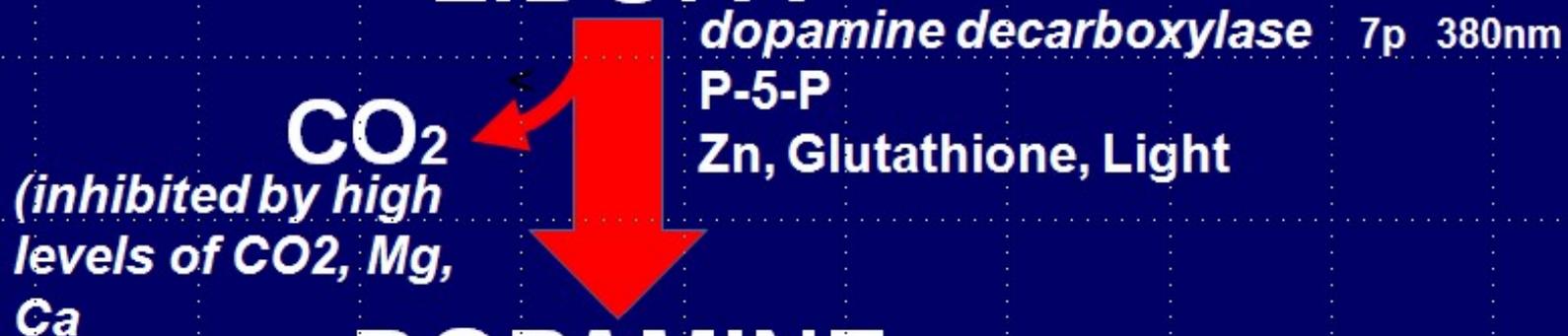


**Noradrenalin
(Norepinephrine)**

TYROSINE



L.DOPA



DOPAMINE



Small Intestine 19 – Low Noradrenalin

Slower heart rate

Slower conduction rate

Slower atrial contractibility

Slower ventricular contractibility

**Small Intestine 19 – Low
Noradrenalin**

Alpha 1 receptors - ?

**Alpha 2 receptors – Inability to
raise blood pressure.**

**Beta 1 receptors – inability to
increase force and contraction of
the heart.**

**Beta 2 receptors – inability to
release renin - hypotension**

NORADRENALIN

monoamine oxidase 14 387nm
23x 400nm

Cu+ FAD

Inhibited by benzoic acid,
caffeine, anthrocyandins,
eugenol, naringen, raison

O₂ + H₂O

H₂O₂

Dihydroxymandelic
acid + NH₂

catechol-O-methyltransferase

Mg⁺⁺, Fe, Mn, Cysteine 22 399nm

Inhibited by epicatechin, 2OH and
CH₃ Estrogens, Vit C, Ca, quercetin,
SAH, SAM,

Vanillylmandelic acid

Fe⁺⁺

Fe⁺⁺⁺

·OH + OH⁺

SAM

Vanillylmandelic acid

*Glutathione (Cysteine,
Glycine, Glutamic acid)*

NAC, Zn⁺⁺, P5P, Sel

a-Lipoic or

Sulfation (PAPs) S, MSM

Taurine or

Glucuronidation (UDP

Gucuronic acid) Glucuronate,

Vit C, or

Acetylation (Acetyl CoA) B5,

Acetyl CoA

Conjugates excreted through
the bile or urine

Heart 1 – High Noradrenalin

Increased heart rate

Increased conduction rate

Increased atrial contractibility

**Increased ventricular
contractibility.**

Heart 1 – High Noradrenalin

Alpha 1 receptors – Smooth muscle contraction – hypertension

Alpha 2 receptors - Vasoconstriction - hypertension.

Beta 1 receptors are found in the heart and increases force and contraction - Palpitations and Tachycardia Heart Arrhythmias.

Beta 2 receptors cause release of renin - hypertension

Angiotensinogen

↑ **Glucocorticoids and Estrogens**
(made in liver)



renin (made in kidney)

Angiotensin 1



***angiotensin converting enzyme ACE (also degrades bradykinin, a powerful vasodilator)**

Angiotensin 11

powerful vasoconstrictor and stimulates aldosterone



aminopeptidase

Angiotensin 111

less powerful vasoconstrictor



angiotensinases

Degradation products

*Inhibited by soymilk protein proteolytic peptides with lactobacillus and bifido bacteria

Angioteninogen is made in the liver. **Renin** is synthesised in the renal afferent tubules which are supplied by the renal sympathetic nerves.

These mediate the CNS and postural effects of renin release and involves the β -adrenergic receptor.

Stimulants of renin

Decreased BP

**Change from supine
to erect posture**

Salt depletion

β -adrenergic agents

Prostaglandins 2

Inhibitors of renin

Increased BP

**Change from erect to
supine posture**

Salt loading

β -adrenergic antagonists

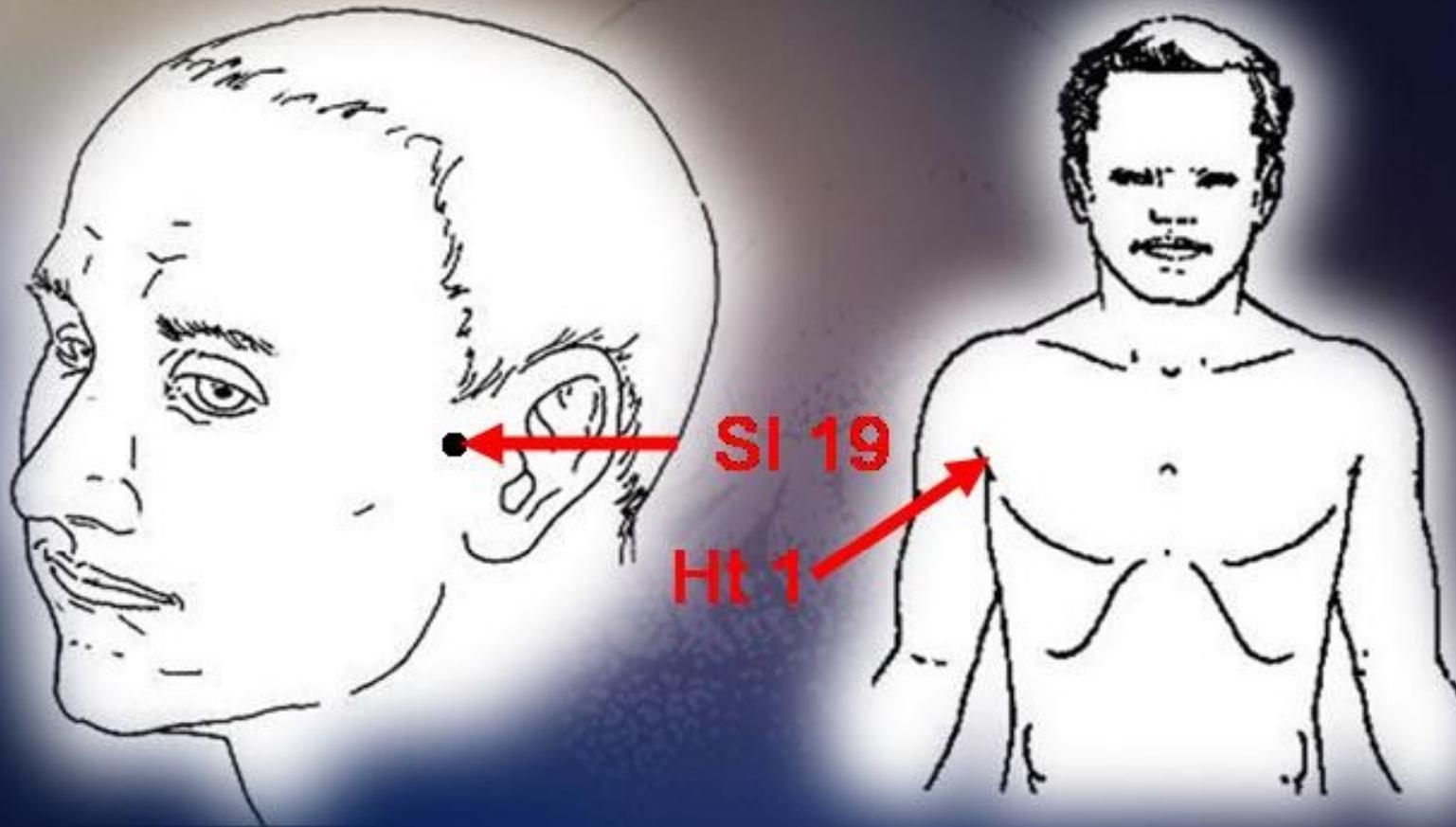
**Prostaglandins 2
inhibitors (GLA, EPA)**

Potassium

Vasopressin

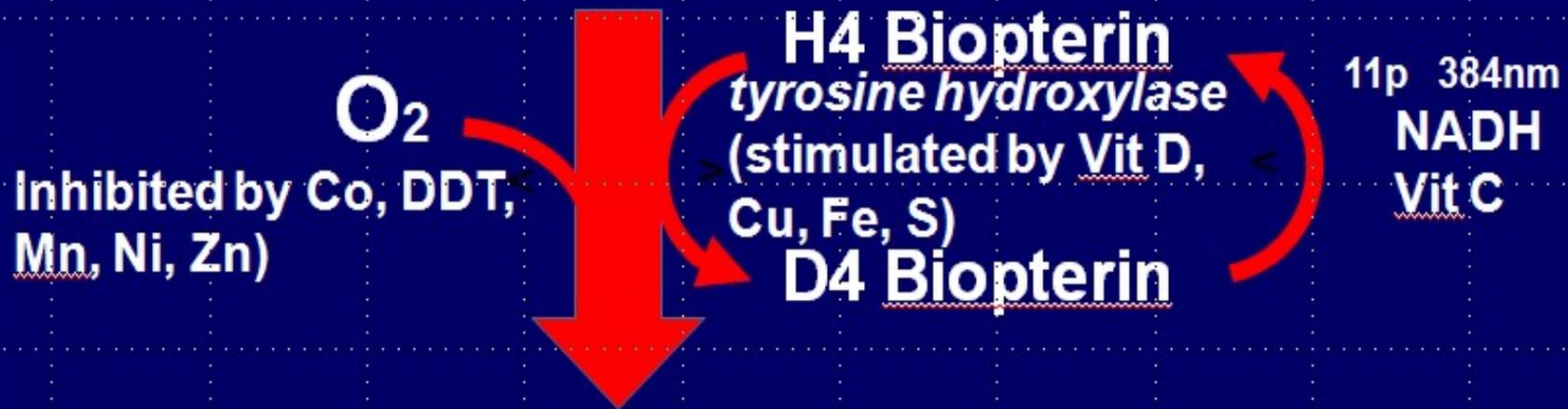
Angiotensin 11

NORADRENALIN MERIDIAN DIAGNOSTIC POINTS
YANG POINTS (DEFICIENCY) YIN POINTS (EXCESS)



Dopamine

TYROSINE



L.DOPA



DOPAMINE

CV24 – Low Dopamine

Dopamine receptors are mainly located in the blood vessels and the heart and respond to exogenously applied dopamine indicating their sensitivity.

Mesenteric and renal arteries causing vasodilation.

DOPAMINE

monoamine oxidase 14 387nm
23x 400nm

Cu⁺ FAD

Inhibited by benzoic acid,
caffeine, anthrocyandins,
eugenol, naringen, raison

O₂ + H₂O

H₂O₂

Fe⁺⁺

Fe⁺⁺⁺

·OH + OH⁺

Dihydroxyphenyl
acetic acid + NH₂

catechol-O-methyltransferase

22 399nm

Mg⁺⁺, Fe, Mn, Cysteine

Inhibited by epicatechin, 2OH and
CH₃ Estrogens, Vit C, Ca, quercetin,
SAH, SAM,

Homovanillic acid

SAM

Homovanillic acid

*Glutathione (Cysteine,
Glycine, Glutamic acid)*

NAC, Zn⁺⁺, P5P, Sel
a-Lipoic or

Sulfation S, MSM

Taurine or

Glucuronidation (UDP

Gucuronic acid) Glucuronate,

Vit C, or

Acetylation (Acetyl CoA) B5,

Acetyl CoA

Conjugates excreted through
the bile or urine

GV27 – High Dopamine

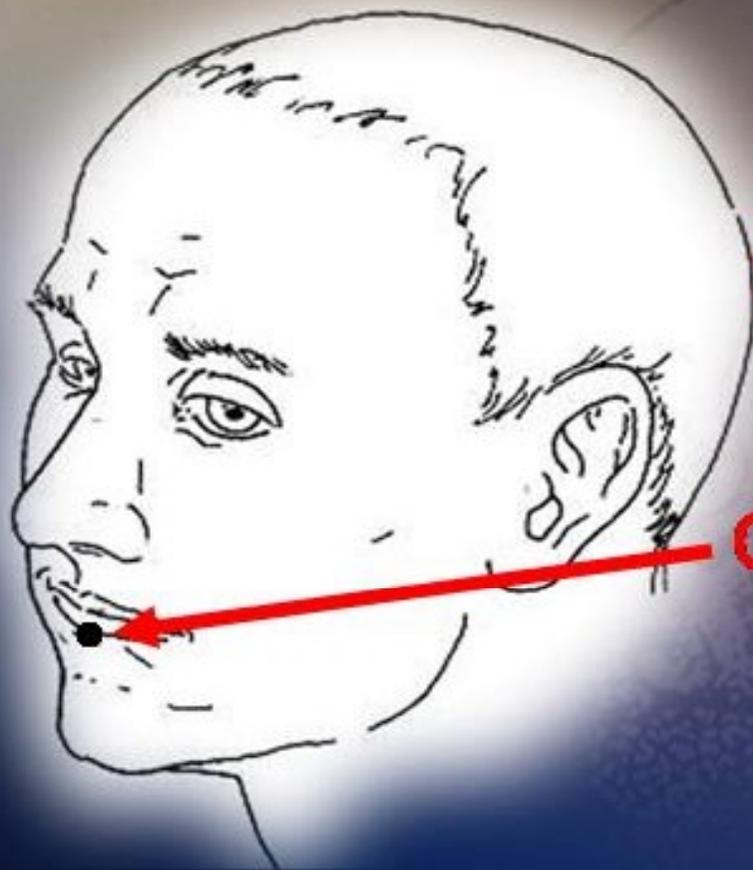
Dopamine receptors are mainly located in the blood vessels and the heart and respond to exogenously applied dopamine indicating their sensitivity.

Mesenteric and renal arteries causing vasodilation.

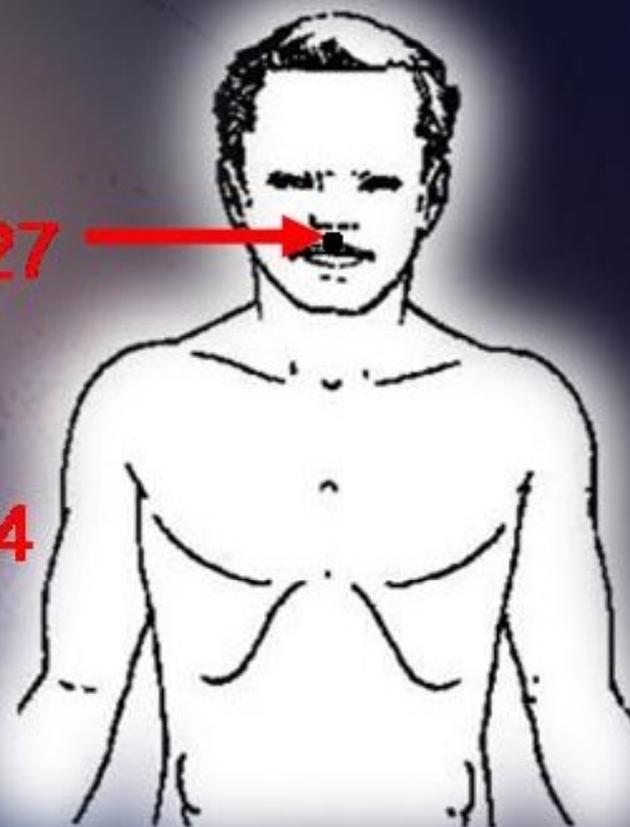
DOPAMINE MERIDIAN DIAGNOSTIC POINTS

YANG POINTS (DEFICIENCY)

YIN POINTS (EXCESS)



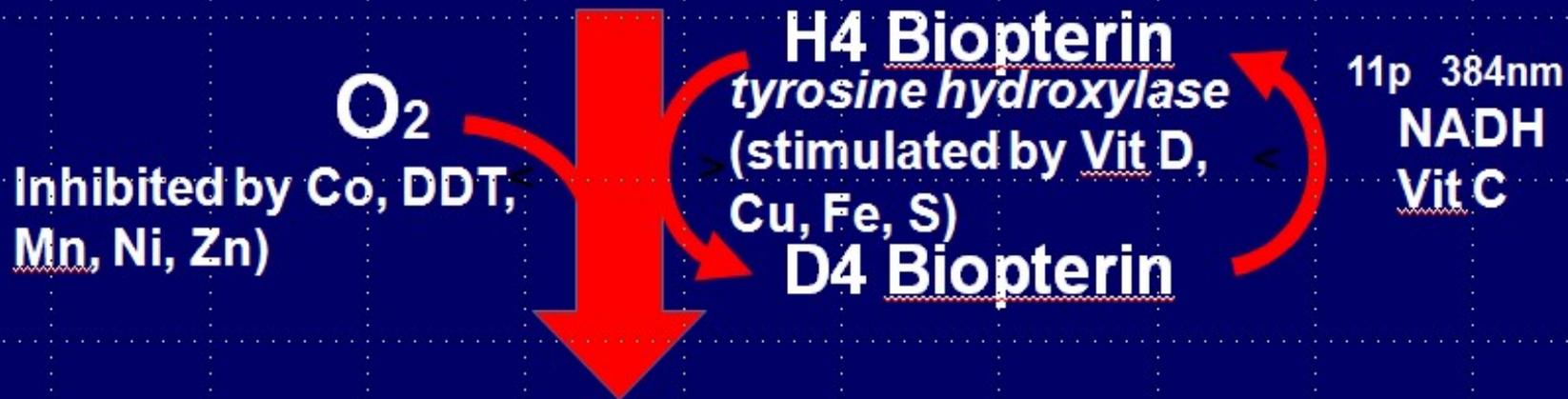
GV27



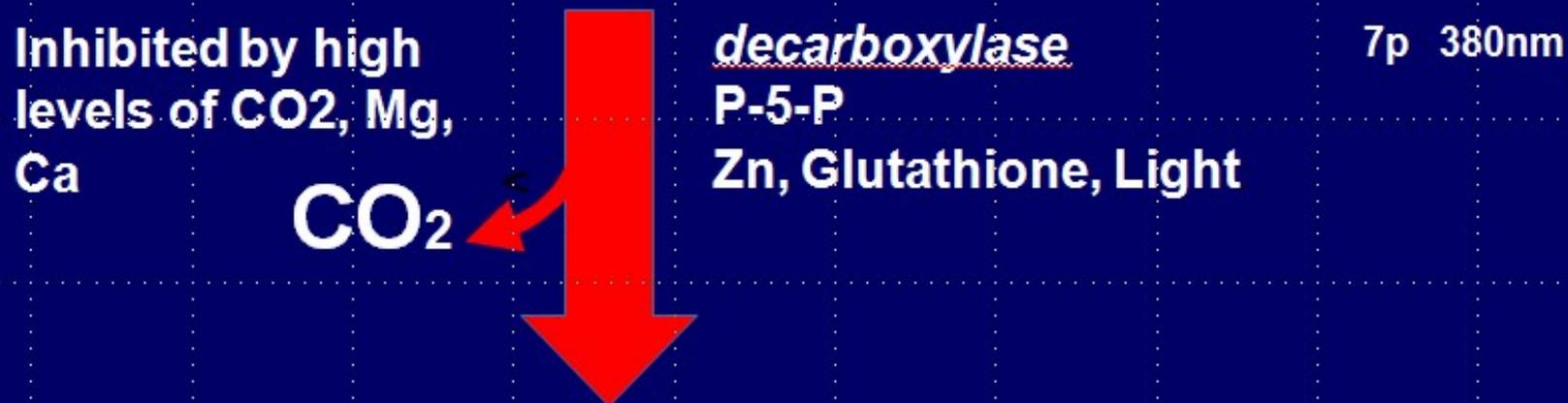
CV24

Serotonin

TRYPTOPHAN



5-Hydroxytryptophan



SEROTONIN

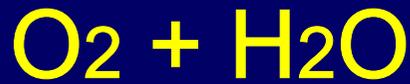
Bladder 1 – Low Serotonin

Low Blood pressure

SEROTONIN

monoamine oxidase 14 387nm
23x 400nm
Cu⁺ FAD

Inhibited by benzoic acid,
caffeine, anthrocyandins,
eugenol, naringen, raison



Hydroxyindole
acetate + NH₄

catechol-O-methyltransferase
22 399nm

Mg⁺⁺, Fe, Mn, Cysteine

Inhibited by epicatechin, 2OH and
CH₃ Estrogens, Vit C, Ca, quercetin,
SAH, SAM,



Methoxyindole acetate



Methoxyindole acetate



*Glutathione (Cysteine,
Glycine, Glutamic acid)*

NAC, Zn⁺⁺, P5P, Sel
a-Lipoic or

Sulfation S, MSM

Taurine or

Glucuronidation (UDP

Gucuronic acid) Glucuronate,
Vit C, or

Acetylation (Acetyl CoA) B5,
Acetyl CoA

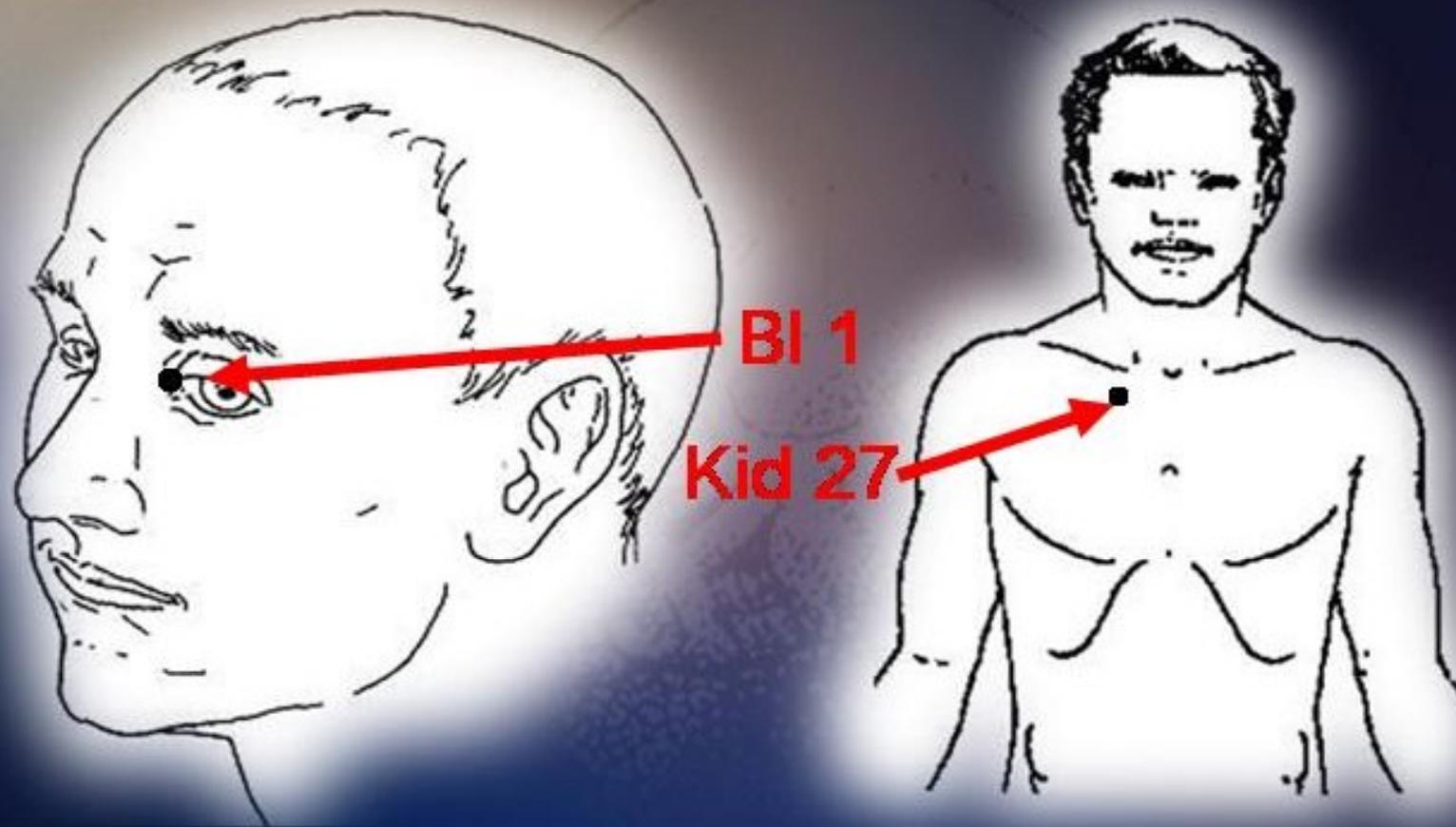
Conjugates excreted through
the bile or urine

Kid 27 - High Serotonin (5-HT)

5-HT release either from nerves or from platelets causes vasoconstriction of all large blood vessels (but vasodilation of the mid meningeal artery by "gating" incoming sensory inputs and by decreasing sympathetic nervous outflow).

SEROTONIN MERIDIAN DIAGNOSTIC POINTS

YANG POINTS (DEFICIENCY) YIN POINTS (EXCESS)



Histamine

HISTIDINE



Vit B6 (or Vit B1)
Mg, Zn

CO
2



decarboxylase 15q 388nm
*(inhibited by high levels
of CO₂)*

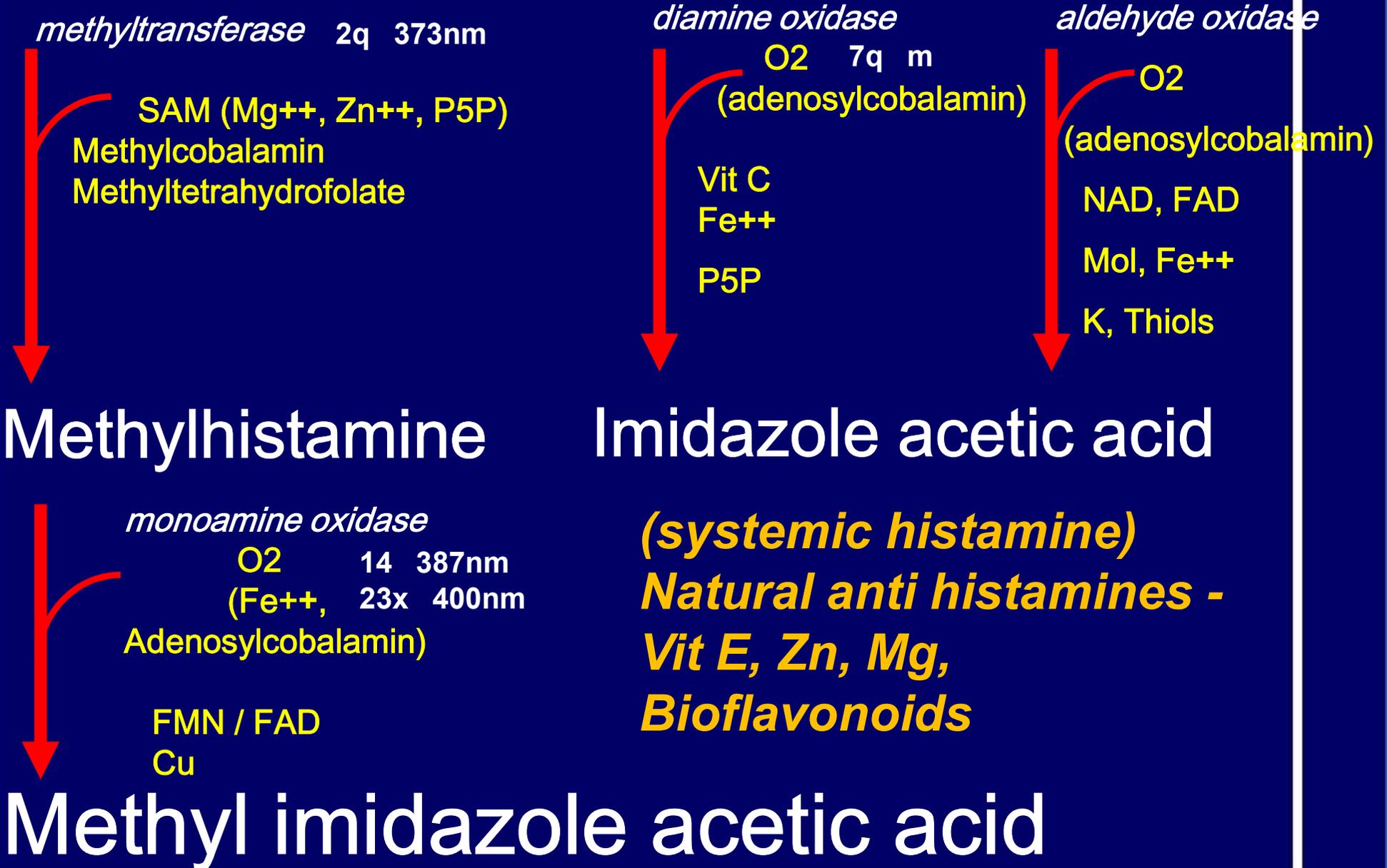
HISTAMINE

Stomach 1 – Low Histamine

Low iNOS within the immune system may lead to high Blood Pressure.

HISTAMINE

2q 373nm
18 393nm



Spleen 21 – High Histamine

High iNOS within the immune system may lead to low Blood pressure.

Paradoxically histamine activates cAMP which may increase Blood pressure.

Histamine H1 receptors outside the CNS

Constriction of large arteries and veins.

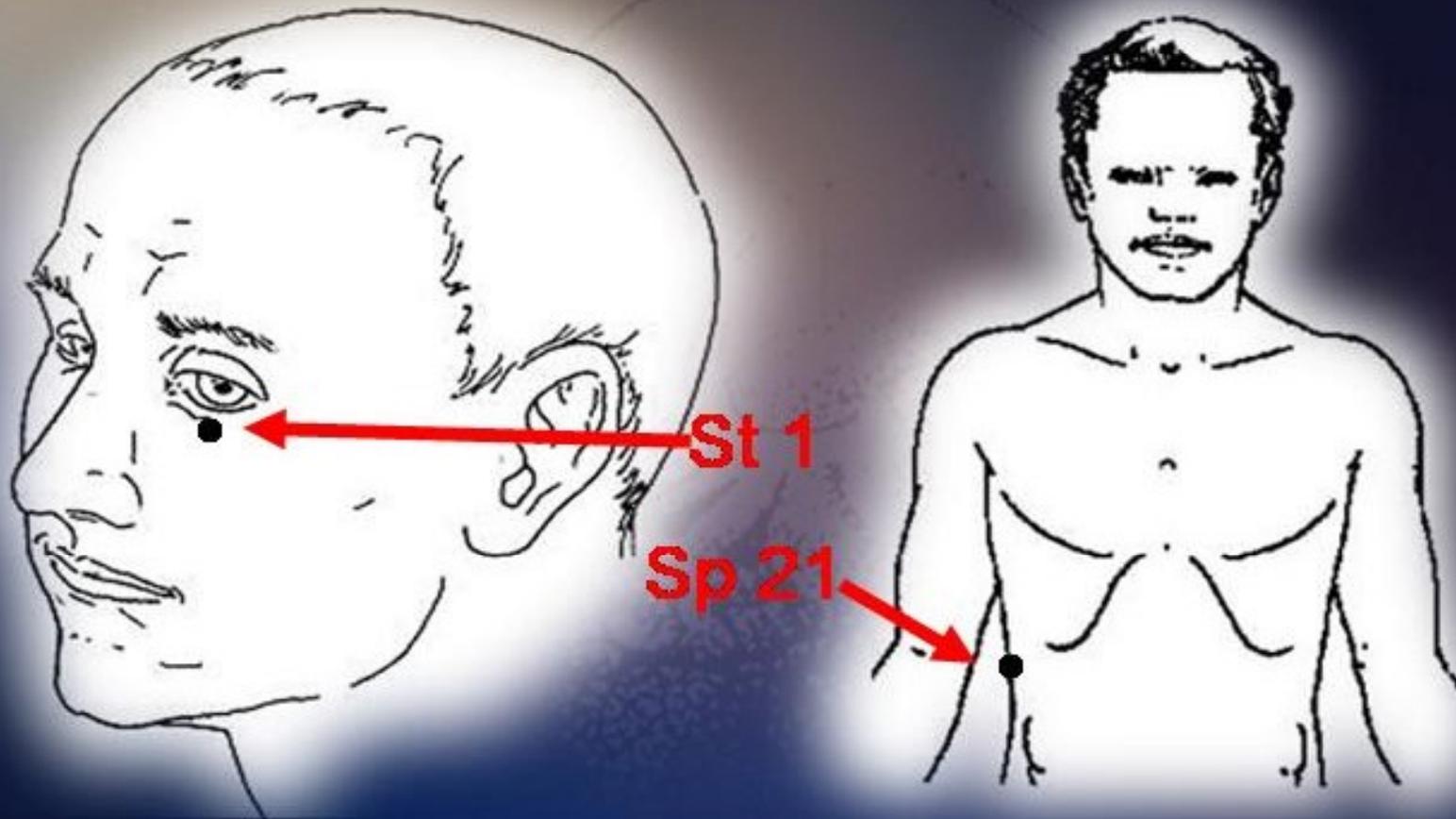
Relaxation of arterioles, small veins and capillaries especially in the brain.

Increased capillary permeability.

HISTAMINE MERIDIAN DIAGNOSTIC POINTS

YANG POINTS (DEFICIENCY)

YIN POINTS (EXCESS)



GABA

GLUTAMATE

Inhibited by
Cysteine, NO, O₂

glutamate decarboxylase

P5P (Thiamine pyro) 2q 373nm
10p 383nm

Mg

Zn

CO₂

GABA

2-oxoglutarate

GABA transaminase 16p 389nm

P5P

Glutamate

Succinic semialdehyde

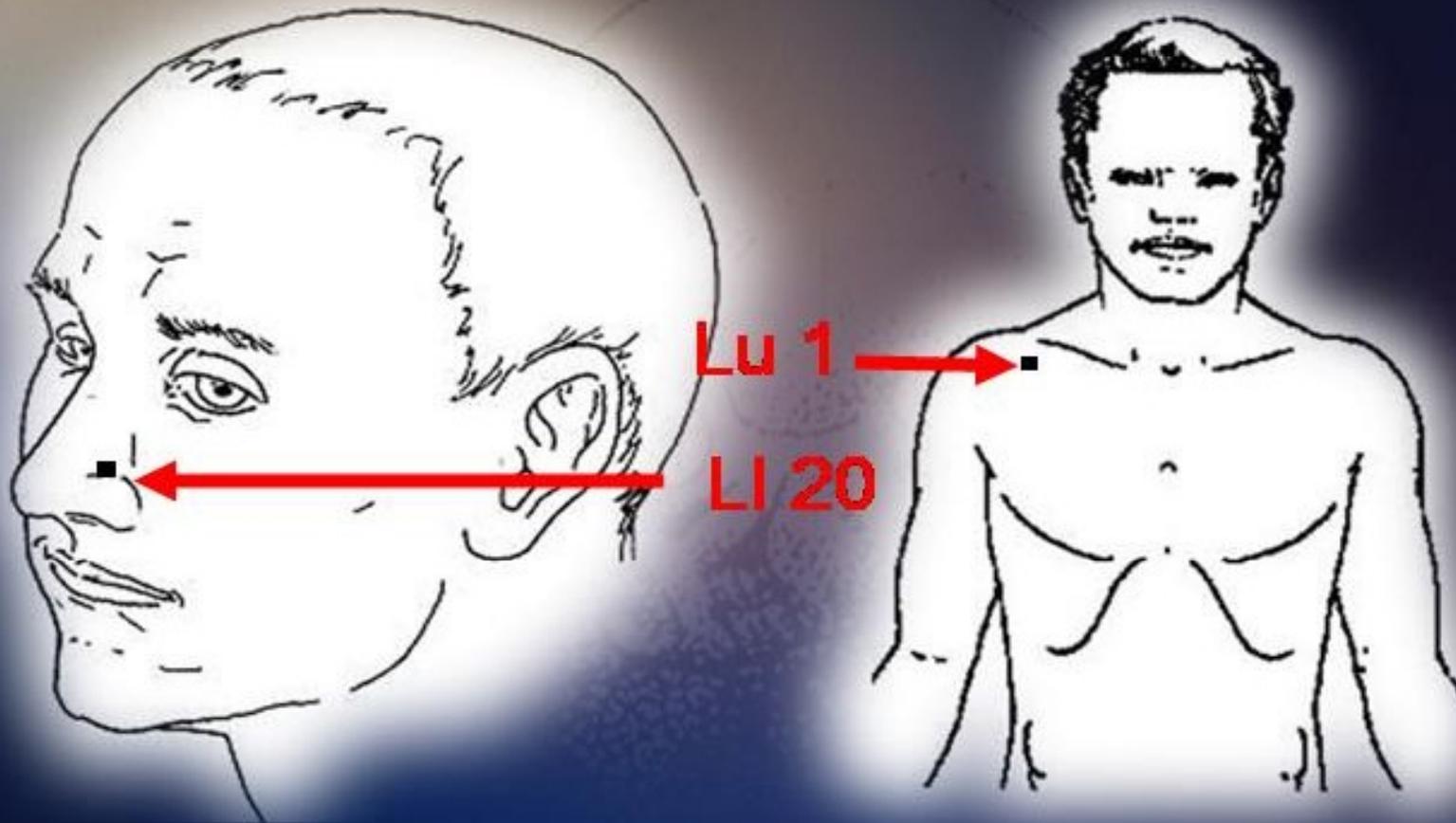
GABA is an inhibitory neurotransmitter in the CNS

It is not present in peripheral nerves.

GABA C receptor for benzodiazepine may relax a person emotionally and thus reduce BP.

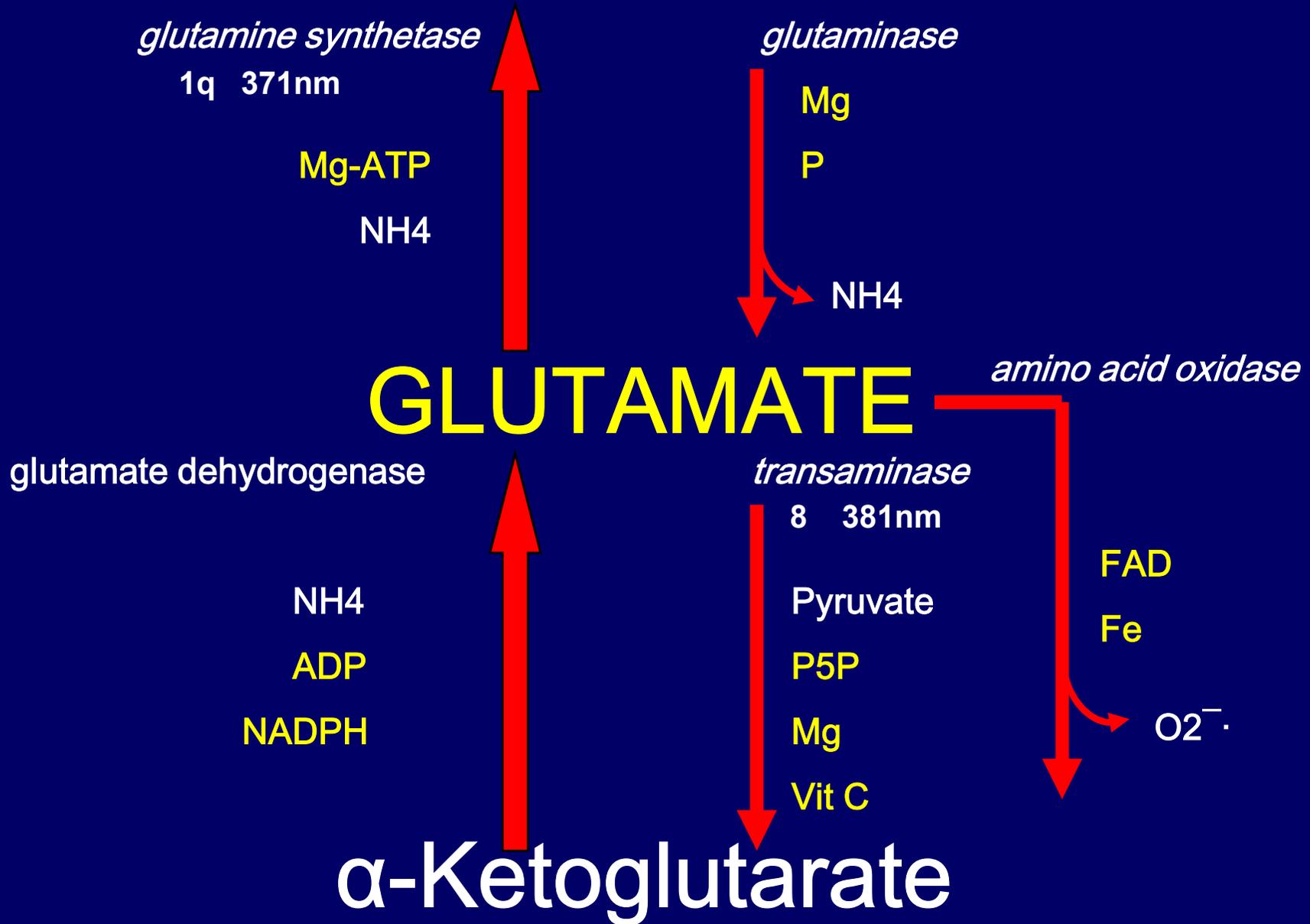
GABA MERIDIAN DIAGNOSTIC POINTS

YANG POINTS (DEFICIENCY) YIN POINTS (EXCESS)



**Glutamic acid
(Glutamate)**

Glutamine



TW 23 – Low Glutamate

May indicate low NO as glutamate increases cNOS leading to an inability to lower raised blood pressure.

Cx 1 – High Glutamate

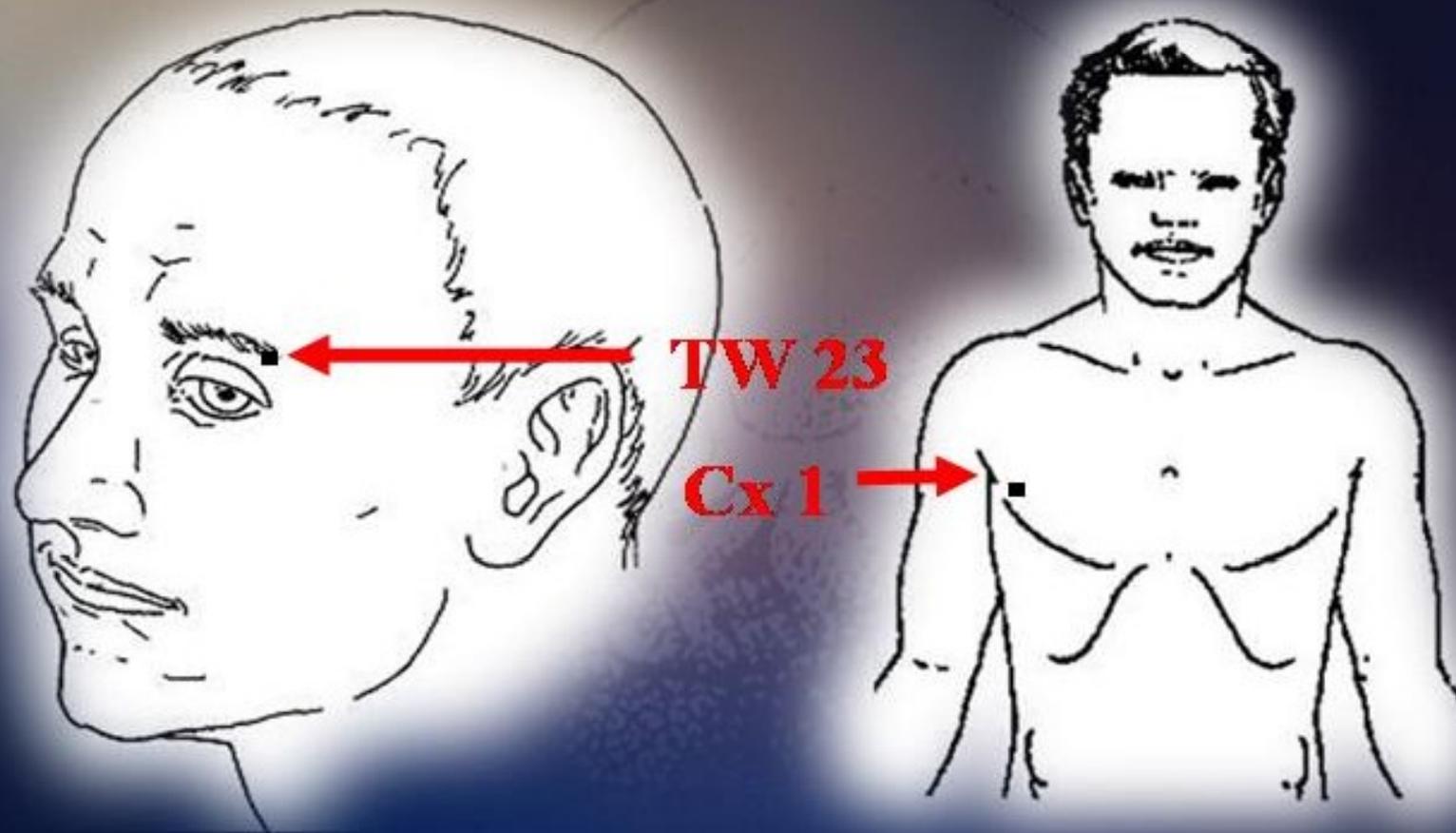
May indicate high NO as glutamate increases cNOS leading to low blood pressure.

However, both aspartate and glutamate (and their agonists – aspartame and MSG) may increase heart rate and lead to tachycardia.

EXCITATORY MERIDIAN DIAGNOSTIC POINTS

YANG POINTS (DEFICIENCY)

YIN POINTS (EXCESS)



Nutritional and Natural Medicines

Taurine

Adenosine

Magnesium

Calcium

Potassium

Vitamin C

GLA and /

or EPA

CoQ10

Allum Sativa

Cayenne pepper

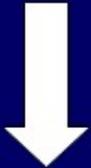
Hawthorn

Berry(Crateagus)

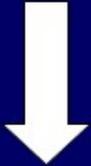
Rutin

CHOLESTEROL
and
TRIGLYCERIDES

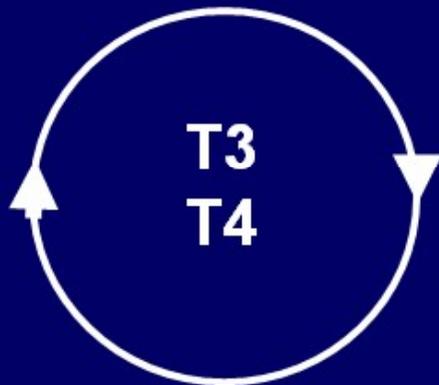
Glucose



Pyruvate



Acetyl CoA



Electron transport ATP

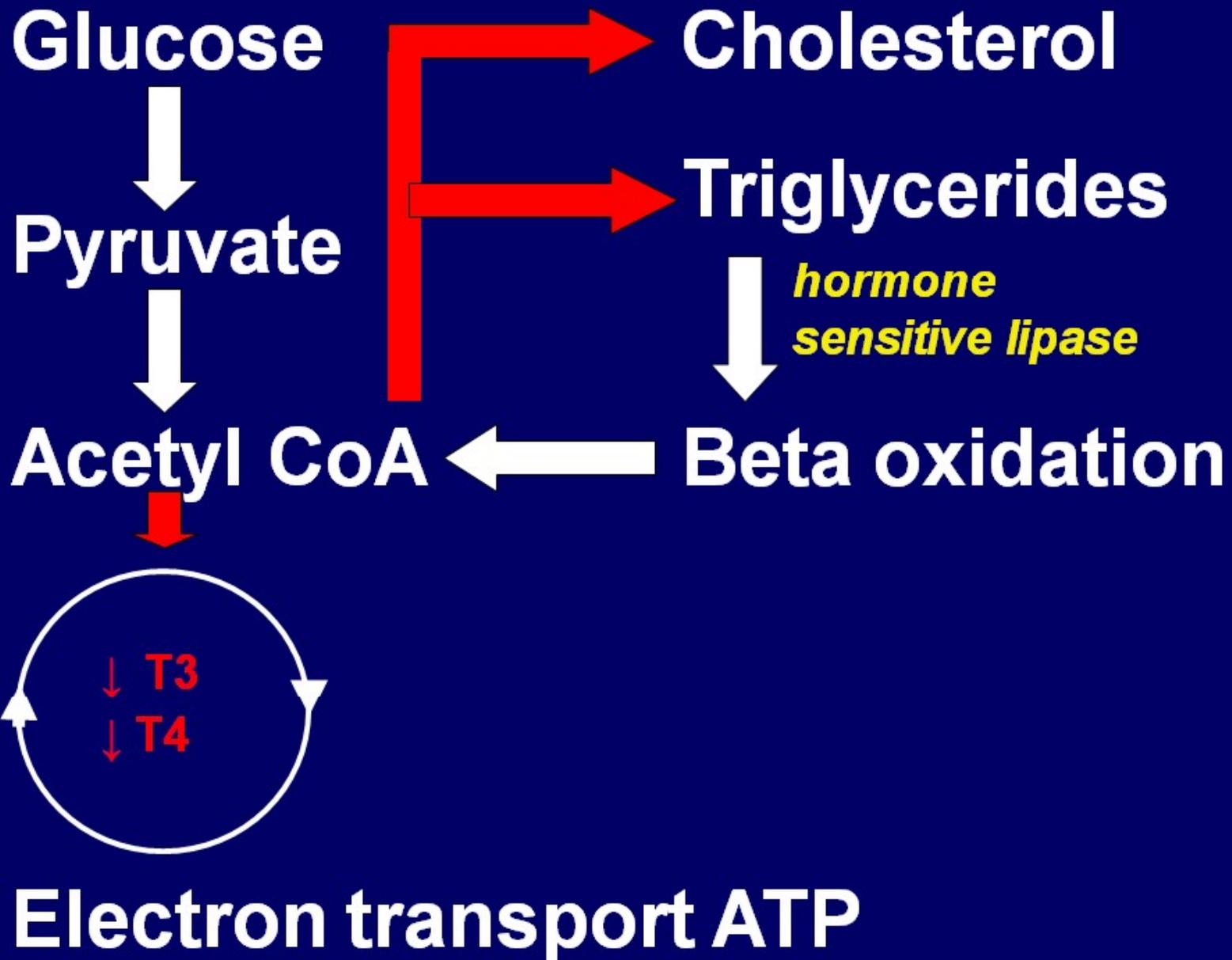
Triglycerides

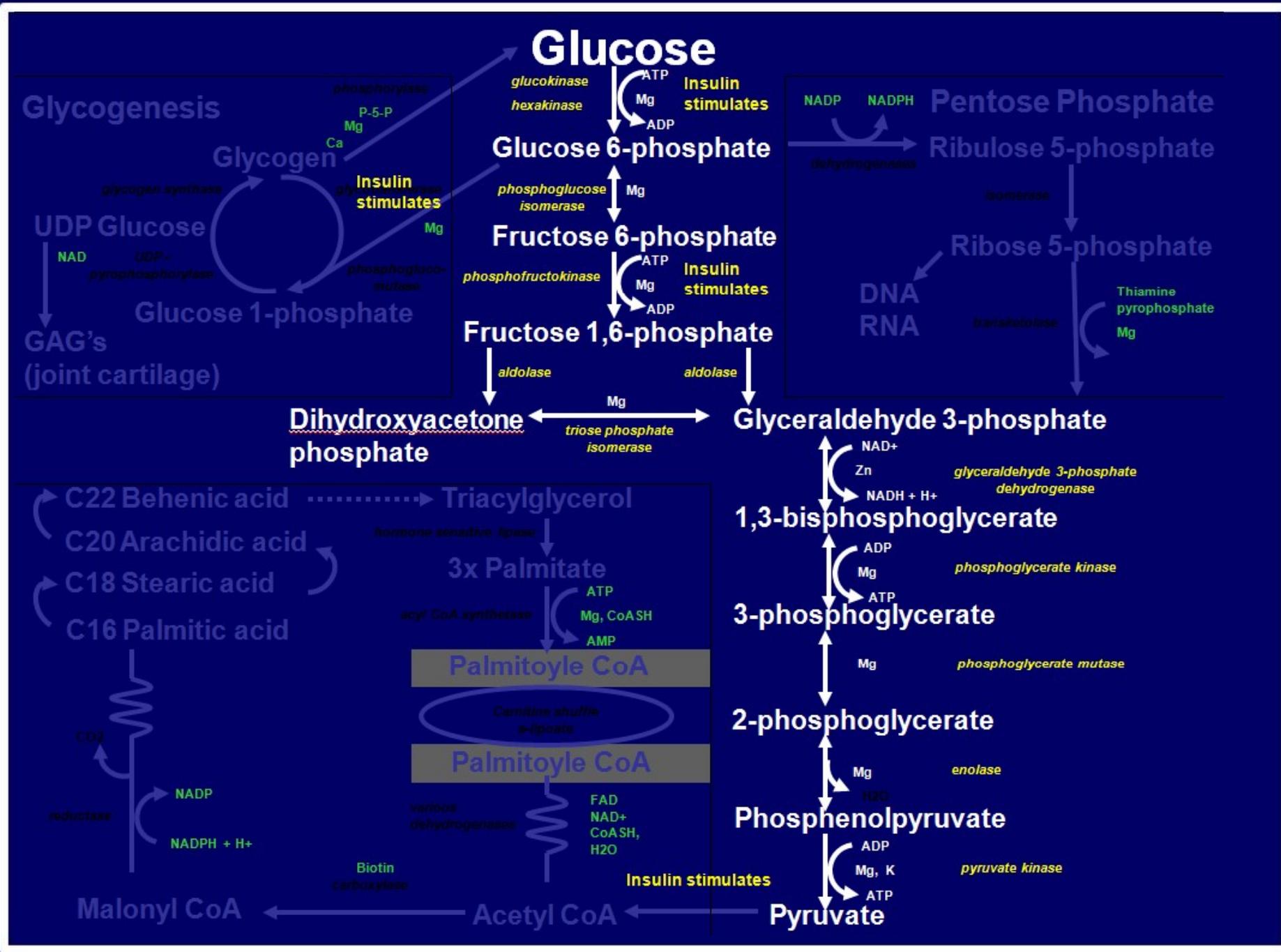


*hormone
sensitive lipase*

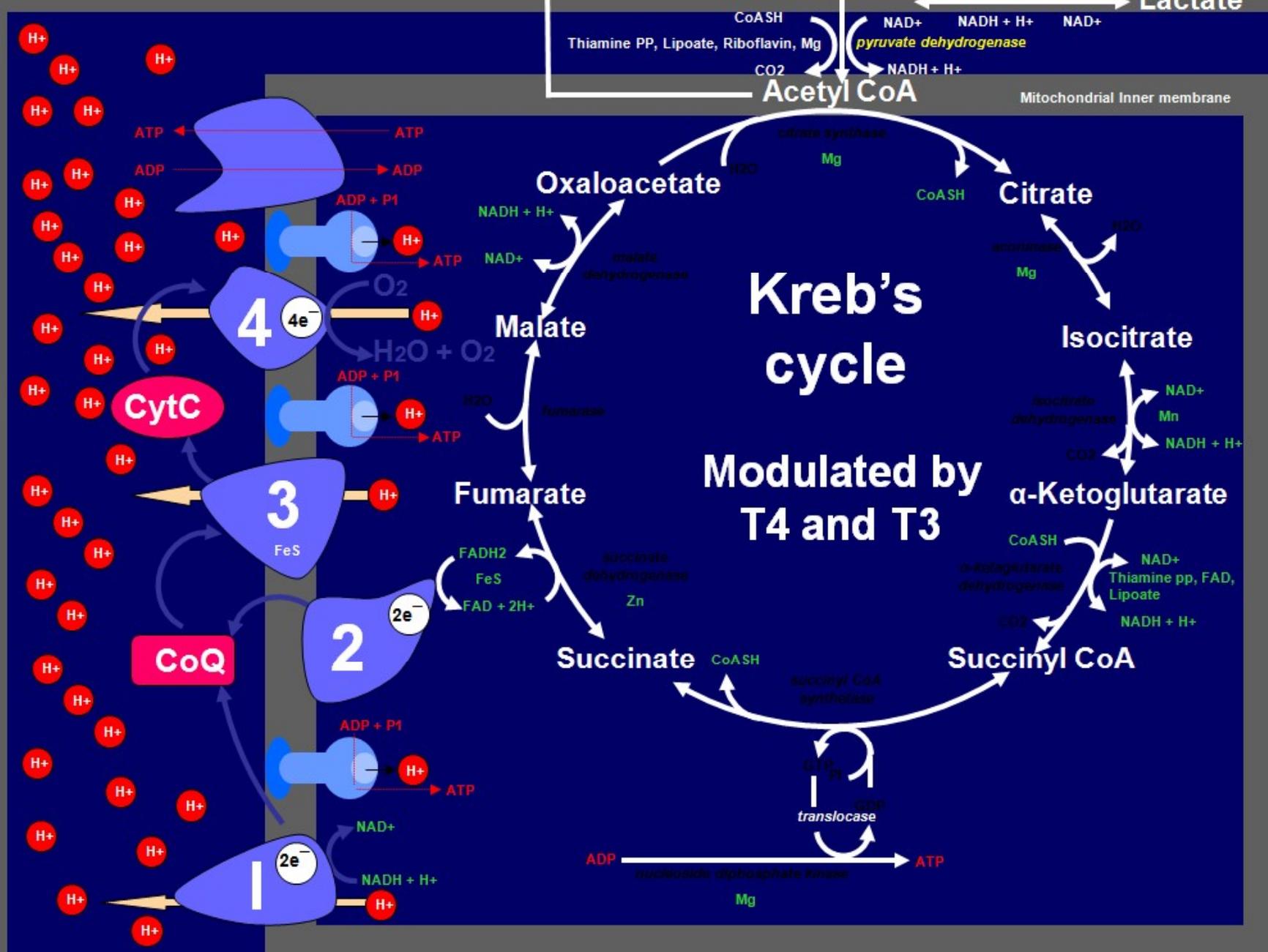
Beta oxidation

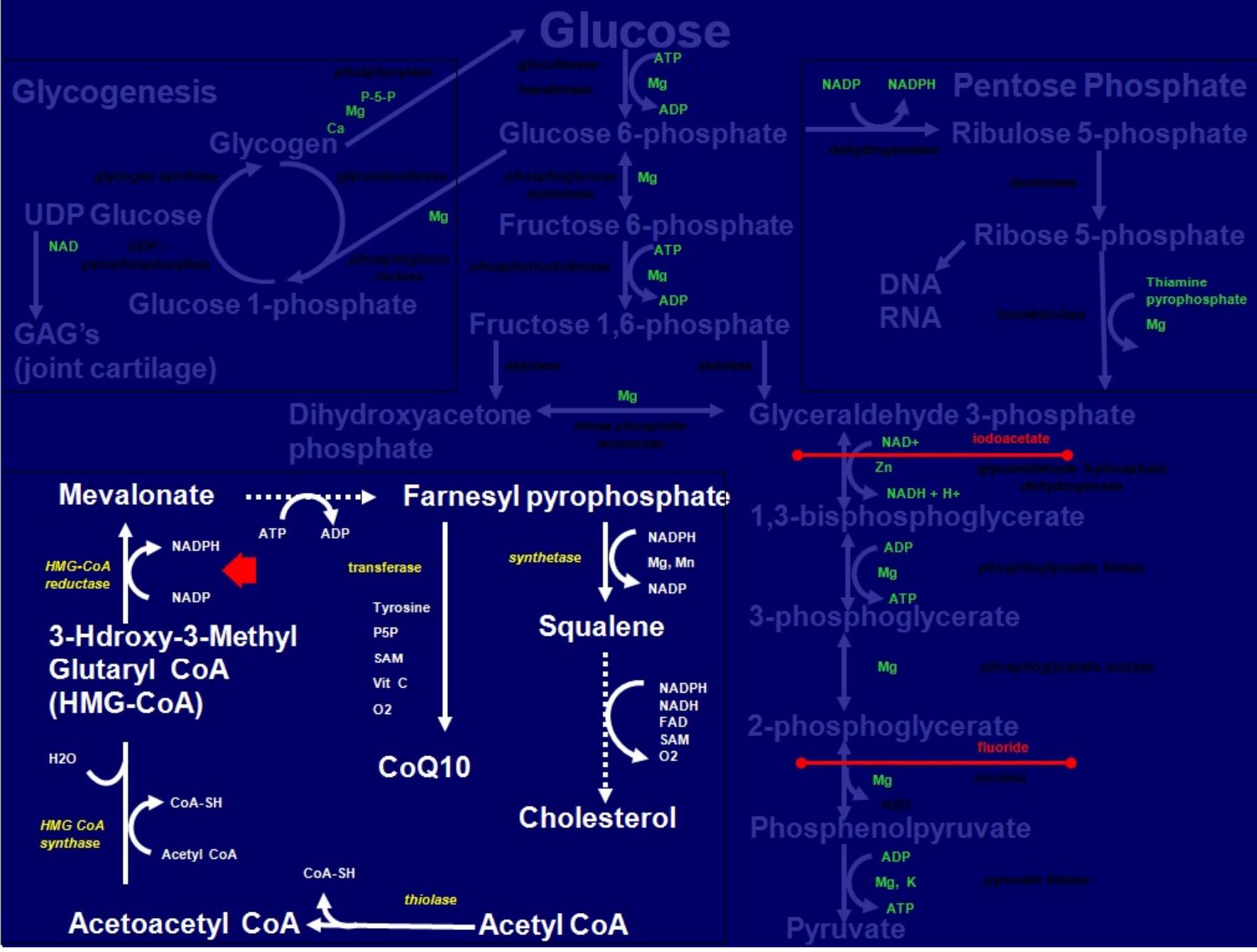




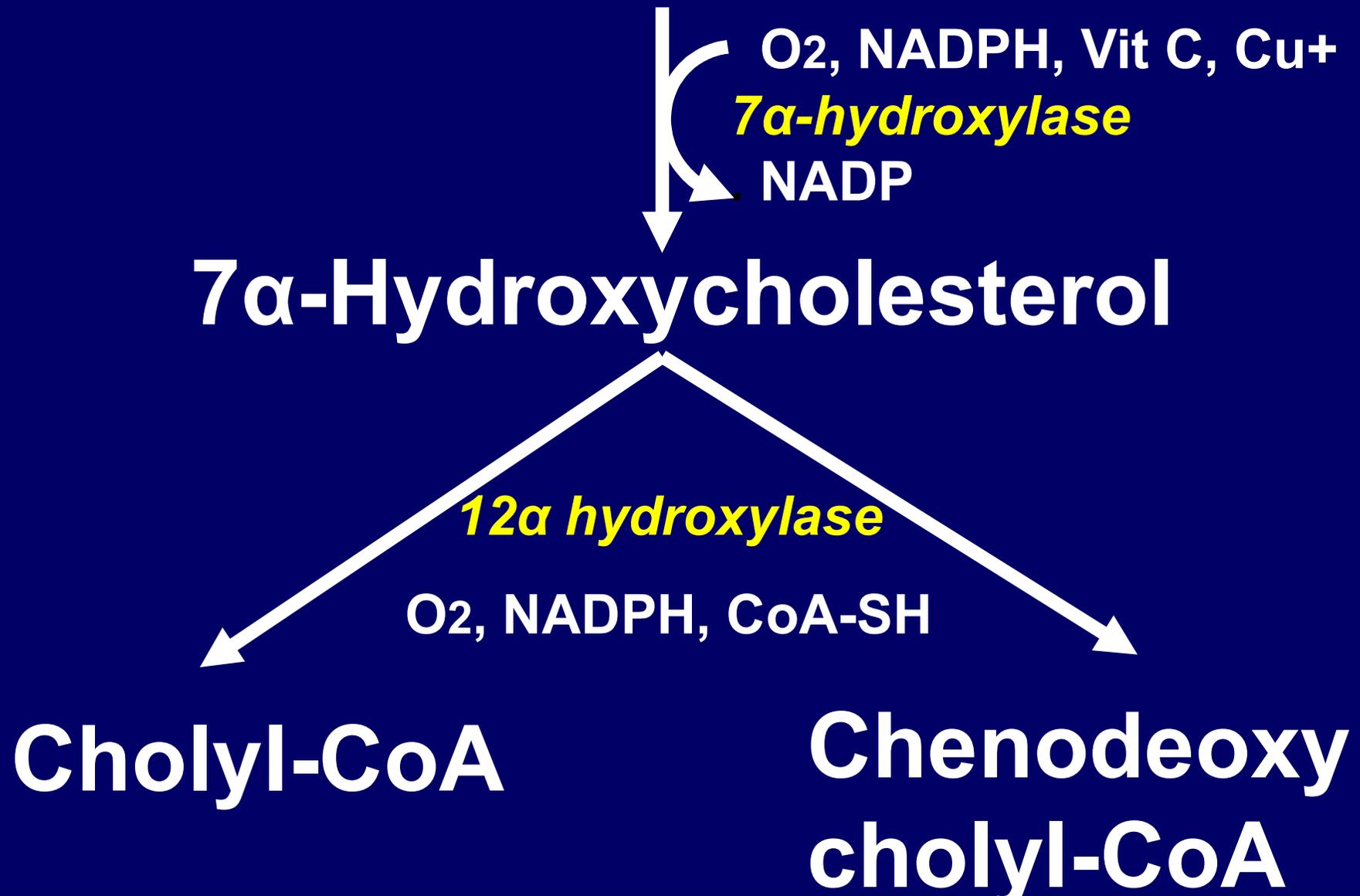


Mitochondrial Outer membrane





Cholesterol Metabolism



Cholyl-CoA

Taurine

conjugase

CoA-SH

Glycine

conjugase

CoA-SH

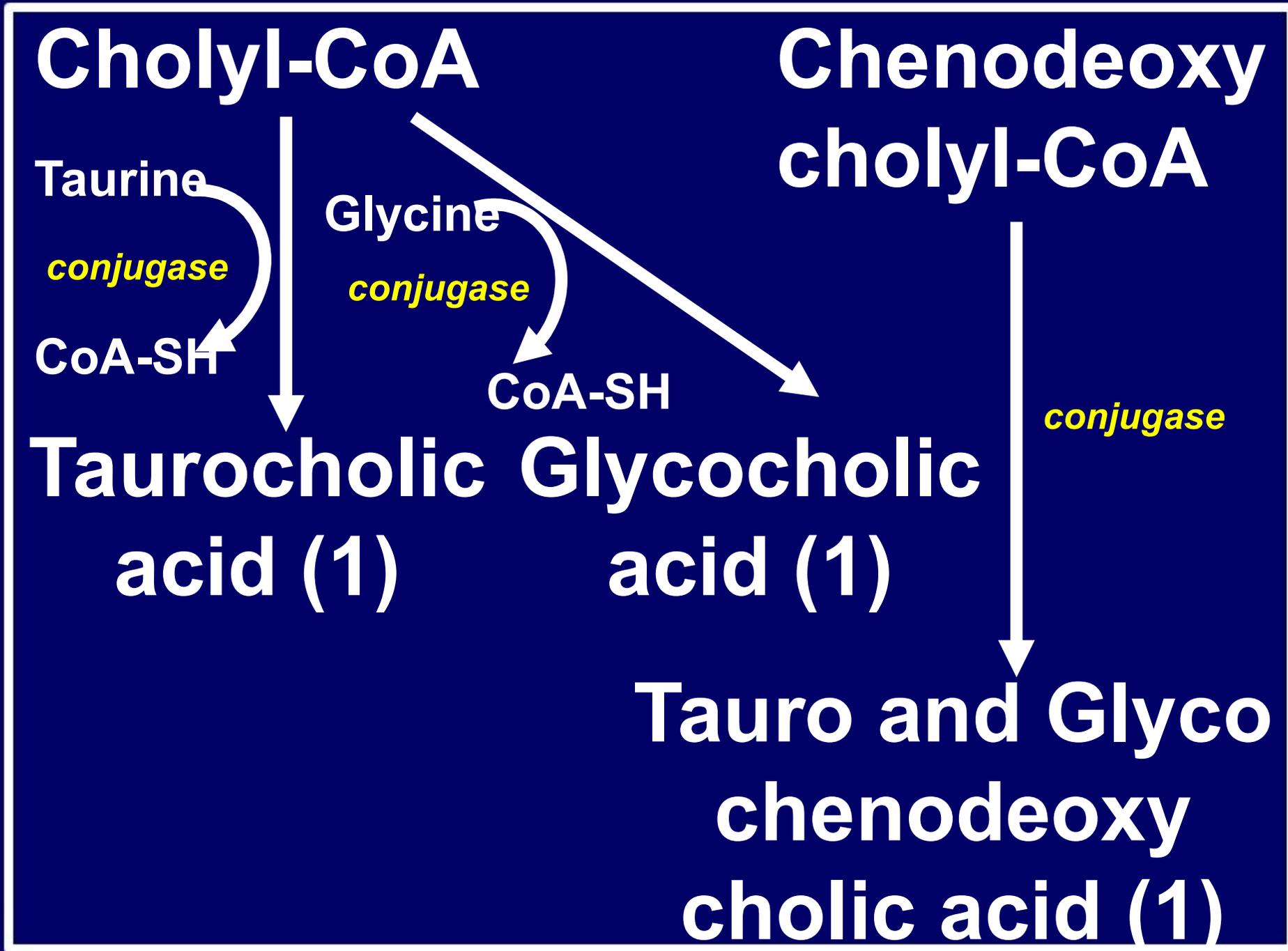
**Chenodeoxy
cholyl-CoA**

conjugase

**Taurocholic
acid (1)**

**Glycocholic
acid (1)**

**Tauro and Glyco
chenodeoxy
cholic acid (1)**



**Taurocholic
acid (1)**



**Taurodeoxy
cholic
acid (2)**

**Glycocholic
acid (1)**



**Glycodeoxy
cholic
acid (2)**

**Tauro and Glyco
chenodeoxy
cholic acid (1)**



**Lithocholic
acid (2)**

Key Nutrients in Cholesterol Metabolism

B3 (NADPH)

Vit C

Cu+

B5

Glycine

Taurine

Phosphatidylcholine

Omega oils

Selenium

Iodine

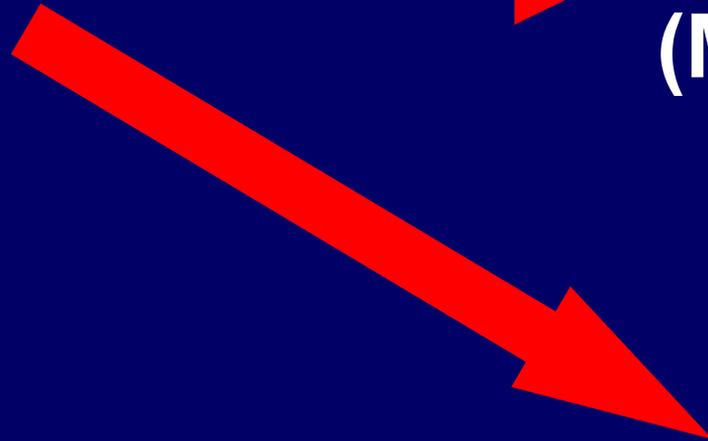
Tyrosine



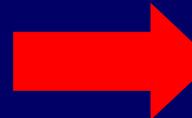
**Diodotyrosine
(DIT)**



**Monoiodotyrosine
(MIT)**

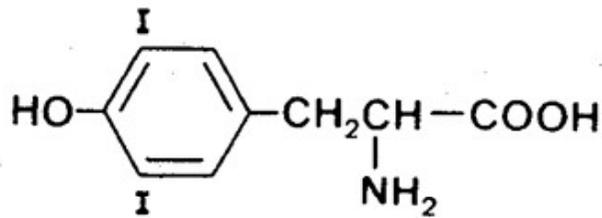


**Tetraiodothyronine
THYROXIN (T4)**

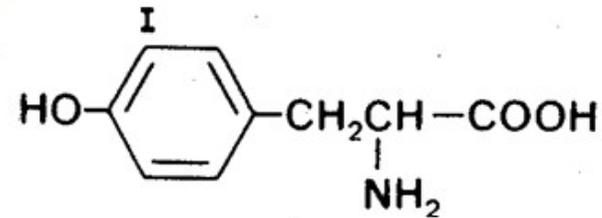


**Triiodothyronine
(T3) or Reverse T3**

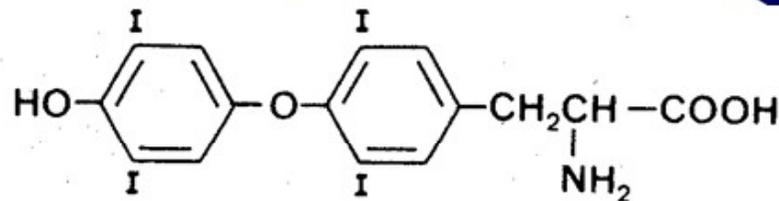
Tyrosine



**Diodotyrosine
(DIT)**

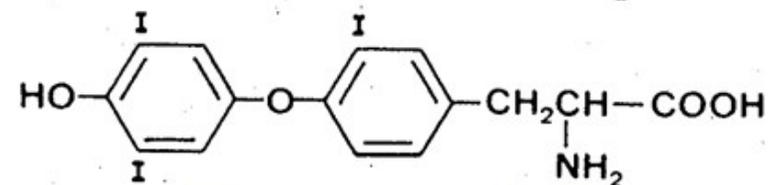
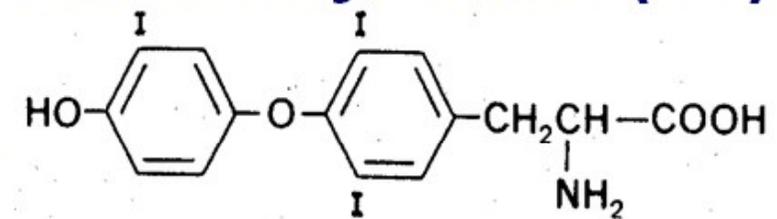


**Monoiodotyrosine
(MIT)**



**Tetraiodothyronine
THYROXIN (T4)**

Triiodothyronine (T3)



or Reverse T3

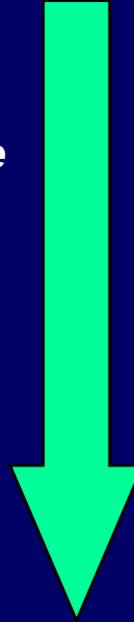
Thyroxin (T4)

*Type 1&2
deiodinase*



**35% deiodination
to T3**

Type 1&3 deiodinase



**45% deiodination to
reverse T3 and T4 to DIT**



**20% conjugation mainly
with glucuronate in the liver**

Challenge Pathways - Cholesterol

**Weakness to
Cholesterol**



**Challenge against
B3 (NADPH)**

Vit C

Cu+

O2 (Adenosylcobalamine, Fe)

B5 (CoA),

Taurine

Glycine

**Phosphatidylcholine (Omega
oils)**

Iodine

Selenium

Low ratio of coenzyme Q10 to low-density lipoprotein (LDL) cholesterol is a strong indicator of risk of atherosclerosis (clogging of the arteries).

Nutritional and Natural medicines

B3 (NADPH)

Vit C

Cu+

O2 (Adenosylcobalamine, Fe)

B5 (CoA),

Taurine

Glycine

Phosphatidylcholine

Omega oils

Iodine

Selenium

Cayenne pepper

Garlic

Niacin

Niacin is a member of the Vitamin B family, known as vitamin B3. Niacin is a natural cholesterol-lowering agent that alone has been shown to out-perform prescriptive drugs in mild and even moderate cases. It works on the cellular level and increases the health of the digestive system, improves circulation, promotes healthy skin and the sound functioning of the nervous system.

Cayenne

Cayenne has been revered for thousands of years for its healing powers. Cayenne has nutritional attributes as well, being rich in vitamins A and C along with the complete B complex. It is also a source of calcium and potassium, which again benefits the blood and the heart.

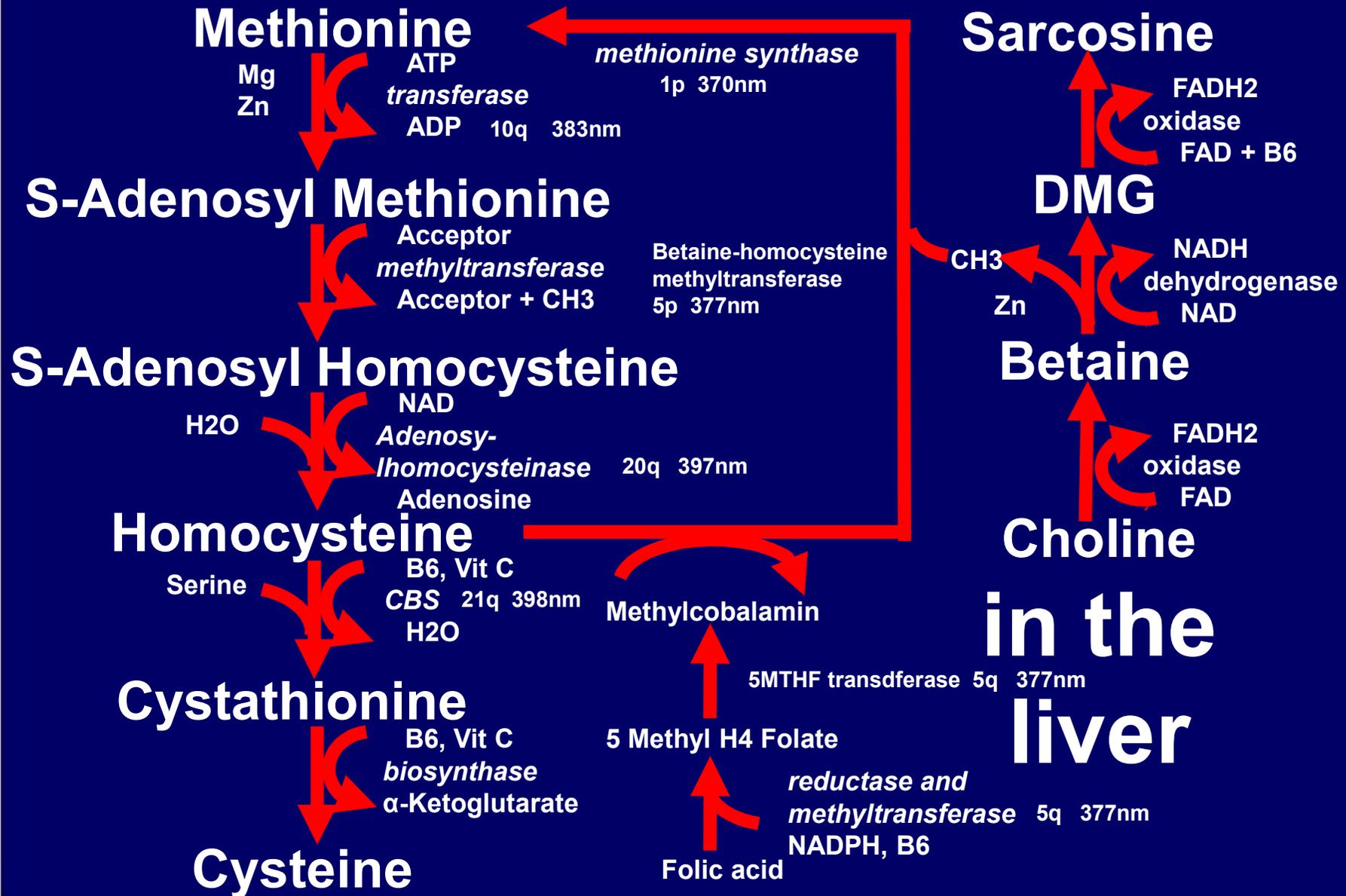
Garlic

Garlic is known to reduce cholesterol as well as defend against bacterial and fungal infections. Garlic contains 40 organic compounds and includes over 100 bioavailable chemicals.

In 1994 Adesh K. Jain, M.D. of the Clinical Research Center at the Tulane University School of Medicine, New Orleans, reported how garlic can lower blood levels of "total cholesterol" and specifically LDL cholesterol.

HOMOCYSTEINEAMIA

S Adenosylmethionine (SAM)



Procollagen is released into the extracellular space (cofactored by Zinc and Vitamin A).

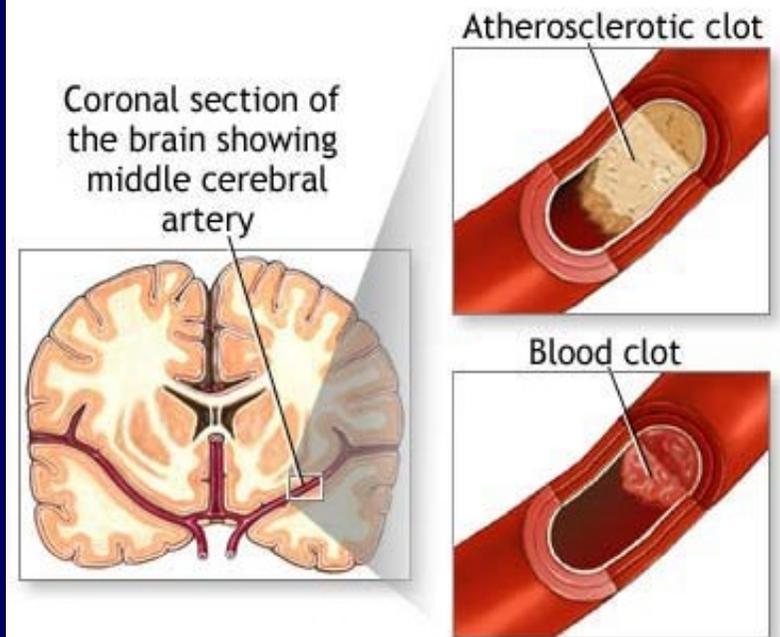
Here it is converted to collagen by peptidase enzymes and strengthened by crosslinking of the microfibrils by lysyl oxidase, a **copper** dependant enzyme which is inhibited by high levels of **homocysteine**.

Effect of Hyperhomocysteinemia on Coagulation

**Sauls DL, Wolberg AS , Hoffman
M: Hyperhomocysteinemia
induces alterations in fibrinogen
function and fibrin clot structure in
a rabbit model. *J of Thromb
Haemostas*, In Press for 2003.**

We are now also studying the mechanism by which elevated plasma homocysteine leads to accelerated atherosclerosis and thrombosis.

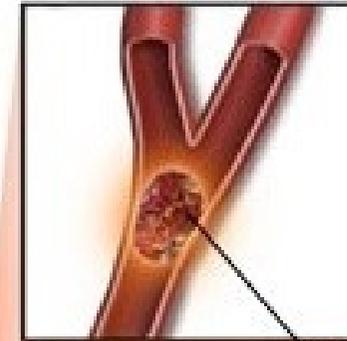
There are two kinds of strokes. An **ischemic stroke** occurs when the blood supply to the brain is interrupted, usually by a blood clot.



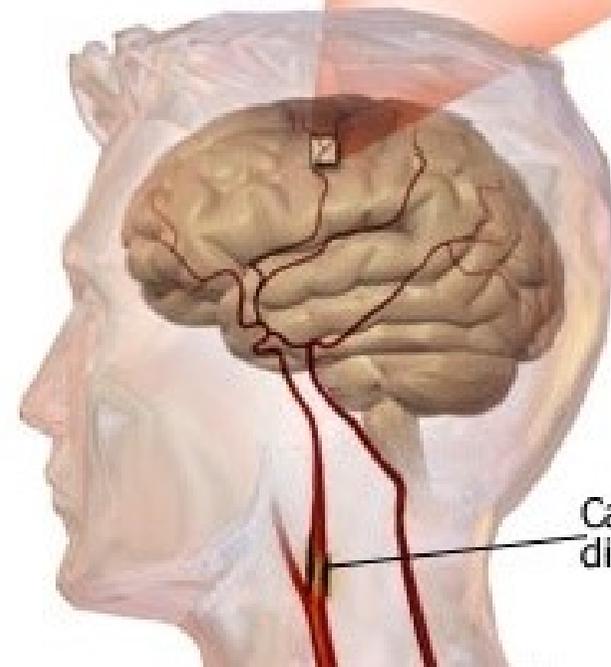
These clots may be caused by “hardening of the arteries” in the carotid arteries, which feed the head and brain with oxygen-rich blood.

Ischemic Stroke

Ischemic stroke is a life-threatening event in which part of the brain does not receive enough oxygen, usually due to a blood clot lodged in a cerebral artery.

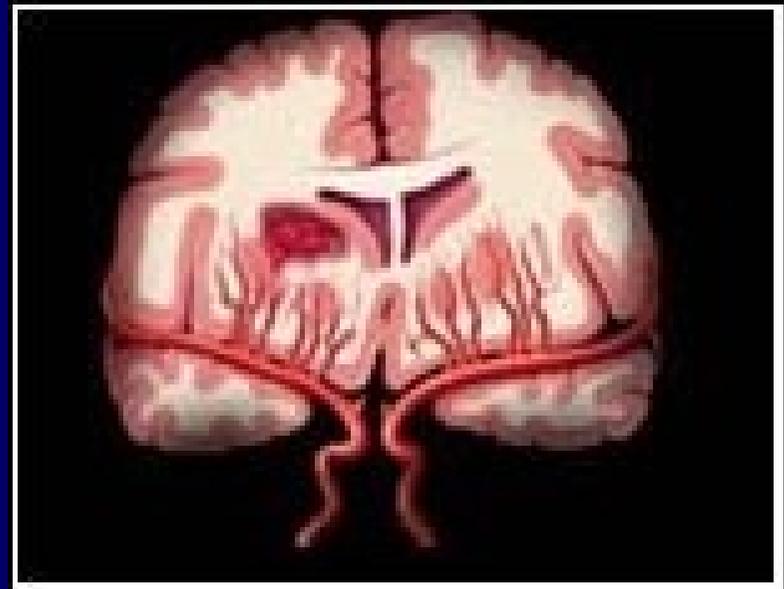


Blood clot



Carotid artery disease

The second kind of stroke is a ***hemorrhagic stroke***, which occurs when there is bleeding into or around the brain.



Ischemic stroke

Probably due to oxidised cholesterol.

Omega 3 to prevent platelet aggregation.

Vitamin K2, PEA, Magnesium, ginger, garlic to stimulate prostacyclins.



Hemorrhagic stroke

Due to weakened artery endothelial cells due to high homocysteine.

**Consider homocysteine factors
P5P, Vit C, Methylcobalamin,
Methyl H4 folate, Betaine, DMG.**

↑ H_2O_2



Lipid
peroxidation



Fe^{++}
 Cu^+

↑ OH^-

Avoid Iron
and Copper
supplements

Post Cerebral Vascular Accident

Patients often weaken to Ammonia.

If positive challenge against

Magnesium

Vitamin K2

Ornithine

Biotin

P5P

Vit C

NAD

Zinc

Manganese

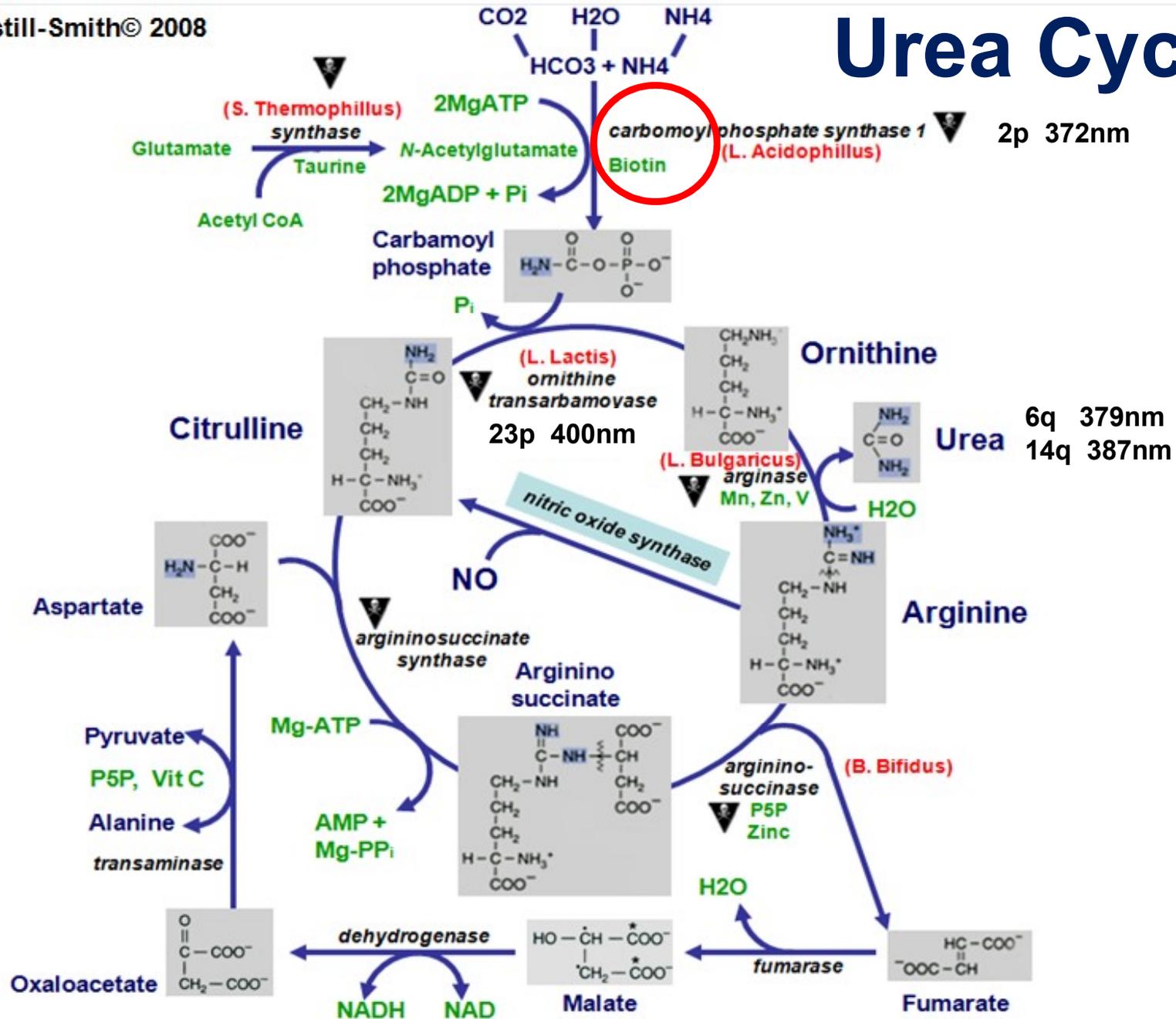
Cloves

Phosphatidylcholine

PEA

Omega 3

Urea Cycle



Symptoms of Hyperammonia

Tremor

Slurred speech

Blurred vision

Joint pains

Muscle pains

Poor sleep

Stinging eyes

Hot flushes

Night sweats

Impaired memory

Cardiac arrhythmia

Skin

Chronic fatigue

pH imbalance

Muscle fasciculations

**Feelings of doom and gloom
especially at night and early
morning on waking.**



Doom →

← **Gloom**

**ASYMMETRICAL
DIMETHYLARGININE-RELATED
RISK OF CARDIOVASCULAR
DISEASES**

Asymmetrical dimethylarginine (ADMA) is an endogenous inhibitor of the nitric oxide (NO) synthase enzymes.

Elevation of serum ADMA has been proven to be a novel independent risk factor for endothelial dysfunction and coronary heart disease.

Markedly high serum ADMA levels have been detected in **hypercholesterolaemia** as well as in congenital heart disease, **hypertension** and renal failure. On autopsy study of atherosclerotic carotid arteries, the thickness of the intima and media has been found to be strongly related to serum ADMA levels.

Tissue accumulation of ADMA is considered a causative factor in the development of **multiple organ failure** acting by critical reducing of nitric oxide production.

Nutritional and Natural medicines

Retinol

Magnesium

Pyridoxal -5-phosphate

(all for the activation of

dimethylarginine

***dimethylaminohydrolase*, the**

enzyme specifically inactivating

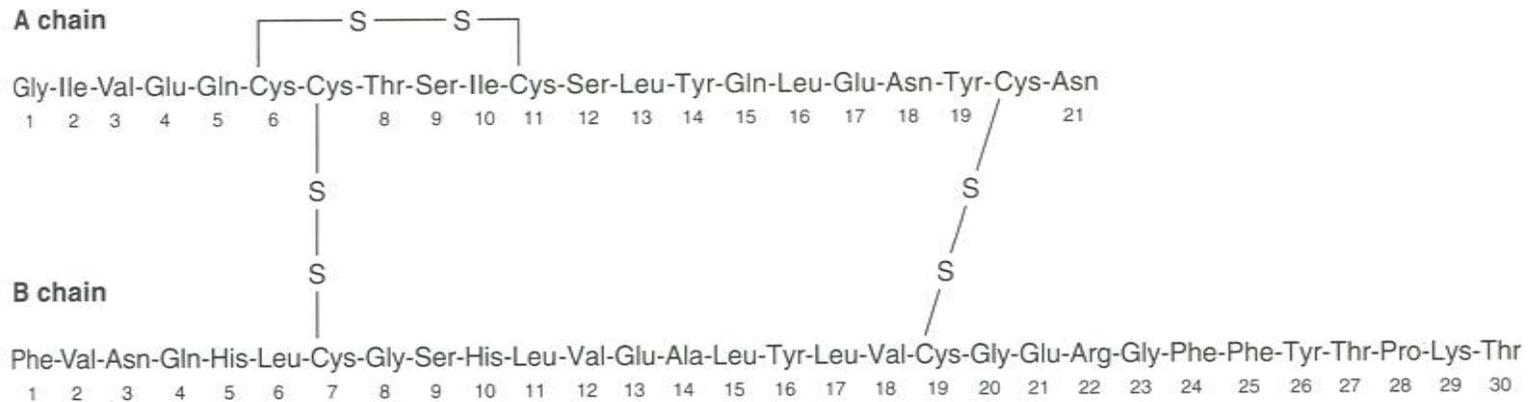
ADMA)

Vitamin B12 hydroxycobalamin
Glycine
Aspartic acid
(all for the stimulation of
demethylation, an alternate
pathway of ADMA degradation).

DIABETES TYPE 1

10% of diabetics have insulin dependant type 1 diabetes mellitus.

Structure of Human Insulin



Insulin is a polypeptide consisting of two chains A and B linked by two inter-chain disulfide bridges and a third connecting residues 6 and 11 of A chain.

Zinc is present in high concentration in the B cells of the pancreas and forms complexes with insulin and proinsulin.

B24 (Phe) and B26 (Tyr) each form dimers containing two atoms of zinc respectively.

Insulin regulation

1. High plasma glucose levels indirectly results in an inhibition of ATP-sensitive K^+ channels causing depolarisation of the B cell and activation of voltage sensitive Ca^{++} channels. The Ca^{++} influx results in insulin secretion.

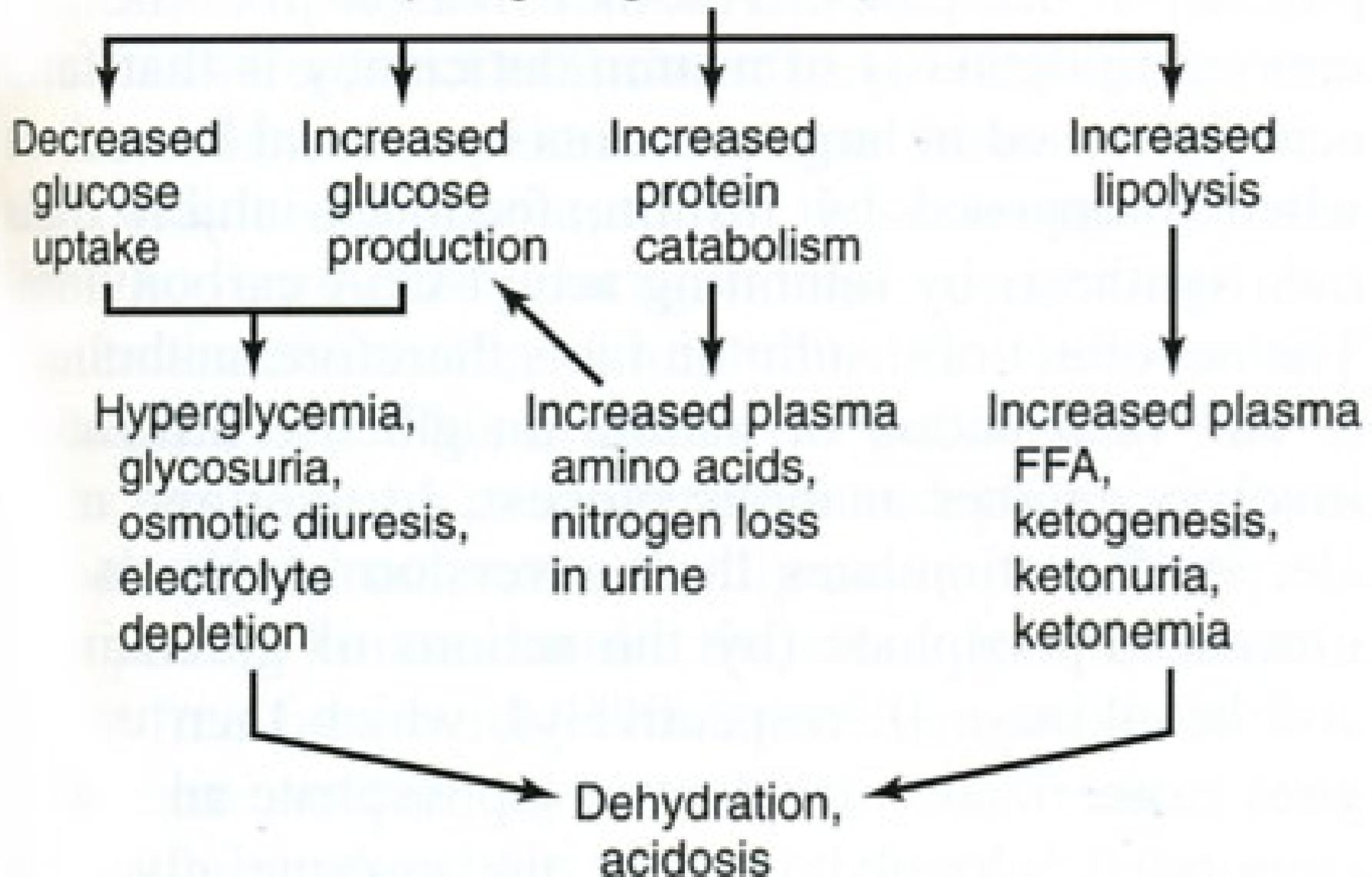
2. Hormone factors

Adrenalin inhibits insulin release. Cortisol, Estrogens, Progestins, Growth hormone, Placental lactogen all increase insulin secretion. (Insulin secretion is higher in the later stage of pregnancy).

3. Pharmaceutical agents

Sulfonylurea compounds stimulate insulin release via the ATP-sensitive K⁺ channels.

Insulin deficiency
(and glucagon excess)



Chlorine + Xanthine



Alloxan

**Potent pancreatic tissue
destroyer**

Nutritional and Natural medicines

Carnitine

Zinc

Manganese

Sulfur (Taurine)

Omega 3 / 6 / 9

DIABETES TYPE 11

90% of diabetics have non insulin dependant type 2 diabetes mellitus.

Such patients are often obese, (only problem if accompanied by high GGT) have elevated plasma insulin levels and have down regulated insulin receptors.

Characteristics of Type 2 Diabetes

Most common in adults, although more younger people are developing this type.

Usually slow onset with thirst, frequent urination, weight loss developing over weeks to months.

Usually runs in families.

Most people who get this type are overweight or obese.

Treatment usually begins with diet and exercise, progressing to use of oral medications and later to insulin as the disease advances.

Blood glucose levels may improve with weight loss, change in diet and increased exercise.

May be prevented or delayed in high-risk individuals by moderate weight loss and exercise.

People at high risk, who already had early signs of impaired glucose tolerance, significantly reduced their risk by losing only **5-7 percent of their body weight** and performing moderate physical activity for 30 minutes/day.

Type 1 diabetes can be a progressive disease that can cause significant, severe complications such as heart disease, kidney disease, blindness and loss of limbs through amputation.

Treatment differs at various stages of the condition. In its early stages, many people with type 11 diabetes can control their blood glucose levels by losing weight, eating properly and exercising.

Many may subsequently need **oral medication**, and some people with type 2 diabetes may eventually need insulin shots to control their diabetes and avoid the disease's serious complications.

Patients with diabetes have an approximately **threefold risk for all cardiovascular diseases and their relative risk of death from all causes is increased by 75%.**

As yet there is **no conclusive evidence** that improved glucose control with oral agents leads to a decrease in the complications of type 11 diabetes. There is some evidence that improved glucose control delays the onset of complications in type 11 diabetes.

In contrast, there is strong evidence that near-normalisation of blood glucose levels with **insulin can delay the development and progression of retinopathy, nephropathy, and neuropathy of patients with type 1 diabetes mellitus (IDDM).**

The risk factors for diabetes are age (≥ 45 years), family history (first degree relative with diabetes), high-risk ethnic group (Aboriginal, Asian, Pacific Islander, Hispanic, African), obesity (BMI ≥ 27 kg/m²), history of gestational diabetes or macrosomic infant (≥ 4.5 kg), hypertension, coronary artery disease.

Sulfonylureas increase insulin secretion and potentiate insulin action on the liver and peripheral tissues.

Metformin decreases hepatic glucose production, increases glucose uptake and possibly decreases appetite.

Alpha glucosidase inhibitors slow the absorption of carbohydrates.

White kidney
bean extract
(Phasiolamine)

Troglitazone decreases insulin resistance.

Nutritional and Natural medicines

α -lipoic acid

Biotin

Chromium

Manganese

Zinc

Vanadium

Glucosamine

Cinnamon

Gymnema

sylvestre

Bilberry

Cinnamon

USDA research indicates that Cinnamon reduces the amount of insulin necessary for glucose metabolism. Furthermore, Cinnamon has been shown to stimulate glucose uptake and glycogen synthesis to similar level as insulin.

Gymnema Sylvestre

A Harvard study indicates the Gymnema can lower blood sugar levels in Type 1 and Type 11 diabetics. A King's College, London, study states that Gymnema acts by increasing cell permatibility, therefore reducing insulin resistance.

Bilberry

Helps to improve circulation in the little capillaries in the hands and feet. Also valuable in balancing the digestion and strengthening the immune system.

Chromium

Starting in the 1960's reports have shown that Chromium helps cells respond properly to naturally produced insulin. Current estimates show that 90% of Americans are lacking in this essential mineral nutrient.

Chromium helps to normalize blood sugar, potentiating the action of insulin (Glucose Tolerance Factor) and plays an important role in the metabolism of fats and carbohydrates.

Zinc

Zinc contributes many factors in correct insulin function. Zinc is necessary for the pancreas to produce insulin and allows insulin to work effectively, it also helps protect the insulin receptor cells.

When zinc levels are low, the pancreas may not secrete enough insulin, so glucose levels remain high, and the insulin that is released cannot work as efficiently as it could. When this happens, glucose cannot enter the cells properly causing high levels in the blood.

Biotin

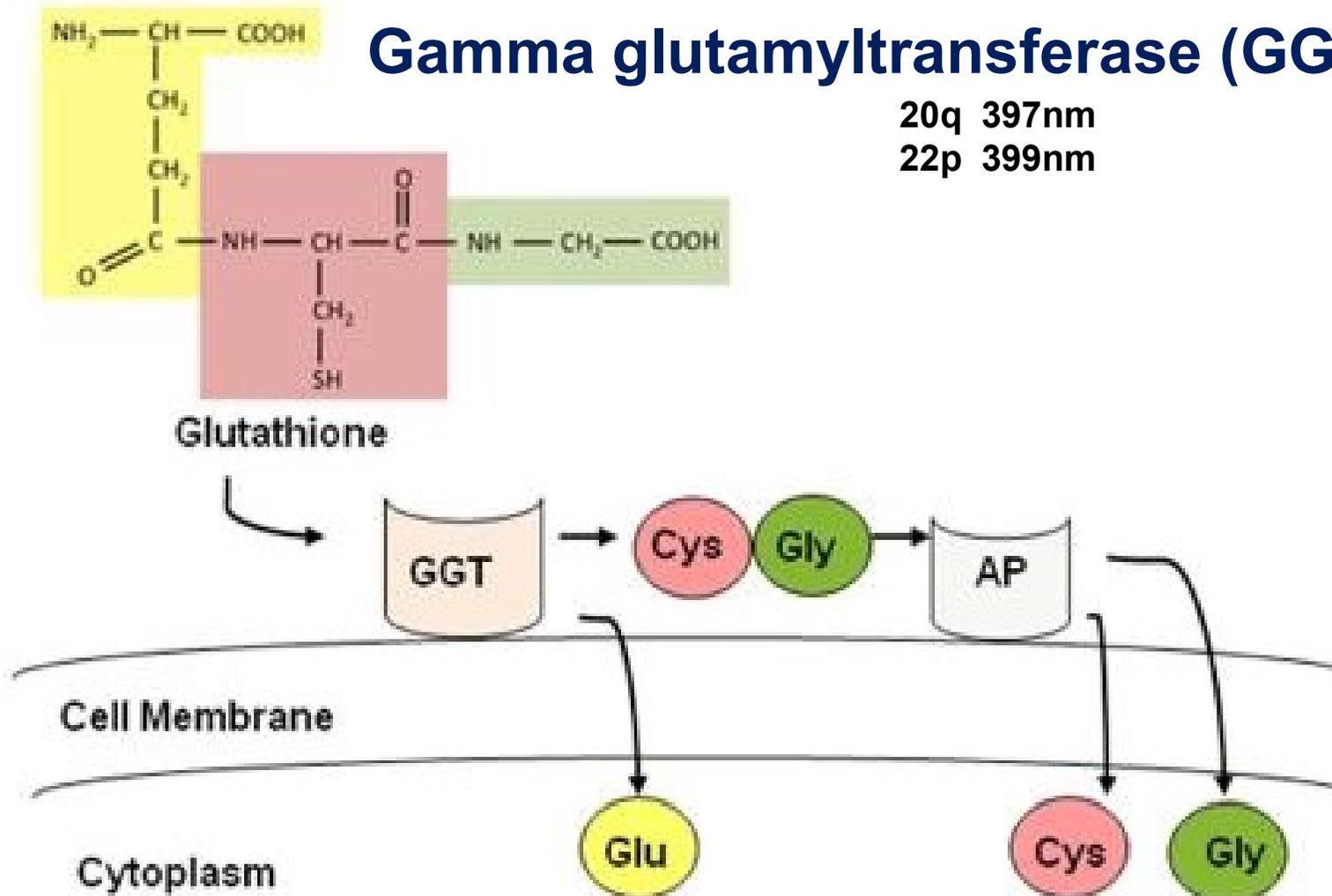
Biotin is a B vitamin needed to process glucose. One study showed that Type 1 diabetics given 16mgs of Biotin for one week cut their fasting glucose by 50%. A type 11 study showed similar results. There is also some indication that Biotin helps relieve pain from diabetic nerve damage.

Gamma glutamyltransferase (GGT)

Gamma glutamyltransferase (GGT)

20q 397nm

22p 399nm



Hydrolysis of extracellular glutathione by GGT. GGT releases glutamate and cysteinyl-glycine. Cysteinyl-glycine hydrolysed by aminopeptidase (AP) releasing cysteine and glycine. All three amino acids can then be taken up into the cell to synthesise glutathione but process not very efficient. Glutathione cannot be taken up intact in most cells.

Gammaglutamyltransferase (GGT)

is a transferase enzyme that catalyzes the transfer of gamma-glutamyl functional groups from molecules such as glutathione to an acceptor that may be an amino acid, a peptide or water (forming glutamate).*

**Tate SS, Meister A (1985). "gamma-Glutamyl transpeptidase from kidney". *Methods in Enzymology*. 113: 400–19*

Research indicates that GGT can also exert a **pro-oxidant role**, with regulatory effects at various levels in cellular signal transduction and cellular pathophysiology.*

** Dominici S, Paolicchi A, Corti A, Maellaro E, Pompella A (2005). "Prooxidant reactions promoted by soluble and cell-bound gamma-glutamyltransferase activity". *Methods in Enzymology*. 401: 484–501.*

GGT is present in the cell membranes of many tissues, including the kidneys, bile duct, pancreas, gallbladder, spleen, heart, brain, prostate and seminal vesicles. Smaller amounts are found in the lungs, testis, and thyroid gland.*

* Raulf M, Stüning M, König W (May 1985). "Metabolism of leukotrienes by L-gamma-glutamyl-transpeptidase and dipeptidase from human polymorphonuclear granulocytes". *Immunology*. 55(1): 135–47.

It is involved in the transfer of amino acids across the cellular membrane and **leukotriene metabolism***.

* Raulf M, Stüning M, König W (May 1985). "Metabolism of leukotrienes by L-gamma-glutamyl-transpeptidase and dipeptidase from human polymorphonuclear granulocytes". *Immunology*. 55(1): 135–47.

GGT alone does not directly cause a particular disease or disorder. High levels of GGT may contribute to disease by acting a **pro-oxidant**. GGT may increase oxidative stress, starting with the breakdown of glutathione (and production of cysteinylglycine). Other toxic molecules are then formed, leading to tissue, cellular, and DNA damage.*

*Human Atherosclerotic Plaques Contain Gamma-Glutamyl Transpeptidase Enzyme Activity Aldo Paolicchi, Michele Emdin

GGT is present in plaques because it attaches itself to circulating fats (LDL). Once in the plaque, GGT can become pro-oxidant, injuring blood vessels (via **oxidative stress**), and contribute to heart disease.*

*Human Atherosclerotic Plaques Contain Gamma-Glutamyl Transpeptidase Enzyme Activity Aldo Paolicchi, Michele Emdin

Gamma-glutamyltransferase (GGT)

High in

**Alcohol abuse, Barbituates, NSAIs,
Aspirin, St John's Wort.**

Biliary, Liver and Pancreas diseases

CVD and Atherosclerosis

**Metabolic syndrome. High body mass
index is associated with type 2
diabetes only in persons with high
serum GGT.**

Lim JS, Lee DH, Park JY, Jin SH, Jacobs DR (June 2007). "A strong interaction between serum gamma-glutamyltransferase and obesity on the risk of prevalent type 2 diabetes: results from the Third National Health and Nutrition Examination Survey". Clinical Chemistry. 53 (6): 1092–8. doi:10.1373/clinchem.2006.079814. PMID 17478563.

High GGT can cause

Liver disease

Biliary tract disease

CHD

CVA

Arteriosclerosis

Heart failure

High BP

Cardiac arrhythmias

Diabetes

Metabolic-
syndrome

Cancer

Kidney disease

Alzheimer's

Thyroid

Bone density loss

To lower GGT levels

Decrease alcohol

Avoid pollutants

More fruit and veg

High protein

More coffee

Less red meat

Moderate exercise

Cloves

Curcumin

Vitamin C

Vitamin D

Vitamin E

Fish/Flax/DHA

Milk thistle

Magnesium

Zinc

Glutathione

Nine of the best foods and supplements you can consume to boost glutathione include:

- 1. Milk thistle**
- 2. Whey protein**
- 3. High sulfur foods including cruciferous vegetables**
- 4. N-acetyl cysteine (NAC)**
- 5. alpha lipoic acid**
- 6. Methylation nutrients like vitamins B6, B9, B12 and biotin**
- 7. Selenium-rich foods like Brazil nuts and sardines**
- 8. Vitamin C and vitamin E**
- 9. Raw liver from organic grass-fed cows**

ARTERIOSCLEROSIS

Atherosclerosis is a type of arteriosclerosis. It comes from the Greek words athero (meaning gruel or paste) and sclerosis (hardness). It involves deposits of **fatty substances, cholesterol, cellular waste products, calcium and fibrin** (a clotting material in the blood) in the inner lining of an artery.

Atherosclerosis can affect the arteries of the brain, heart, kidneys, other vital organs, and the arms and legs. When atherosclerosis develops in the arteries that supply the brain (carotid arteries), a stroke may occur; when it develops in the arteries that supply the heart (coronary arteries), a heart attack may occur.

In the United States and most other Western countries **including the UK**, atherosclerosis is the leading cause of illness and death. In the United States alone, it caused almost 1 million deaths in 1992--twice as many as from cancer and 10 times as many as from accidents.

Despite significant medical advances, coronary artery disease (which results from **atherosclerosis** and causes myocardial infarction) and atherosclerotic stroke are responsible for more deaths than all other causes combined.

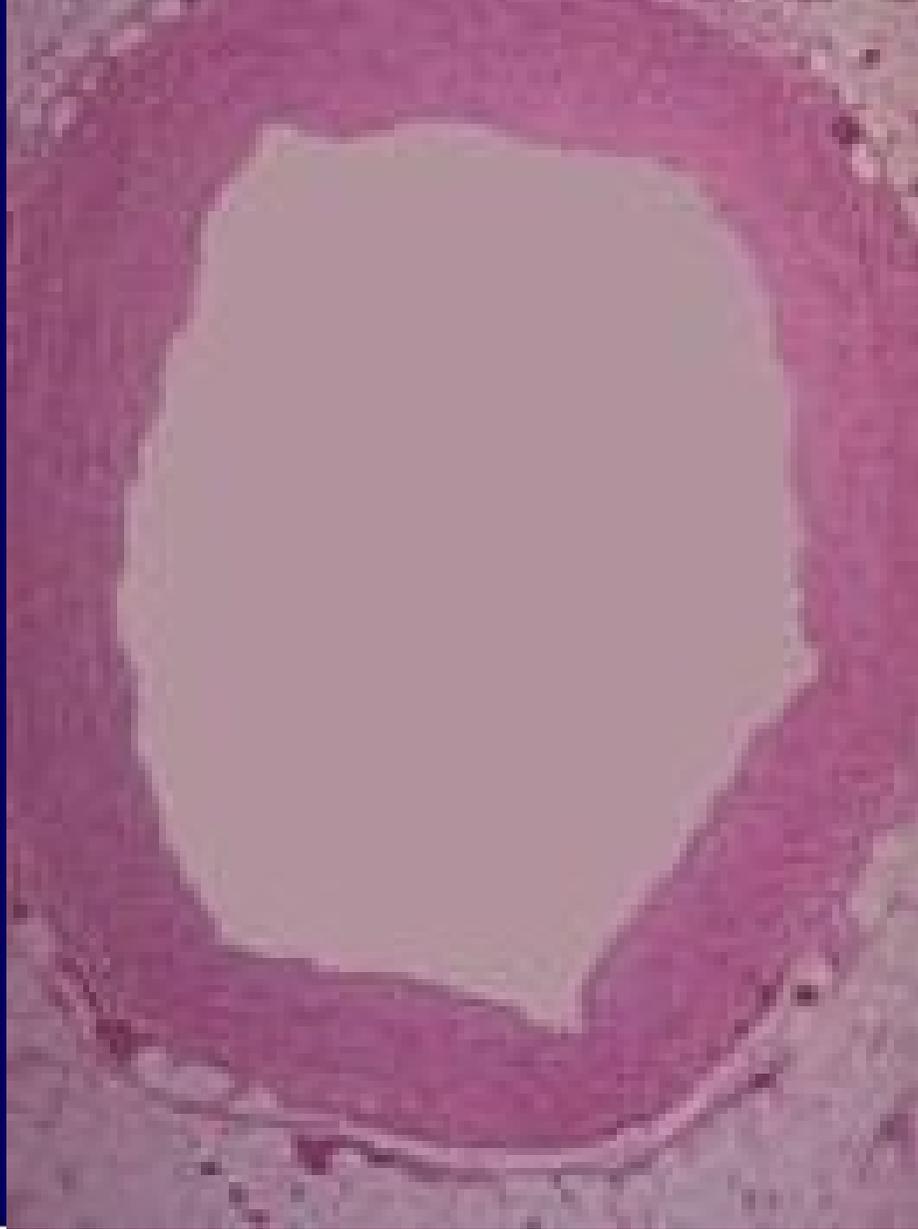
It is a disease of the arterial intima leading to the formation of fibrous (atheromatous) plaques and to stenosis/ occlusion of the lumen. It involves the proliferation of smooth muscle cells and the accumulation of lipids.

Atherosclerosis affects large and medium-sized arteries.

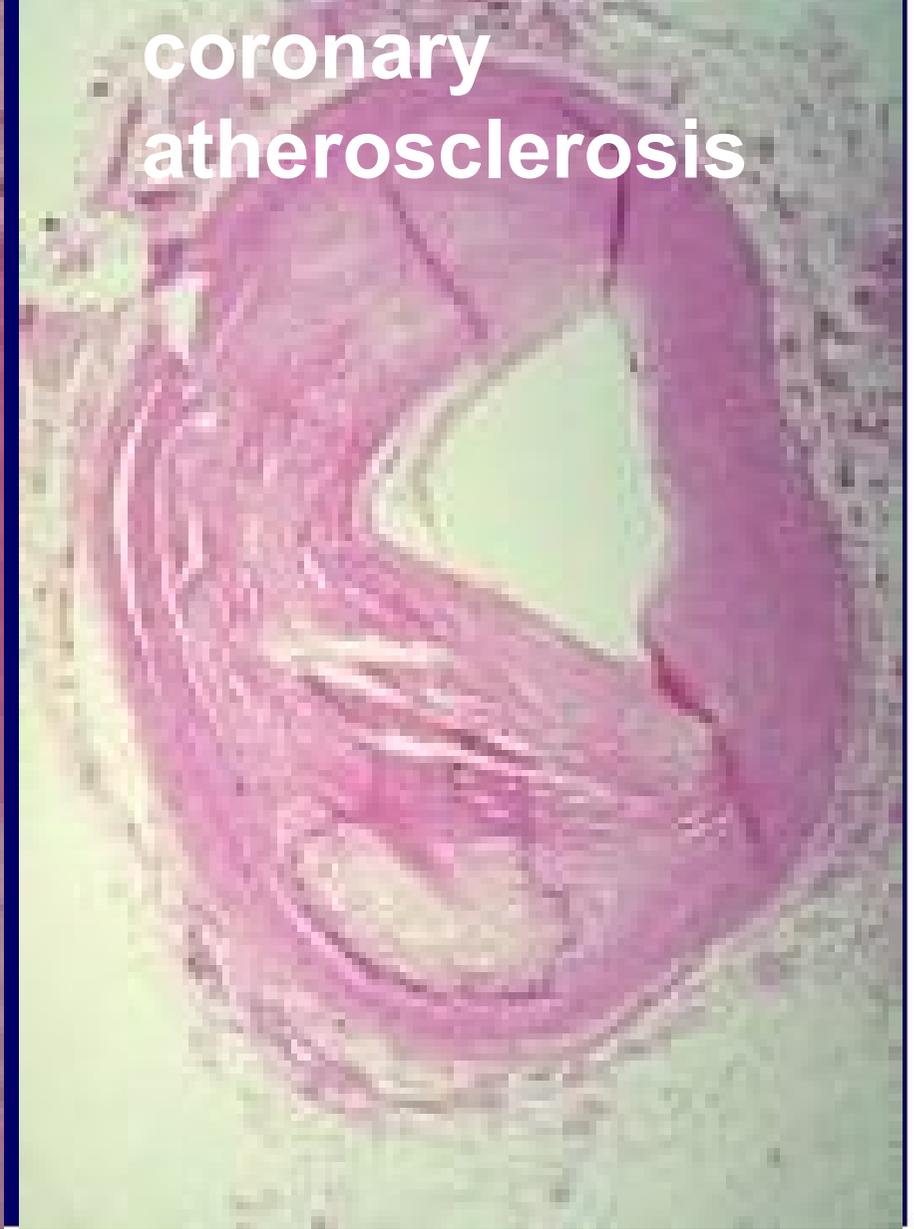
Atherosclerosis is a slow, progressive disease that may start in childhood. In some people this disease progresses rapidly in their third decade.

In others it doesn't become threatening until they're in their fifties or sixties.

Normal coronary artery



**Severe calcified
coronary
atherosclerosis**



What causes Atherosclerosis

- Blood vessels lose a certain amount of elasticity with aging.
- A build up of fatty deposits (plaque) occurs in the blood vessel lining.
- Loss of vessel elasticity is termed **arteriosclerosis**, while fatty deposit build-up is termed **atherosclerosis**.

- **The process is thought to begin early in life.**
- **Cigarette smoking is thought to be a causative factor.**
- **High blood pressure is thought to be a causative factor.**
- **Diabetes is thought to be a causative factor.**
- **Obesity is thought to be a causative factor.**

Atherosclerosis begins when white blood cells called **monocytes** migrate from the bloodstream into the wall of the artery and are transformed into cells that accumulate fatty materials. In time, these fat-laden monocytes accumulate, leading to a patchy thickening in the inner lining of the artery.

Each area of thickening (called an **atherosclerotic plaque** or atheroma) is filled with a soft cheese-like substance consisting of various fatty materials, principally cholesterol, smooth muscle cells, and connective tissue cells.

Atheromas may be scattered throughout the medium and large arteries, but usually they form where the arteries branch off—presumably because the constant turbulence at these areas injures the arterial wall, making it more susceptible to atheroma formation.

Arteries affected with atherosclerosis lose their elasticity, and as the atheromas grow, the arteries narrow. With time, the atheromas collect **calcium deposits**, may become brittle, and may rupture. Blood may then enter a ruptured atheroma, making it larger, so that it narrows the artery even more.

A ruptured atheroma also may spill its fatty contents and trigger the formation of a blood clot (thrombus). The clot may further narrow or even occlude the artery, or it may detach and float downstream where it causes an occlusion (embolism).

Atherosclerosis causes harm by:

- Occluding the arteries slowly over time.
- Occluding the arteries suddenly by rupture of plaques.
- Weakening of walls of the arteries.
- Arterial changes occurring with age:
- Intimal thickening.
- Reduction of elasticity of elastic tissue.
- Changes in lipoprotein composition.

Many researchers think atherosclerosis begins because the innermost layer of the artery, **the endothelium**, becomes damaged. Over time, fats, cholesterol, fibrin, platelets, cellular debris and calcium are deposited in the artery wall.

These substances may stimulate the cells of the artery wall to produce still other substances that result in further accumulation of cells in the innermost layer of the artery wall where the atherosclerotic lesions form. These cells accumulate and many of them divide.

At the same time, fat builds up within these cells and around them. They also form connective tissue. The innermost layer of the artery becomes markedly thickened by these accumulating cells and surrounding material.

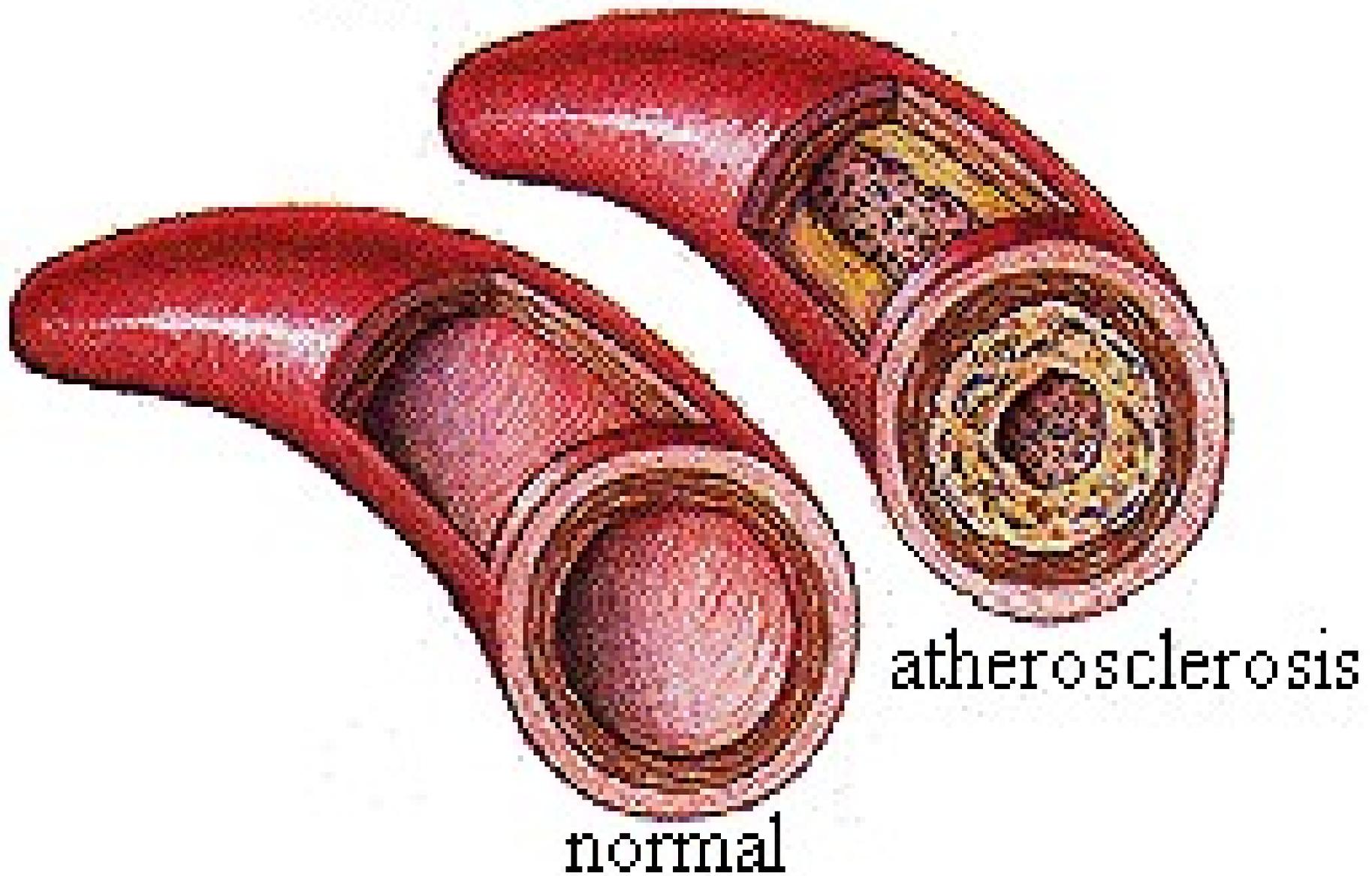
If the wall is thickened sufficiently, the diameter of the artery will be reduced and the amount of blood decreased, thus decreasing the oxygen supply. **If the oxygen supply** to the heart muscle is reduced, a heart attack can occur. If the oxygen supply to the brain is cut off, a stroke can occur.

Researchers are studying other ways in which **platelets** may play a role in atherosclerosis. For example, they're involved in forming a group of substances called prostaglandins, one of which may damage arteries.

They also contain a substance called "platelet growth factor," which can stimulate the growth of smooth muscle cells.

These cells are normally present in the artery wall. But their **abnormal growth** and increase is believed to be one of the earliest events in the atherosclerosis process.

Blood Vessels



Homocysteine is an amino acid in the blood. Epidemiological studies have shown that too much homocysteine is a risk factor for atherosclerosis. People in the highest 25 percent of homocysteine blood level may be at increased risk.

One of the more recent theories suggests that excess lipoproteins, LDL, in the blood are trapped within the artery wall. When this happens and they accumulate, they become oxidized.

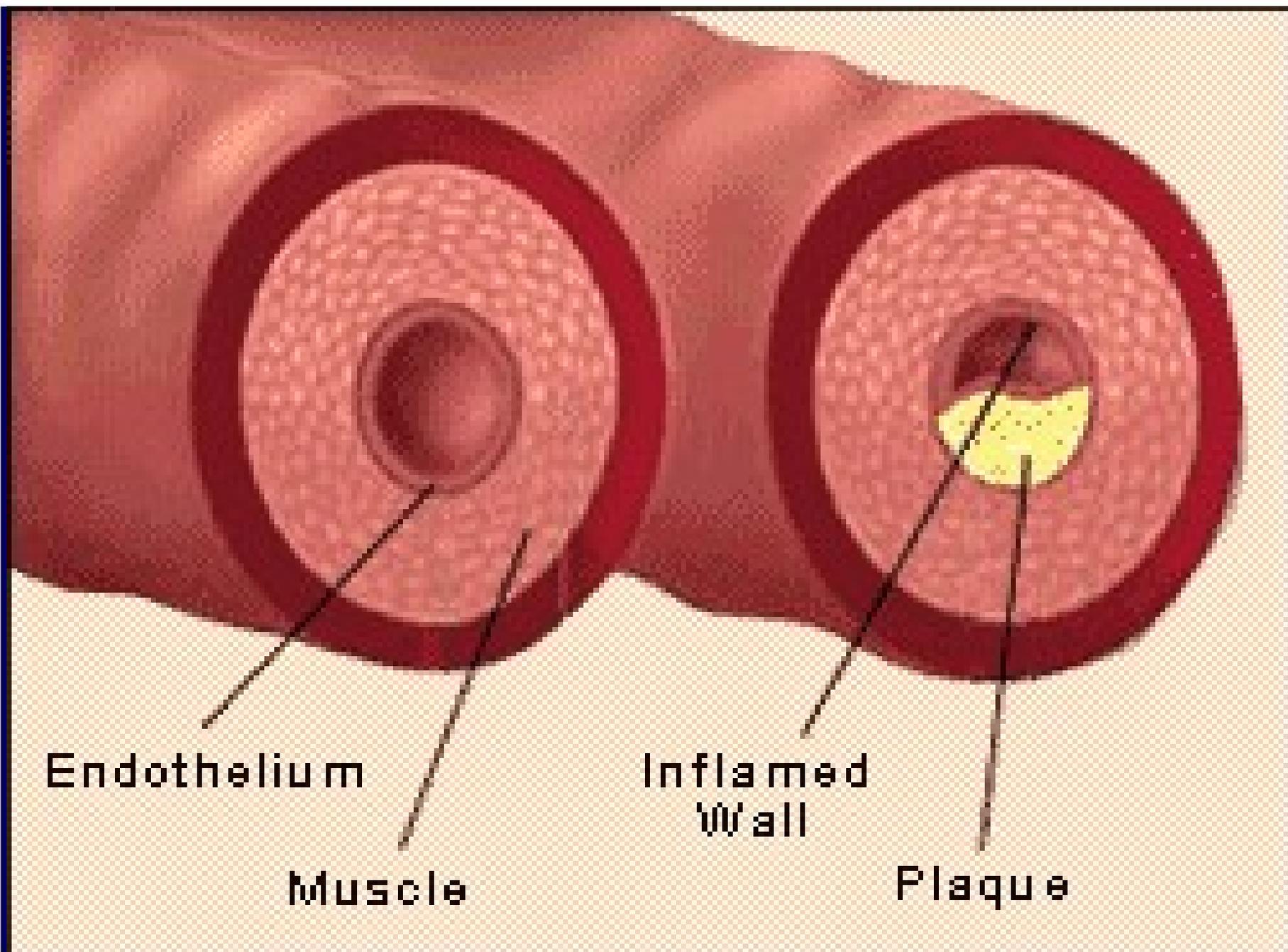
That leads to **"modified"** lipoproteins which are rapidly taken up by smooth muscle cells. This, in turn, leads to the formation of foam cells that lead to the deposition of connective tissue cells and elements.

LDL contains specific functional groups which allow it to be recognised by most cells in the body and remain soluble in blood plasma. Therefore LDL readily passes through the endothelium, contributing to the development of plaques, atheromas.

Over time the **fat deposits accumulate** and grow, narrowing the lumen of the artery. Subsequent damage to the endothelial wall causes blood platelets to adhere and contributes to blood clot formation. Surrounding smooth muscle tissue also proliferates to form larger plaques.

Hardening of the arterial wall is due to **various depositions** within the plaque including lipids, cholesterol crystals, and calcium salts. These depositions make the arteries bone-like rigid tubes.

The narrowing and hardening of the arteries has dramatic effects on blood pressure, resistance and blood flow.



Endothelium

Muscle

Inflamed
Wall

Plaque

Resistance increases when radius decreases, as friction of blood flow against vessel wall increases.

Therefore the circulation of blood flow is reduced, and cells may be deprived of oxygen or experience toxic accumulation of metabolic wastes.

Development of a plaque also deforms the endothelial wall, increasing turbulent flow and increasing resistance. The hardening of the arterial walls, increases resistance to flow, as vessel walls lose their distensibility.

As resistance to flow increases, there is a marked increase in **blood pressure**. Therefore the heart has to work harder to pump blood, causing it to enlarge. This may lead to various heart defects and failures.

Atheromas

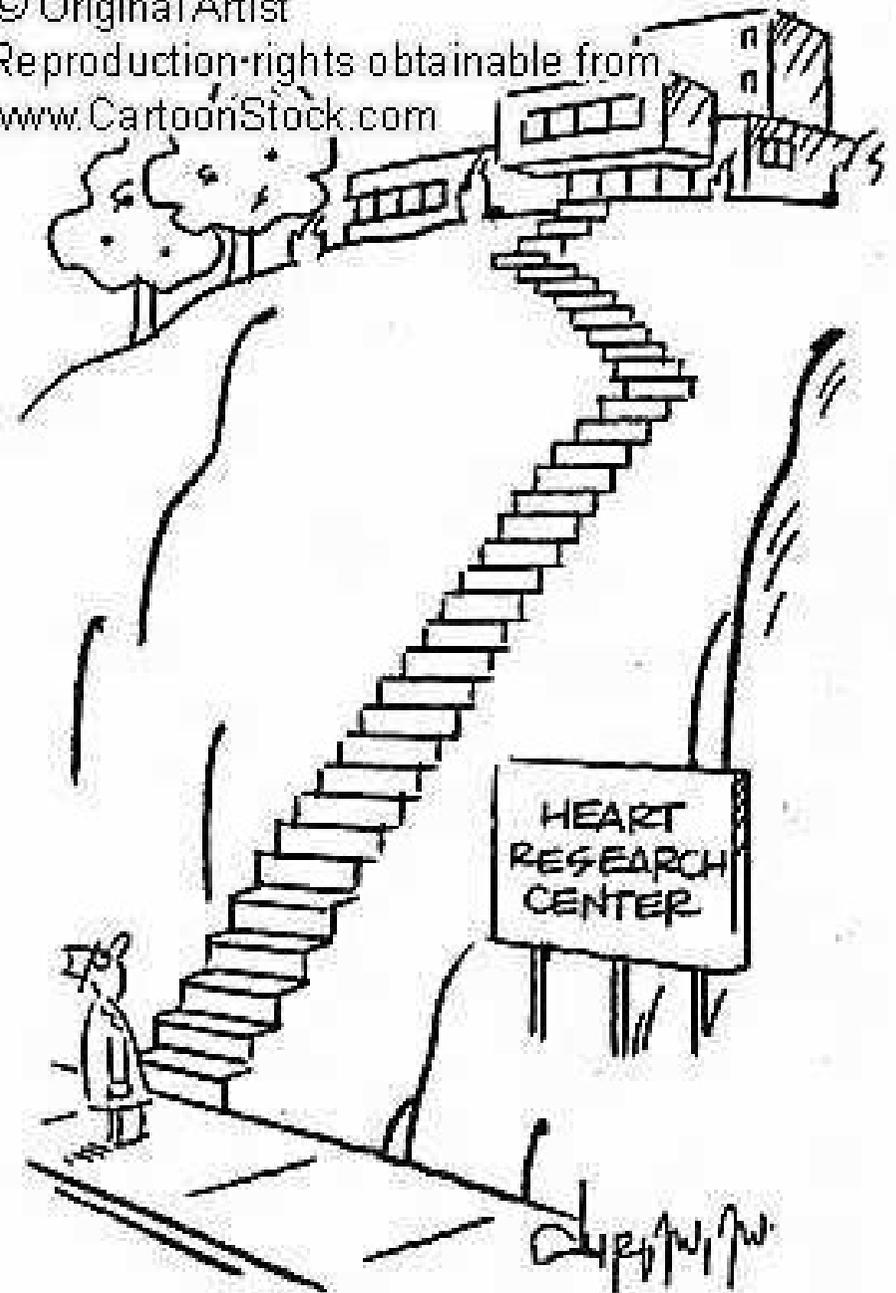
Atherosclerosis is a stereo typed response to injury featuring the accumulation of cholesterol-rich fat in the intima of the large and medium sized arteries of the body. Typically these are phagocytes. These masses form plaques, or atheromas.

Lipid streaks are flat or slightly elevated pale yellow areas, of variable size and shape, found throughout the arterial system of patients of all ages. The lipid is deposited in the intima and can be stained with Sudan IV and other fat stains.

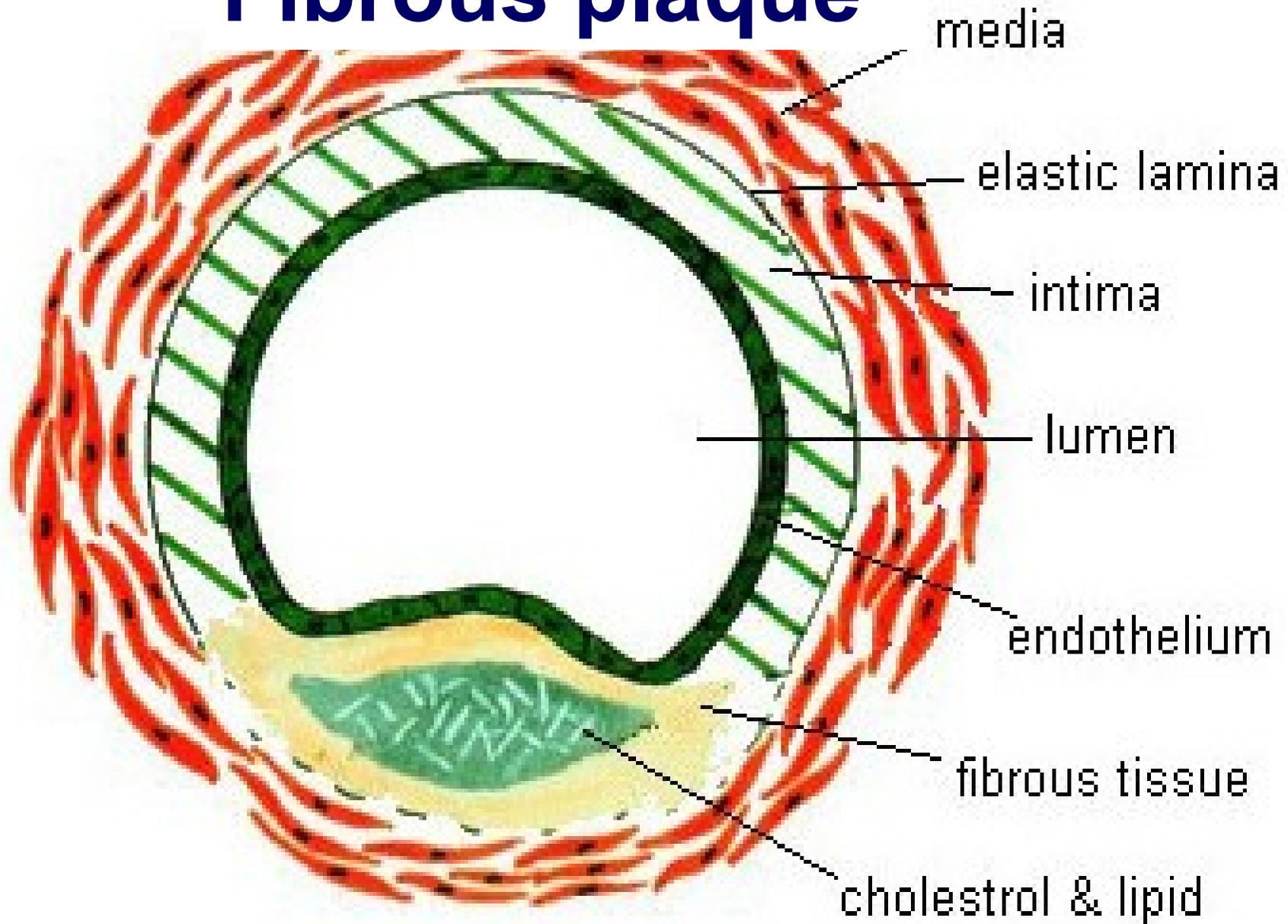
Fibrous plaques are raised firm pale areas in the intima of arteries which on cross section reveal central lipid rich debris with surrounding fibrous tissue.

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Fibrous plaque



Calcification



Hemorrhage



Ulceration

A local defect or excavation, of the surface of an organ or tissue, which is produced by the sloughing of inflammatory necrotic tissue.



Thrombosis. The formation, development or presence of a thrombus. An aggregation of blood factors, primarily platelets and fibrin with entrapment of cellular elements, frequently causing vascular obstruction at the point of its formation. Some authorities thus differentiate thrombus formation from simple coagulation or clot formation.



Symptoms

Usually, atherosclerosis doesn't produce symptoms until it severely narrows the artery, or until it causes a sudden obstruction. Symptoms depend on where the atherosclerosis develops; thus, they may reflect problems in the heart, the brain, the legs, or almost anywhere in the body.

As atherosclerosis **severely narrows an artery**, the areas of the body it serves may not receive enough blood, which carries oxygen to the tissues. The first symptom of a narrowing artery may be pain or cramps at times when the blood flow can't keep up with the body's demand for oxygen.

For instance, **during exercise**, a person may feel chest pain (angina) because of a lack of oxygen to the heart, or while walking, a person may feel leg cramps (intermittent claudication) because of a lack of oxygen to the legs.

Typically, these symptoms develop gradually as the atheroma slowly **narrows the artery.**

However, when an obstruction occurs suddenly, for example, when a blood clot lodges in an artery, the symptoms come on suddenly.

Risk Factors

The risk of developing atherosclerosis increases with high blood pressure, high blood cholesterol levels, cigarette smoking, diabetes, obesity, a lack of exercise, and advancing age.

Having a close relative who developed atherosclerosis at an early age also puts a person at risk. **Men** have a higher risk than women, though after menopause, the risk increases in women and eventually equals that in men.

People with the inherited disease **homocystinuria** develop extensive atheroma formation, particularly at a young age.

The disease affects many arteries but doesn't primarily affect the **coronary arteries**, which supply the heart. In contrast, in the inherited disease familial hypercholesterolemia, extremely high levels of blood cholesterol cause atheromas to form in the coronary arteries much more than in other arteries.

Prevention and Treatment

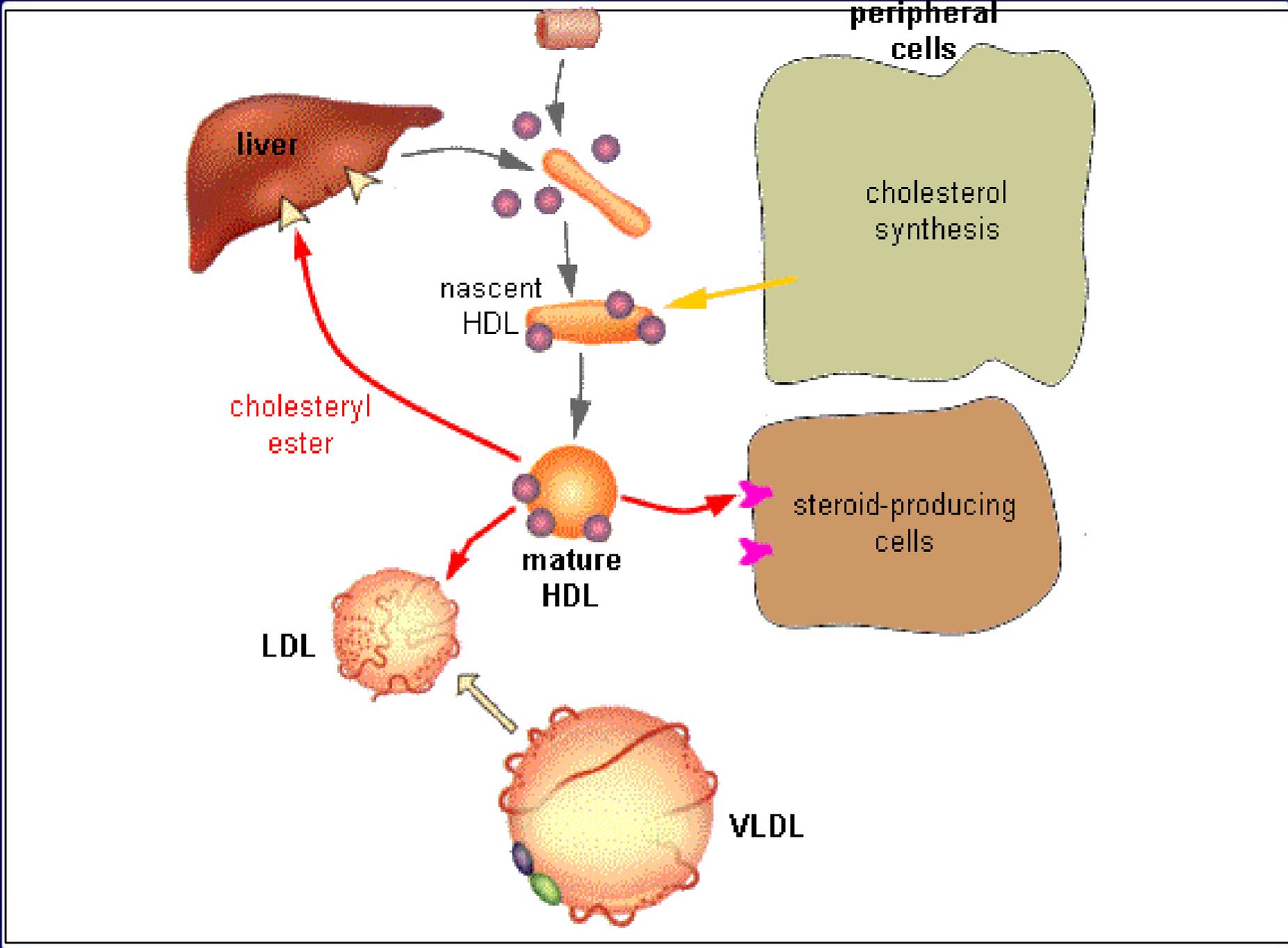
Depending on a particular person's risk factors, prevention may consist of lowering cholesterol levels, lowering blood pressure, quitting smoking, losing weight, and beginning an exercise program.

In people who already have a high risk of heart disease, smoking is particularly dangerous. **Cigarette smoking** decreases the level of good cholesterol (high-density lipoprotein cholesterol or HDL cholesterol) and increases the level of bad cholesterol (low-density lipoprotein cholesterol or LDL cholesterol).

Smoking also raises the level of **carbon monoxide** in the blood, which may increase the risk of injury to the lining of the arterial wall, and smoking constricts arteries already narrowed by atherosclerosis, further decreasing the amount of blood reaching the tissues.

Plus, smoking increases the blood's tendency to clot, so it increases the risk of peripheral arterial disease, coronary artery disease, stroke, and obstruction of an arterial graft after surgery.

A smoker's risk of coronary artery disease is directly related to the number of cigarettes smoked daily.



There are several potential mechanisms by which an intake of saturated fat could lead to increased blood cholesterol:

unsaturated fatty acids tend to favour the formation of HDLs. Presumably this is linked to the reaction involving the enzyme lecithin cholesterol acyl transferase (LCAT) in which unsaturated fatty acids are transferred from plasma lecithin (a phospholipid) onto the cholesterol molecule to form an ester. HDL then transports this cholesterol to the liver where it is broken down. Hence unsaturated fatty acids would tend to reduce plasma cholesterol.

Conversely saturated fatty acid or low levels of unsaturated fatty acid would elevate cholesterol levels in plasma because HDL levels relative to LDL have decreased. low-density lipoprotein (LDL) particles tend to contain cholesterol esters rich in saturated fatty acids, whereas high-density lipoprotein (HDL) contains unsaturated fatty acids. Thus a high proportion of saturated fatty acids in the diet could (in theory at least) increase LDLs saturated fatty acids and cholesterol tend to be present together in foods such as animal fat. fatty acid.

Hence consumption of food rich in saturated fatty acids would raise cholesterol levels concurrently.

(However, a recent study by Kromhout (1999) indicates that this does not apply in the case of stearate which does not raise LDL) saturated fatty acids may have some regulatory effects on cholesterol synthesis which tends to raise plasma cholesterol levels (via LDL), an effect which is not produced by unsaturated fatty acid.

There is clear epidemiological evidence that diets rich in cholesterol and saturated fats lead to an increased risk of coronary heart disease as a consequence of high plasma cholesterol levels promoting the development of atherosclerotic plaques in blood vessel walls. Polyunsaturated fatty acids are regarded as being protective because they reduce the risk of atheroma development.

Saturated fats taken in with the diet are incorporated into cholesterol esters (and triglycerides) which form part of the LDL complexes in the blood, whereas polyunsaturated fatty acids are taken up mainly into cholesterol esters in HDL. The chemical composition of these two particles is quite different: LDL contains 80% fat, of which 50% is made up of cholesterol, and 20% protein. HDL contains 55% fat, of which 25% is cholesterol, and 45% protein. Increased levels of LDL have been linked to an increased risk of coronary heart disease and led to its description as "bad cholesterol".

Conversely increased HDL levels reduce the risk of coronary heart disease and HDL is referred to as "good cholesterol". Since cholesterol is an essential component of cells and has numerous other roles throughout the body, for example providing a raw material for the synthesis of steroid hormones, we must be careful in the way we attach such labels and keep in mind the need for an appropriate balance.

A large proportion of LDL cholesterol is in the form of esters containing saturated fatty acids, whereas HDL contains unsaturated fatty acids. HDL lipoproteins tend to transport excess cholesterol from peripheral tissues back to the liver where it is broken down. HDL is assembled from apolipoprotein-AI, phospholipid, and free cholesterol discs secreted by the liver and intestines. The free cholesterol is esterified in the mature HDL and can then be transferred to LDL or taken up by the liver or steroid-metabolising tissues.

In contrast, LDL particles are involved in the uptake of cholesterol into tissue cells via a receptor-mediated process. A decrease in the number of LDL receptors on tissue cells has been linked to high circulating levels of plasma cholesterol. Decreased receptors means decreased cholesterol uptake and the raised LDL levels that ensue increase the risk of atheroma.

About 20 years ago it was discovered that mutations in the gene that encodes the LDL receptor protein causes familial hypercholesterolaemia. In this condition there are high levels of LDL but tissue cells are unable to take up cholesterol from the blood. People affected by this mutation are at a much higher risk of coronary heart disease and stroke than other people (Brown 1984).

Several factors can influence the levels of circulating HDL (Scott, 1999). For example, there is a difference in levels between women and men: women have higher levels of HDL than men up until the menopause, and this offers them some protection against heart disease. Levels of HDL can also be increased by exercise, weight loss, moderate alcohol intake, and chemicals such as fibric acid derivatives, nicotinic acid, and tamoxifen. It is currently thought that monounsaturated fatty acids may be beneficial to our health by reducing levels of LDL-cholesterol in the blood. *Myristic acid* is the main saturated fatty acid associated with high blood cholesterol levels.

The n-3 fatty acids can reduce the levels of triglycerides in the blood. The n-6 fatty acids can reduce the amount of LDL-cholesterol circulating in the blood; this is sometimes accompanied by a small rise in HDL-cholesterol, but the overall effect is an increase in the HDL/LDL ratio, which is associated with a reduced risk of heart disease.

However, polyunsaturated fats are vulnerable to chemical alterations within the body, which result in highly damaging 'lipid peroxides'.

Polyunsaturated fats in the body are vulnerable to attack from 'free radicals' and can undergo chemical reactions known as 'lipid peroxidation'. This results in fats that are more likely to contribute to hardening of the arteries, and also leads to the generation of more 'free radicals', which are damaging to health. A healthy supply of antioxidant vitamins can help to reduce the risk of damage from free radicals.

Processing food can result in a subtle alteration of the structure of unsaturated fatty acids, making them *trans* fatty acids. These have similar properties to saturated fatty acids and tend to raise LDL-cholesterol and lower HDL-cholesterol.

Cholesterol is another important dietary fat. It is found mainly in meat, egg yolks, dairy products, offal and shellfish. Cholesterol is a member of the *sterol* group of fats; it is not a triglyceride or a fatty acid.

Cholesterol is made in the body, mostly in the liver, and has many important functions in the body.

Levels of cholesterol in the blood are determined by a number of factors including genetics, saturated fat intake, body weight and activity level.

The effect of dietary cholesterol is thought to be minimal in many people. However, if you have raised blood cholesterol levels, reducing the amount of high-cholesterol foods that you eat may help.

Raised blood cholesterol is a significant risk factor for the development of heart disease and circulation problems. In particular, it is the LDL fraction of cholesterol that is harmful.

All fats are carried in the blood on *lipoproteins*.

These are classified according to how tightly packed their contents are. The proportions of triglyceride vs cholesterol and (other materials) in the lipoprotein particle are important.

**LDL-cholesterol HDL-cholesterol
Low density lipoprotein (LDL) High density lipoprotein (HDL)
50 % cholesterol 20 % cholesterol
LDL takes cholesterol from liver to tissues
HDL mops up spare cholesterol and takes it back to the liver
Low LDL reduces health risk
High HDL reduces health risk**

Nutritional and Natural medicines

Vitamin E
(mixed tocopherols)
Phosphatidylcholine

B3 (NADPH)
Vit C
Cu+
O2 (Adenosylcobalamine, Fe)
B5 (CoA),
Taurine
Glycine
Omega oils
Iodine
Selenium

Guggul

Cayenne pepper

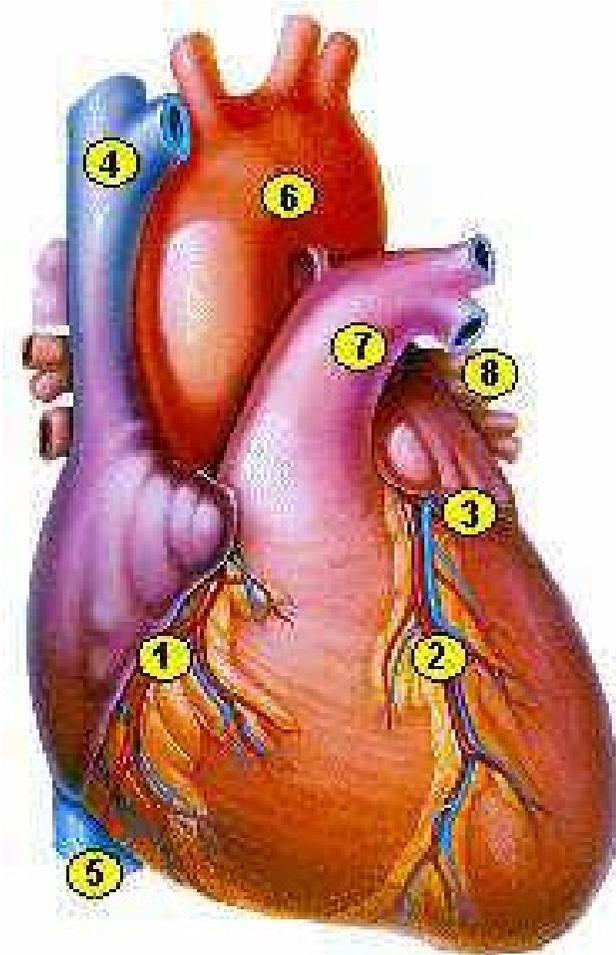
Garlic

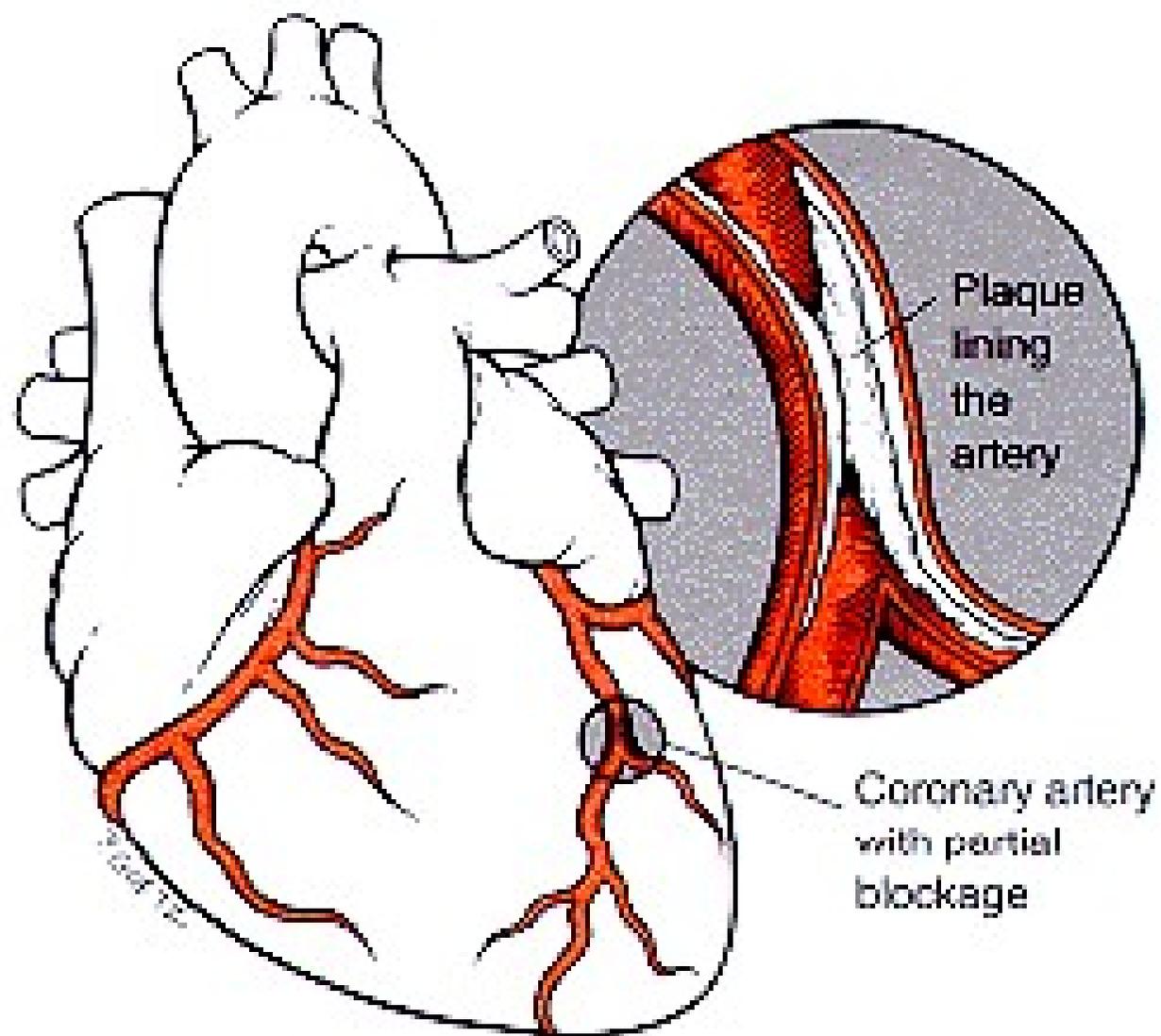
**Challenge
with Oxidised
Cholesterol**

ANGINA PECTORIS

Coronary Arteries and Great Vessels

- 1. Right Coronary**
- 2. Left Anterior Descending**
- 3. Left Circumflex**
- 4. Superior Vena Cava**
- 5. Inferior Vena Cava**
- 6. Aorta**
- 7. Pulmonary Artery**
- 8. Pulmonary Vein**





When a clogged artery keeps the heart from getting enough blood and oxygen, angina can occur.

Symptoms

Angina describes the pain, and sometimes breathlessness or choking feeling, caused by restricted blood flow in the arteries that supply the heart. The word angina comes from the Latin *angerer*, which means to strangle.

A first attack of **angina pain typically starts during exercise such as walking uphill. It may be feel like a heavy weight or a tightening across the upper chest. Angina pain is especially likely to occur when walking after a meal, or in cold, windy weather. Anger or stress tend to makes it worse.**

The pain can move **to the neck, throat or arms** — making you feel that you are choking or that both arms are dead weights. The pain doesn't usually last for more than a few minutes and goes fairly quickly after resting. As well as the pain, there may be breathlessness, sweatiness and a sense of fear.

Angina affects about one in 50 people and can have different causes. Traditionally it is controlled with a combination of medication and lifestyle changes.



Causes

Most angina is due to disease of the coronary arteries (atherosclerosis) that results when the arteries become furred up with fatty deposits. The diagram above shows the blood supply of the heart, including the coronary arteries.

The **narrowing** of these arteries means the heart muscle cannot receive enough blood (and therefore oxygen and nutrients), especially when extra demands are made on it through exertion.

Other causes of angina. These include:

- **Narrowing of the aortic heart valve**
- **Anemia,**
- **Fast, abnormal heart rhythms,**
- **Diseases of the heart muscle.**

Types of angina

There are three main types of angina:

Stable angina

Stable angina is associated with coronary heart disease, and is brought on by exertion. In this case, the angina pain usually lasts for only a few minutes. After resting, the pain subsides, but it will usually return when the effort begins again.

Unstable angina

With this type of angina, the pain comes on after only a little effort (such as just taking a few steps) or even when the person is resting. It is usually the result of a very severe narrowing (stenosis) in a coronary artery.

Variant angina

This type of angina occurs without warning, usually in women. It is due to spasm of a coronary artery. A doctor may need to make detailed investigations to diagnose this type of angina. During an attack, there can be irregularities in the heart's normal rhythm.

Myocardial Infarction

If a coronary artery becomes completely blocked, the section of heart muscle supplied by that artery will die, unless the blockage is relieved quickly.

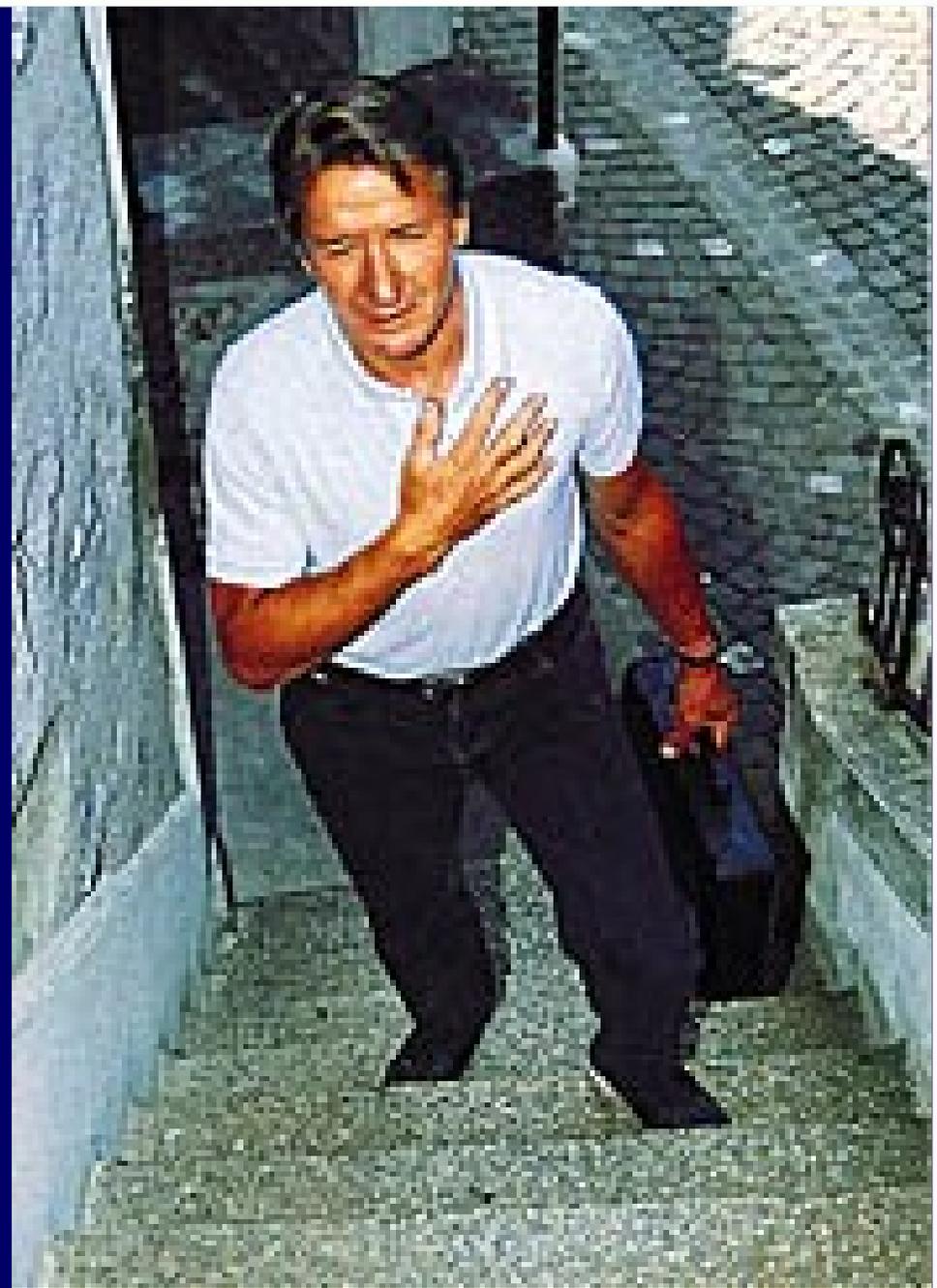
This is a myocardial infarction, or MI, and the pain is more severe and prolonged than angina.

Someone having a heart attack will also feel sick, breathless and sweaty, and may vomit.

Angina occurs more often in older people. When it occurs in younger people (under age 50), it's more common in men than women. You are more prone to angina if you:

- **Smoke,**
- **Have a high cholesterol level,**
- **Have high blood pressure,**
- **Have diabetes,**
- **Do little physical activity.**

In some cases, angina runs **in families**, so if close relatives have had angina, you may be at a greater risk of getting it too.



Nutritional and Natural Medicines

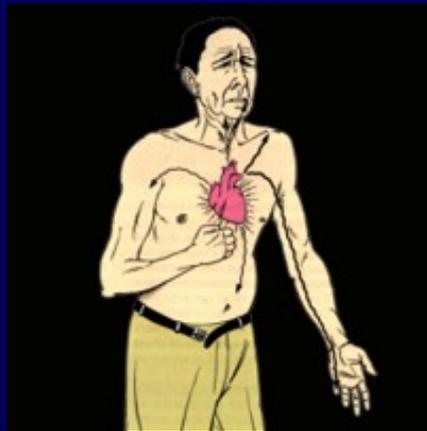
Mixed Tocopherols

EPO, BSO, Borage

Omega 3

Omega 3,6,9

Phosphatidylcholine



Cayenne pepper



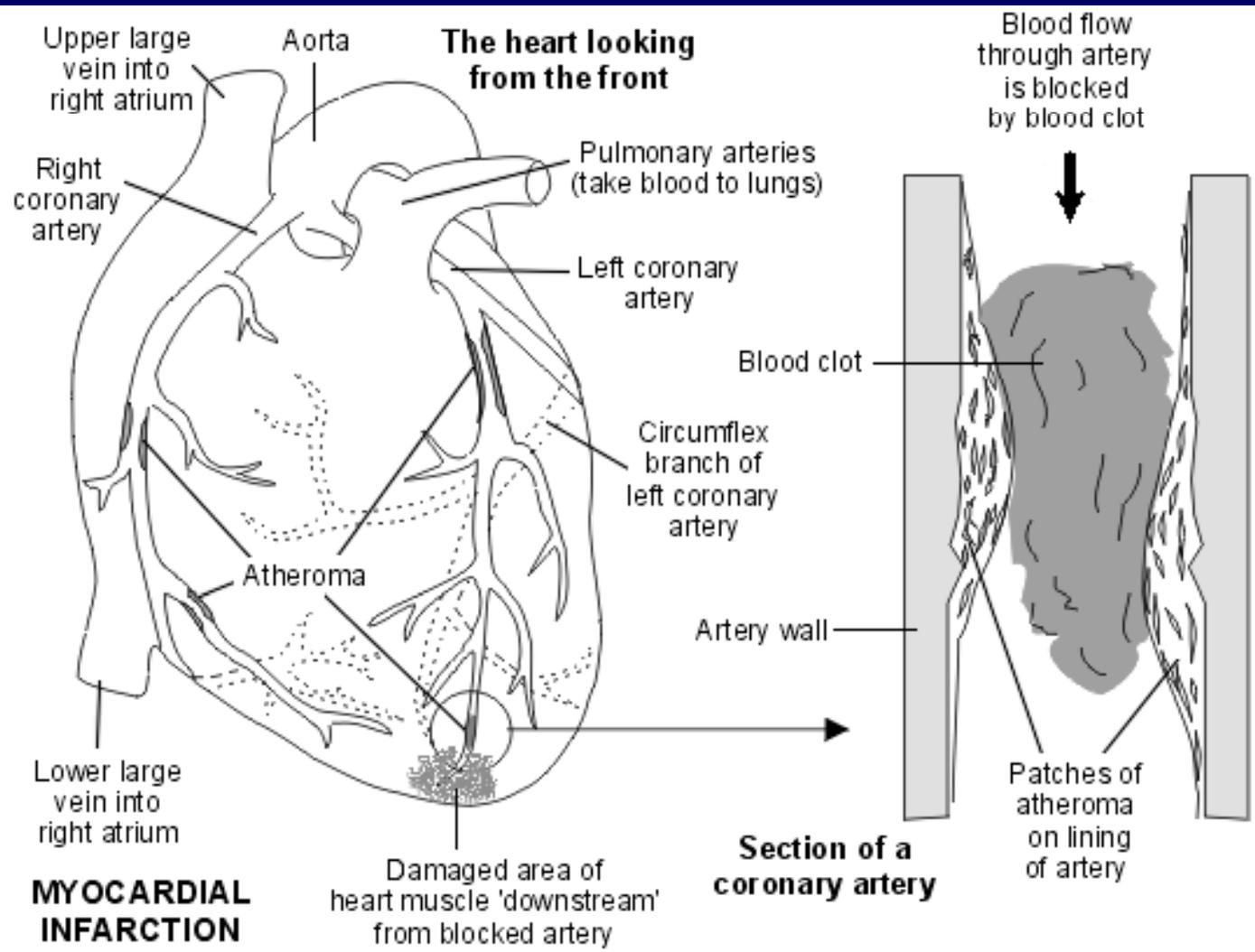
MYOCARDIAL INFARCT

Understanding the heart and coronary arteries

The heart is mainly made of special muscle. The heart muscle pumps blood into arteries (blood vessels) which take the blood to every part of the body.

The coronary arteries take blood to the heart muscle. The main coronary arteries branch off from the aorta. (The aorta is the large artery which takes oxygen-rich blood from the heart chambers to the body.) The main coronary arteries divide into smaller branches which take blood to all parts of the heart.

What happens when you have a myocardial infarction?



Elevated C-Reactive Protein is associated with an increased 10-year risk of Coronary Heart Disease - C-reactive protein (CRP) is a marker for inflammation that has been reported to be a risk factor for myocardial infarction in many studies. High CRP is associated with increased coronary heart disease.

In a study conducted by Mary Cushman M.D., MSc et al from the Departments of Medicine and Pathology at the University of Vermont, baseline CRP and 10-year incidence of first MI or CHD death were compared.

This observational cohort study, published in the July 5, 2005 issue of *Circulation*, determined that in older men and women, elevated CRP measurement was associated with an increased 10-year risk of CHD.

General description

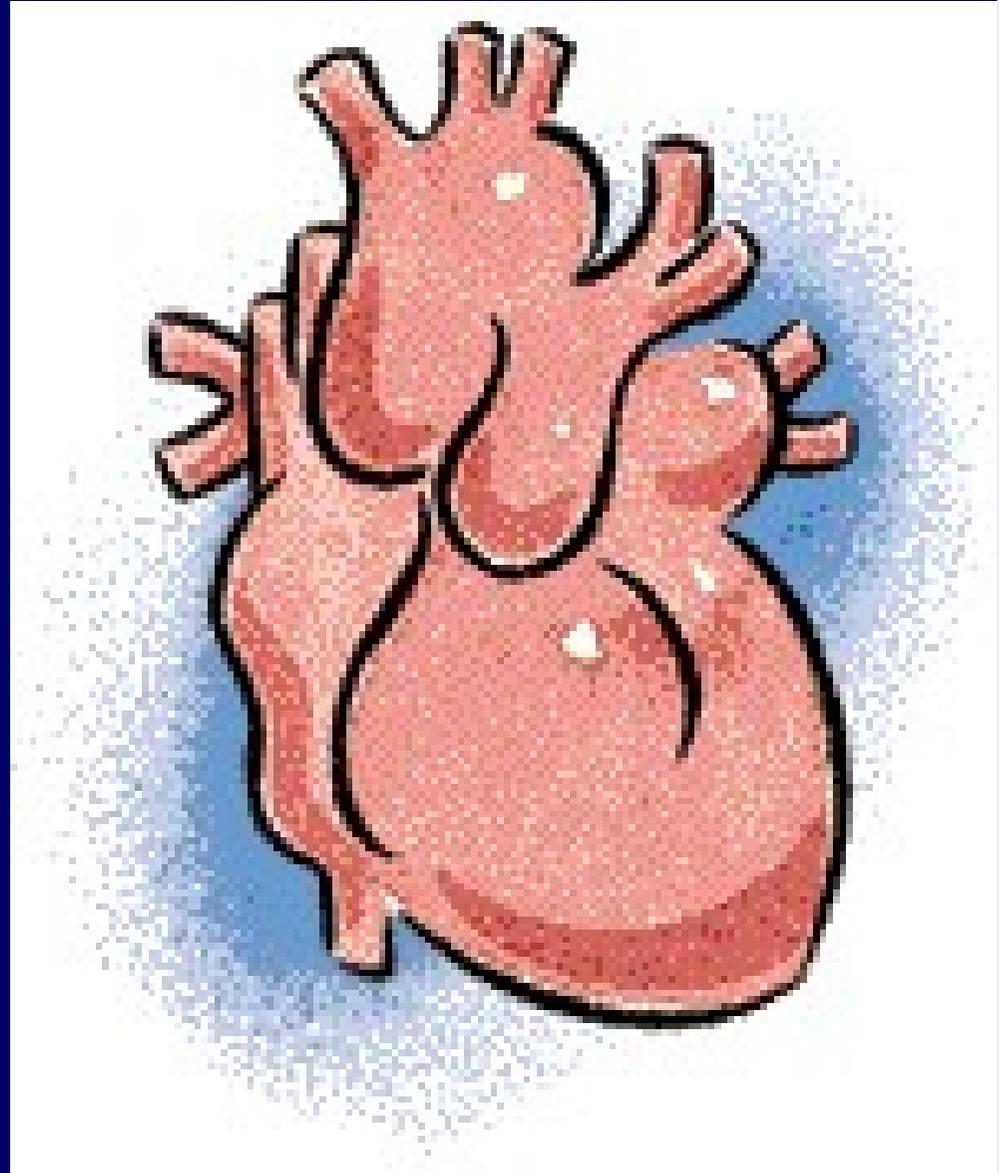
Acute myocardial infarction (MI) results from prolonged ischemia of myocardial tissue due to reduced coronary artery perfusion.

In the US there are approximately 1.5 million cases of Acute Myocardial Infarction occur each year.

About 180,000 people in the UK are admitted to hospital each year with an MI.

Symptoms

**Chest pain,
radiating to the
arm.**

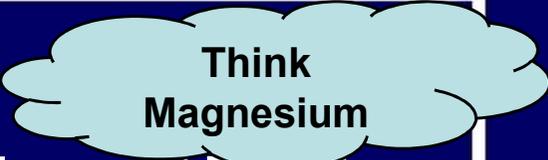


Pathophysiology

- 1. Decreased oxygen flow to myocardium results in anaerobic metabolism.**
- 2. ATP synthesis decreases within 1-2 min, and it is reduced to 50% by 10 minutes.**

3. Reduction in ATP leads to membrane channel (Na/K ATPase) disruption and subsequent increase in cell membrane permeability.

4. With increased permeability, myocytes swell.



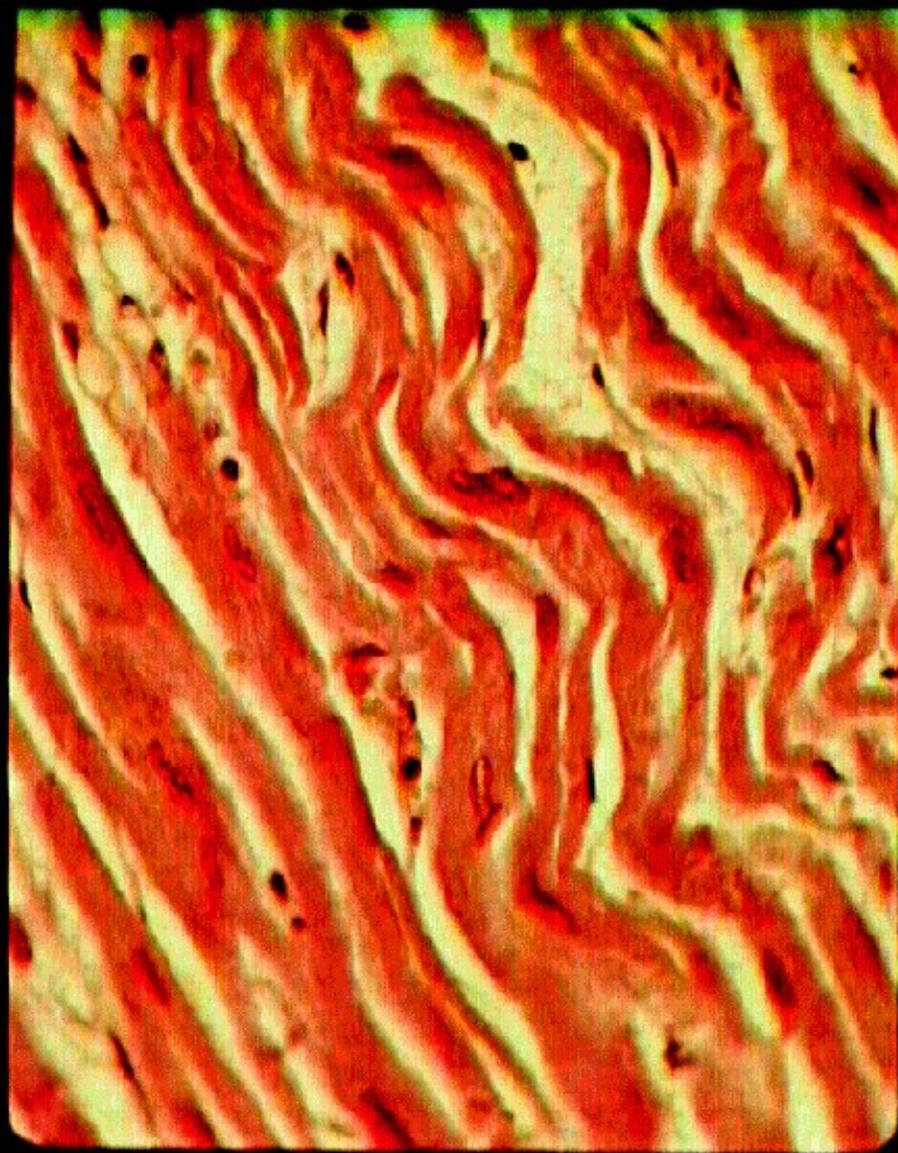
Think
Magnesium

5. There is calcium ion influx that activates various degradative enzymes (e.g., lipase, protease, nuclease, etc), which further disrupts cellular function.

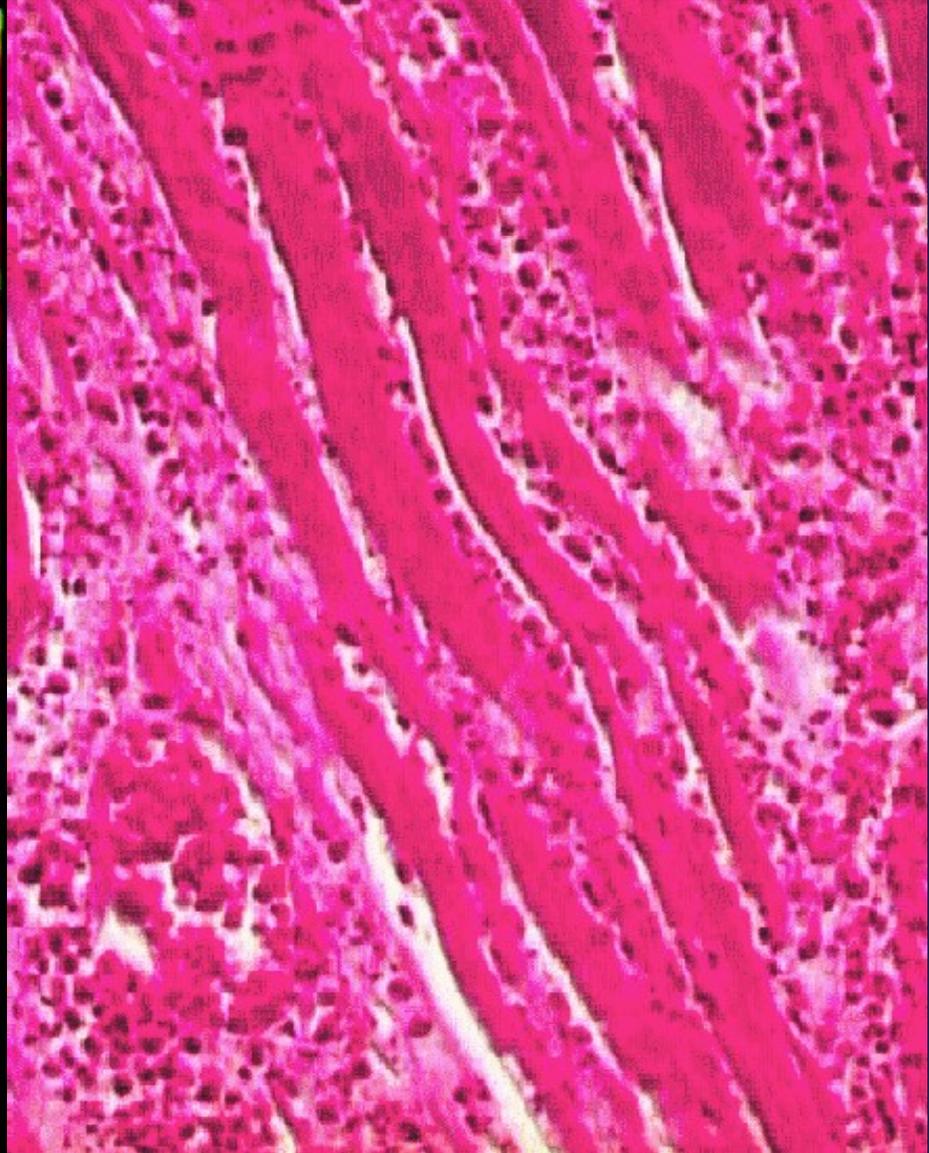
6. Irreversible cell death occurs in about 15-20 minutes.

Acute Myocardial infarction is the irreversible necrosis of heart muscle secondary to prolonged ischemia. Acute Myocardial Infarction usually results from an imbalance of oxygen supply and demand. The appearance of cardiac enzymes in the circulation generally indicates Myocardial Necrosis.

**Myocardial infarct, early stage
1-12 hours**



**Myocardial infarct, 12-
72 hours neutrophilic infiltrate**



A myocardial infarct (M.I.) can be followed chronologically by observing the morphological changes that occur in the myocardium. One hour after the onset of ischemia, stretching and waviness of the myocytes may be seen at the border of the infarct.

Often, large vacuoles are found within the injured myocytes.

Coagulative necrosis is not visible at this time. After twelve hours, but within seventy-two hours, a typical pattern of coagulative necrosis is seen and there is a neutrophilic infiltrate.

During days three to seven, **macrophages** remove the necrotic tissue. From day seven to day ten, collagen replaces the necrotic tissue and a dense scar begins to form. At day ten, most of the necrotic myocardium has been removed, but the fibrous scar tissue has not yet been substantially formed.



It is at this time that **rupture** of the heart is most common.

What causes myocardial infarction?

The usual reason is that a blood **clot (thrombosis)** forms inside a coronary artery, or one of its branches. This blocks the blood flow to a section of the heart. Blood clots do not usually form in normal arteries.

However, a clot may form if the artery has some **atheroma** on its inside lining.

Atheroma is like fatty patches or 'plaques' which develop on the inside lining of arteries. (This is similar to water pipes which get 'furred up'.)

Plaques of **atheroma** may gradually form over a number of years in one or more places in the coronary arteries. (However, atheroma may develop in any section of the coronary arteries.)

A 'crack' in a patch of **atheroma can trigger the clotting mechanism in the blood to form a blood clot. Therefore, a build up of atheroma is the root problem that leads to most cases of MI.**

Prothrombin

(liver protein)

Intrinsic system

prothrombinase

Extrinsic system

coagulation factors

anti coagulation

factors

Heparin

Warfarin



Thrombin

(enzyme)



Fibrinogen



Fibrin

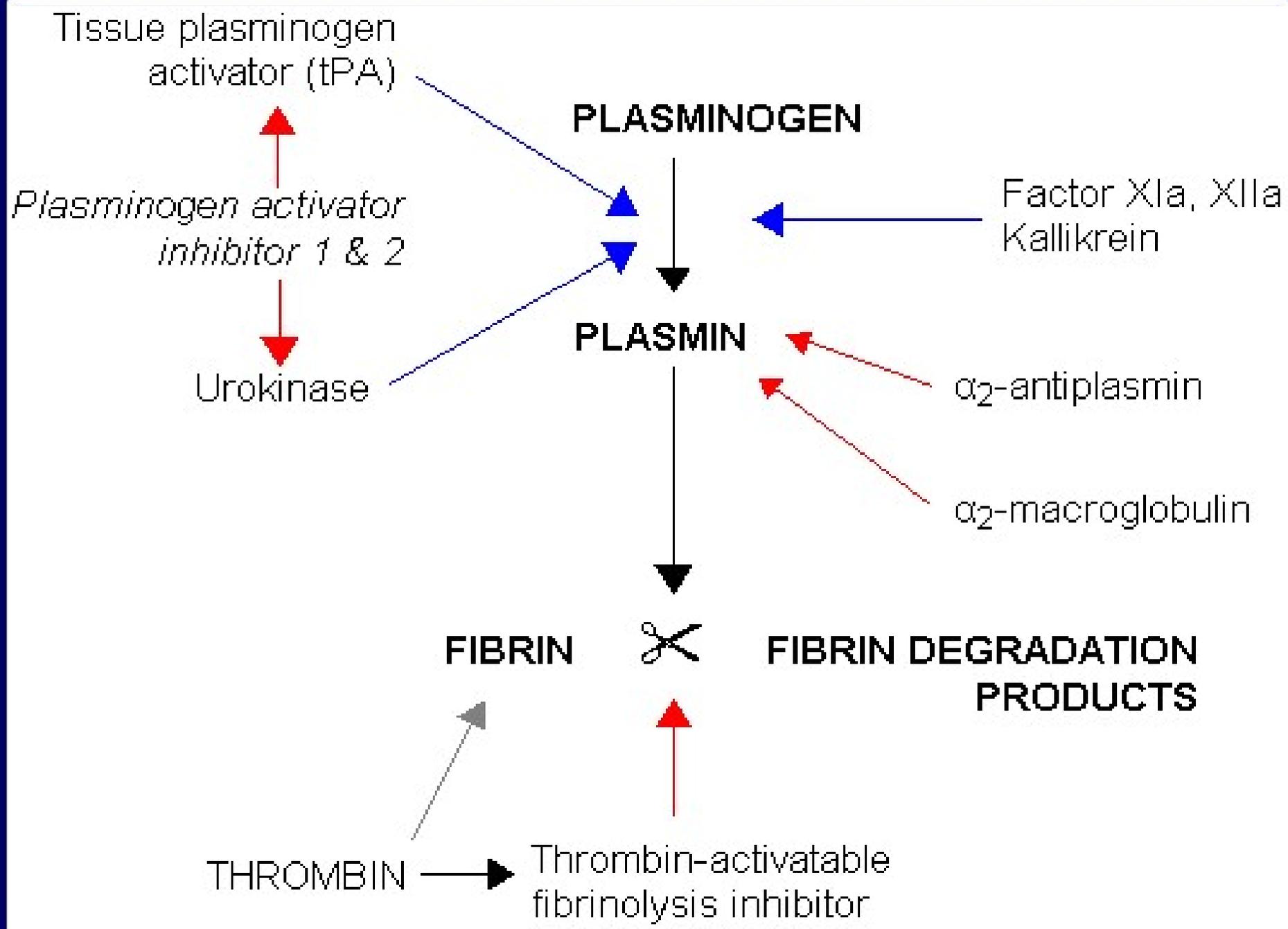
(soluble liver protein)

+ ↑ **homocysteine**

(insoluble protein)

The membrane **phospholipids** of circulating blood platelets and red cells are asymmetrically arranged with virtually all phosphatidylserine on the inside.

Fibrinolysis is the process where a fibrin clot, the product of coagulation, is broken down. Its main enzyme, *plasmin*, cuts the fibrin mesh at various places, leading to the production of circulating fragments that are cleared by other proteinases or by the kidney and liver.



Stop smoking.

Change your eating habits.

Maintain a healthy weight.

Get physical activity.

Reduce stress and relax.

If you have high blood pressure, diabetes etc., remember to take your medicine.

Have your blood pressure checked regularly.

Have regular dental and medical check ups.

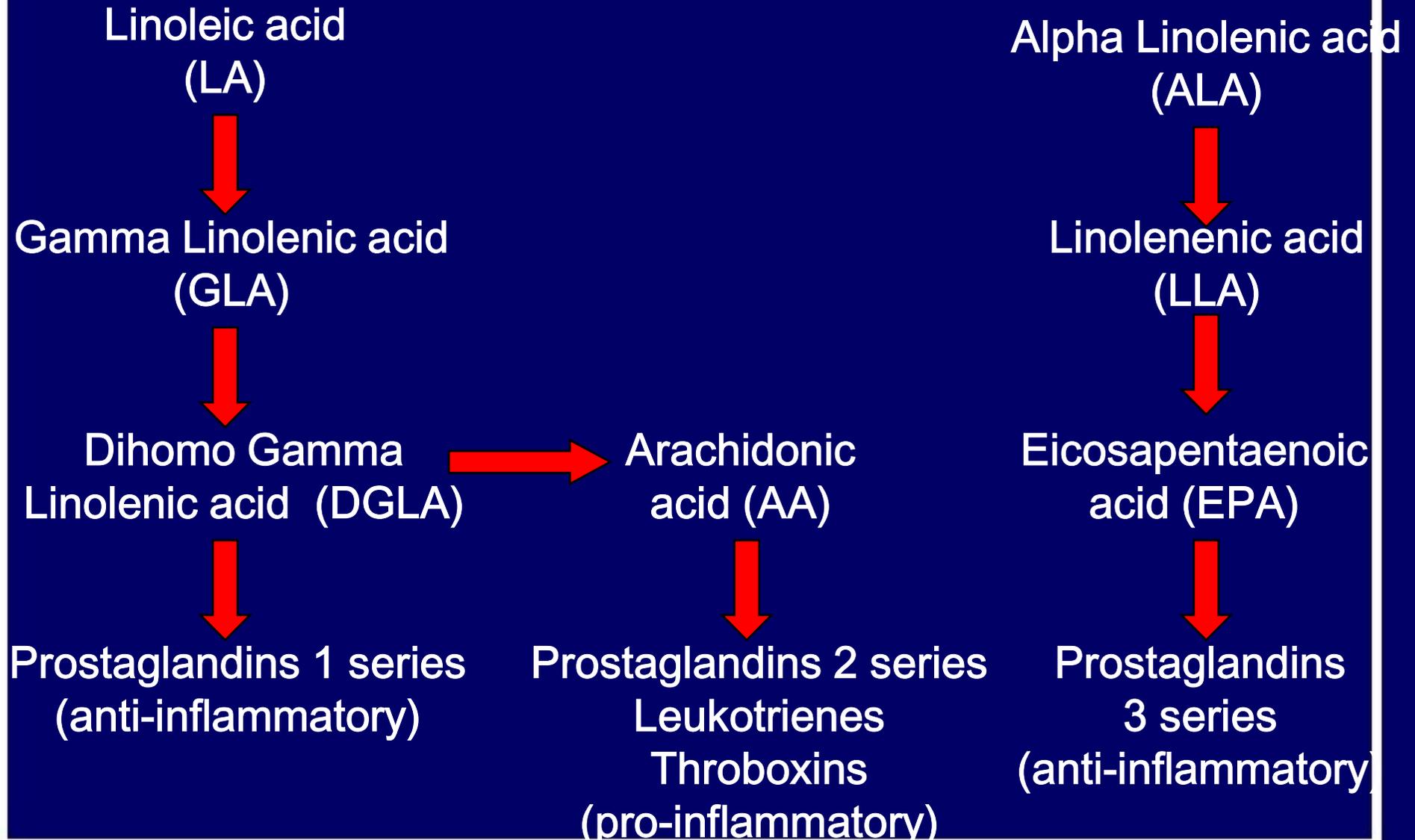
Aspirin - to reduce the 'stickiness' of platelets in the blood which helps to prevent blood clots forming. If you are not be able to take aspirin then an alternative 'anti-platelet' medicine such as clopidogrel may be advised.

A beta-blocker - to slow the heart rate, and to reduce the chance of abnormal heart rhythms developing.

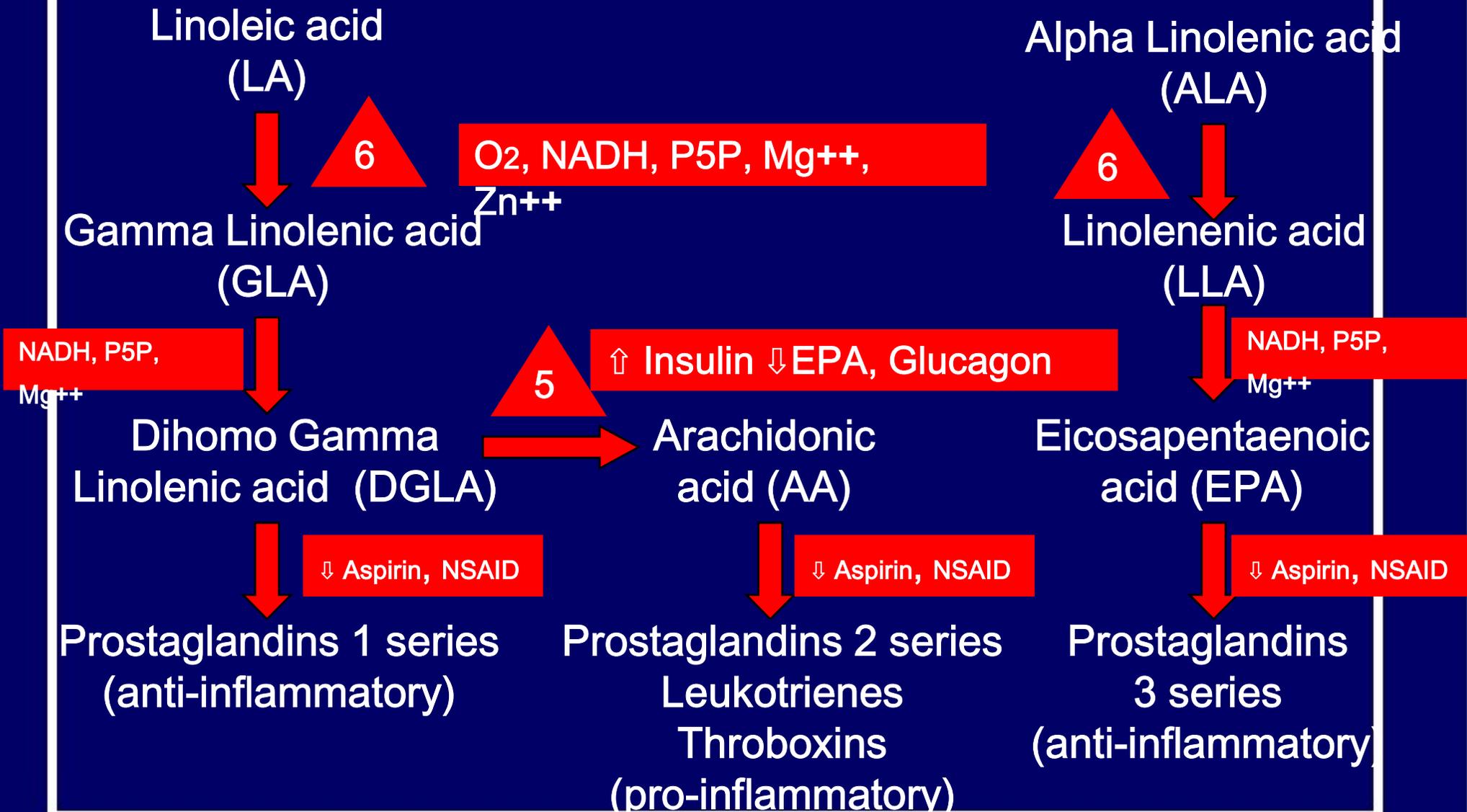
An ACE (angiotensin converting inhibitor) - especially if you have any heart failure.

A cholesterol lowering medicine to help prevent the build-up of atheroma. This is especially important if your cholesterol level is high.

Eicosanoids



Eicosanoids



Membrane phospholipid

phospholipase A2

↑ angiotensin 1

Bradykinin, Adrenalin

Thrombin

Arachidonic acid

lipoxygenase

cyclo-oxygenase

Leukotriens

Lipoxins

Prostaglandins

Thomboxanes

Prostacyclin

Eicosanoids

Arachidonic
acid (AA)

lipoxygenase

↓ Vit E, Vit C, GLA, EPA, Zn, Sel.

↑ aspirin, NSAID

Leukotrienes
(pro-inflammatory)

cyclooxygenase

↑ dairy, estrogen

↓ aspirin, NSAID, EPA

Prostaglandins 2 series
Thromboxins
(pro-inflammatory)

prostacyclin synthase (P450)

↓ OH radical

↑ Ginger, Garlic, Onion, Mg

Prostacyclin
(anti-inflammatory)

DGLA

Group 1

Prostanoids

PGE1

PGF1

TXA1

Leukotrienes

LTA3

LTC3

LTD3

Arachidonic

Group 2

Prostanoids

PGD2

PGE2

PGF2

PGI2

TXA2

Leukotrienes

LTA4

LTB4

LTC4

LTD4

LTE4

Lipoxins

LXA4

LXB4

LXC4

LXD4

LXE4

EPA

Group 3

Prostanoids

PGD3

PGE3

PGF3

PGI3

TXA3

Leukotrienes

LTA5

LTB5

LTC5

By **Julie Wheldon**
Science Correspondent

Why olive oil works as well as an aspirin

IT has long been regarded as an essential part of the Mediterranean diet for healthy living.

Now scientists believe they have discovered exactly what it is that makes extra virgin olive oil so good for us.

A study suggests the oil can prevent inflammation in the same way as common headache pills. In doing so, it helps stave off long-term health problems such as cancer and heart disease.

The researchers, based at the University of Pennsylvania, found the main compound in the oil, oleocanthal, contained the same properties as the painkiller ibuprofen.

Ibuprofen has been linked to a lower risk of cancer and heart problems, as has aspirin, which belongs to the same class of anti-inflammatory drugs, called COX inhibitors.

The study concluded that extra virgin olive oil - made from the first pressing of the olive - may offer similar long-term advantages.

The extra virgin oil costs around twice as much as the standard version. While the ordinary oil offers some health benefits, these are less pronounced.

Dr Paul Breslin, who led the research, said extra virgin could not actually be used to cure headaches

'Various health benefits'

because a daily dose of 50g would only be equivalent to 10 per cent of a normal dose of ibuprofen.

He added, however, that a long-term Mediterranean diet which included the oil could help build the body's natural defences to conditions such as cancer and heart problems.

'Our findings raise the possibility that long-term consumption of oleocanthal may help to protect against some disease by virtue of its ibuprofen-like activity,' he explained.

'It is known that regular low doses of aspirin for instance, another COX inhibitor, confer cardiovascular

Nature, adds to the growing evidence of the health benefits of a Mediterranean diet typically rich in fish, unsaturated fats and vegetables.

The diet also includes an occasional glass of wine.

In April, researchers at Athens University concluded that a healthy man of 60 who followed a Mediterranean diet could expect to live a year longer than one of the same age who ate differently.

The team looked at more than 74,000 people in nine European countries.

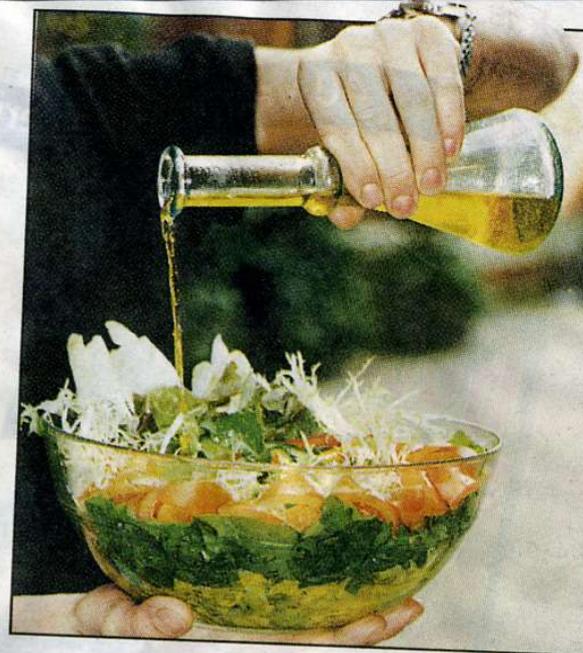
They found that the Greeks adhered most closely to the recommended diet, followed by the Spanish, Italians and French.

The British were fifth - ahead of the Danes, the Germans, the Swedes and the Dutch.

Last year, a study found that eating a Mediterranean diet can be especially beneficial for the elderly.

For the study, Dutch researchers looked at the eating habits of healthy men and women aged 70 to 90 in 11 European countries.

They discovered that, along with exercise, moderate drinking and not smoking, a Mediterranean diet



Well-dressed: Olive oil is a mainstay of a healthy diet

1p seats are one way, include taxes and charges and apply to Internet bookings only. Advertised fares are subject to date and time of travel and availability. This offer is limited and is valid from 8am until 8pm on Thursday 1st September 2005. A total of 500 one way seats at 1p will be available during this time. Credit card handling fee of £4 per transaction applies.

Fly away to Europe for

Nutritional and Natural Medicines

Carnitine

Taurine

Magnesium

CoQ10

Omega 6

Omega 3

Mixed tocopherols

Cayenne

Garlic

Ginger

Hawthorne

Onions

Cloves

Turmeric

CARDIAC MYOPATHY

Cardiomyopathy is a type of heart disease in which the heart becomes abnormally enlarged (enlarged heart), thickened and/or stiffened. As a result, the heart muscle's ability to pump blood is usually weakened. This condition is generally progressive and may lead to heart failure.

Cardiomyopathies may be caused by a wide range of conditions, including chronic diseases, alcoholism, viral diseases, heart attacks and many others.

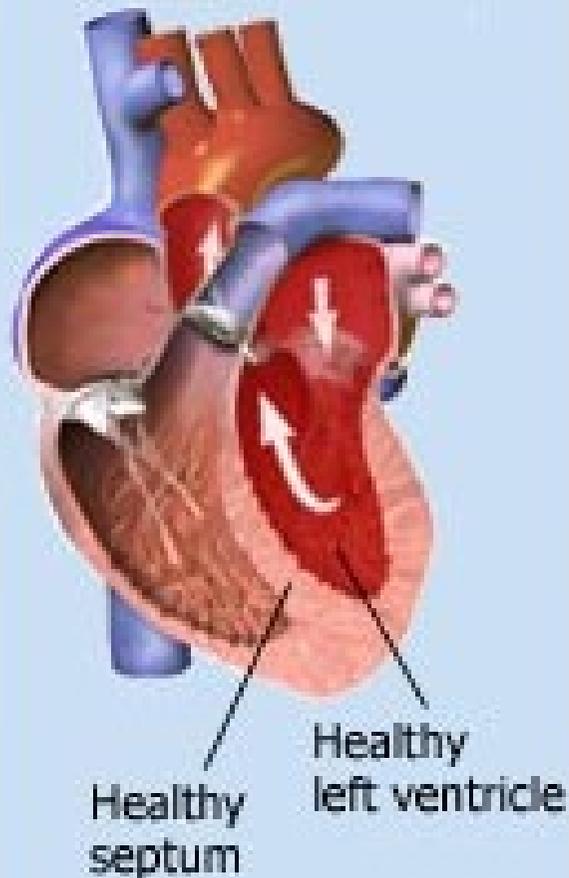
An affected heart may grow larger either by dilatation, thickening hypertrophy or both.

Additionally, the heart may suffer from a reduced ability to relax.

Abnormalities found in **cardiomyopathy include:**

- 1. Thickened and/or dilated ventricles, especially the left ventricle. The upper chamber (atria) may also be involved and enlarged.**
- 2. Scar tissue, possibly left over after a heart attack.**
- 3. Overall enlargement of the heart.**

Normal



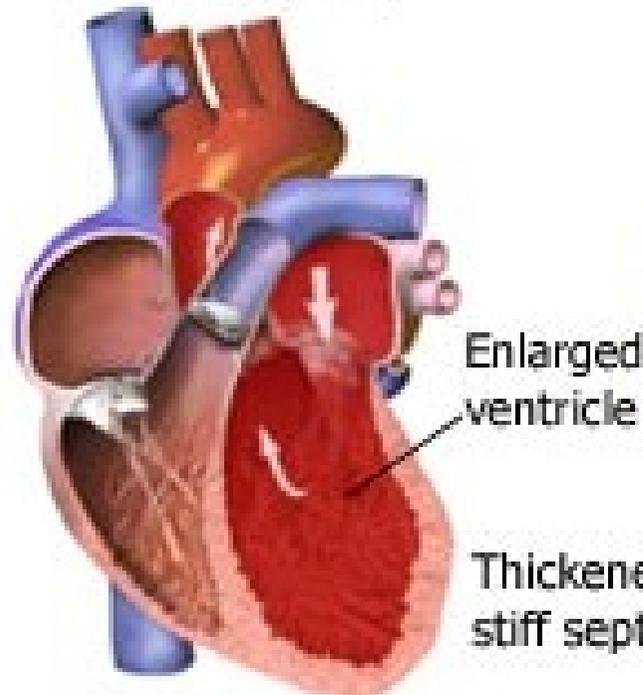
A healthy left ventricle pumps enough oxygenated blood to meet the body's needs.

© 2004 - Duplication not permitted

Cardiomyopathy

A condition in which a ventricle has become enlarged, thickened and/or stiffened. As a result, the heart's ability to pump is reduced. Two types of cardiomyopathy include:

Dilated cardiomyopathy



An enlarged, weakened left ventricle struggles to pump enough blood to meet the body's needs.

Hypertrophic cardiomyopathy



Left ventricle cannot fully relax between heartbeats, resulting in less blood flow.

Symptoms including shortness of breath, chest pain, fainting, dizziness and a reduced ability to exercise. The muscle damage that develops with all types of non-ischemic cardiomyopathies can lead to congestive heart failure or abnormal heartbeats known as arrhythmias.

Particularly severe arrhythmias may lead to fainting (syncope) or even sudden cardiac death.

They may also cause arrhythmias (potentially dangerous abnormal heart rhythms).

More than 27,000 deaths each year are caused by cardiomyopathy in the USA.

Ischeamic cardiomyopathy is a chronic disorder caused by either recurrent heart attacks or coronary artery disease (CAD) – a disease in which there is hardening (atherosclerosis) of the arteries on the surface of the heart.

CAD often leads to episodes of cardiac ischemia, in which the heart muscle is not receiving enough oxygen-rich blood.

Additionally, as a result of one or more large heart attacks, the heart enlarges because of the scar, with resulting less functioning heart muscle to pump blood.

Recurrent ischemia and small heart attacks may also lead to fibrosis (*scarring*) and weakening of the heart, resulting in ischemic cardiomyopathy. Ischemic cardiomyopathy, with all of the heart muscle preserved and still functioning, can become so ischemic that the heart muscle

pumps less blood, particularly under emotional or physical stress, yet functions normally under rest conditions. The ischemic heart becomes **“stunned, stiff and noncontractile”** when the stress on the heart exceeds the blood supply.

Non-ischemic cardiomyopathies

are less common, progressive diseases. Unlike ischemic cardiomyopathies, which tend to develop in older adults, non ischemic cardiomyopathies frequently occur in young people.

Non-ischemic cardiomyopathies

affect about 50,000 people in the United States, and are a leading factor necessitating heart transplant surgeries. Non-ischemic cardiomyopathies can be difficult to diagnose because many are *idiopathic* (i.e., their cause is unknown).

However, known causes include genetic factors, viral infection, the build-up of fat and proteins (*amyloidosis*) in the heart muscle,

or an excess of iron
(hemochromatosis) in organs
such as the heart.

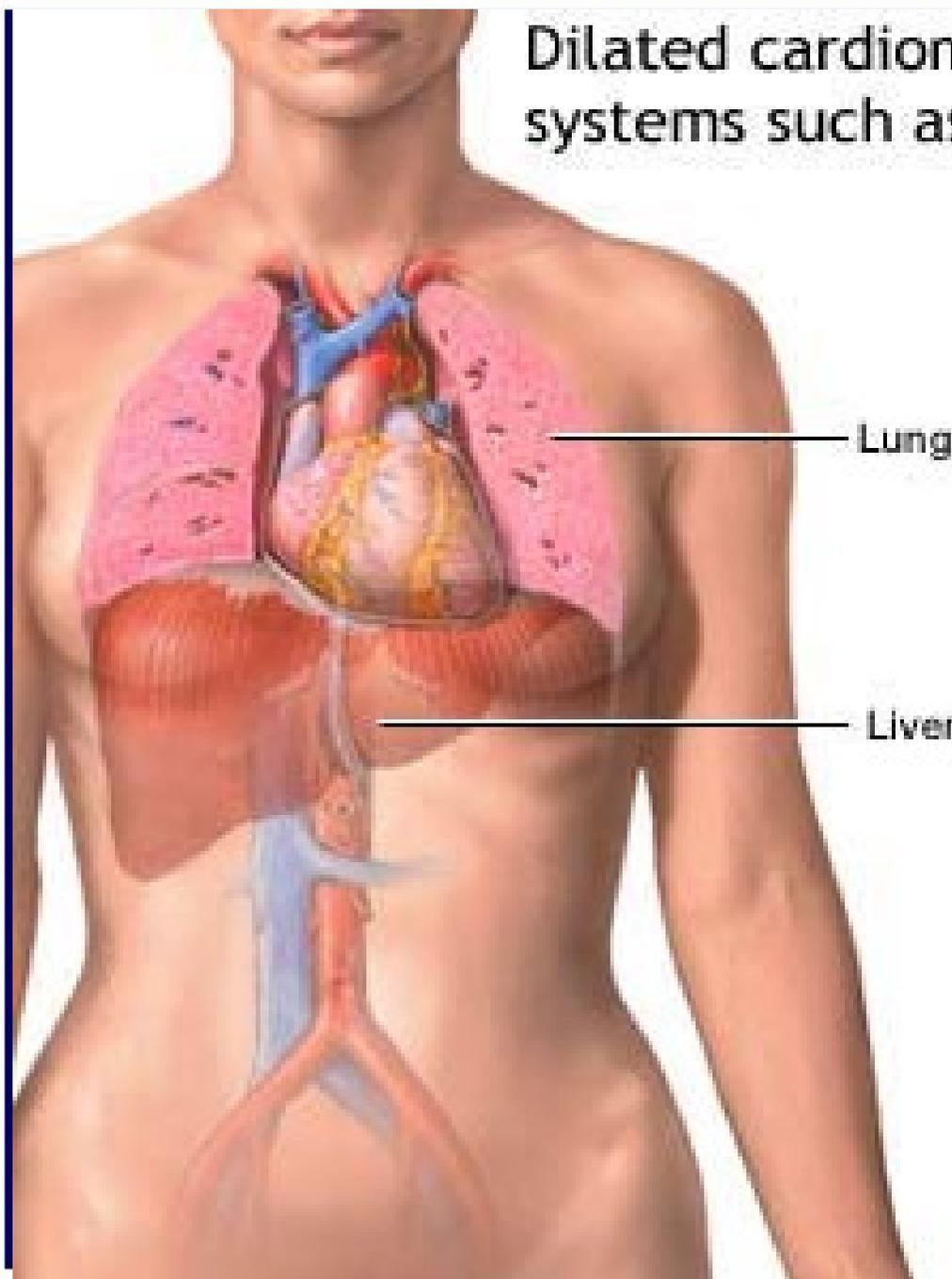
The excessive use of alcohol or
other substances can also play a
role in the development of the
disease.

There are three main types of non-ischemic cardiomyopathies:

1. Dilated cardiomyopathy

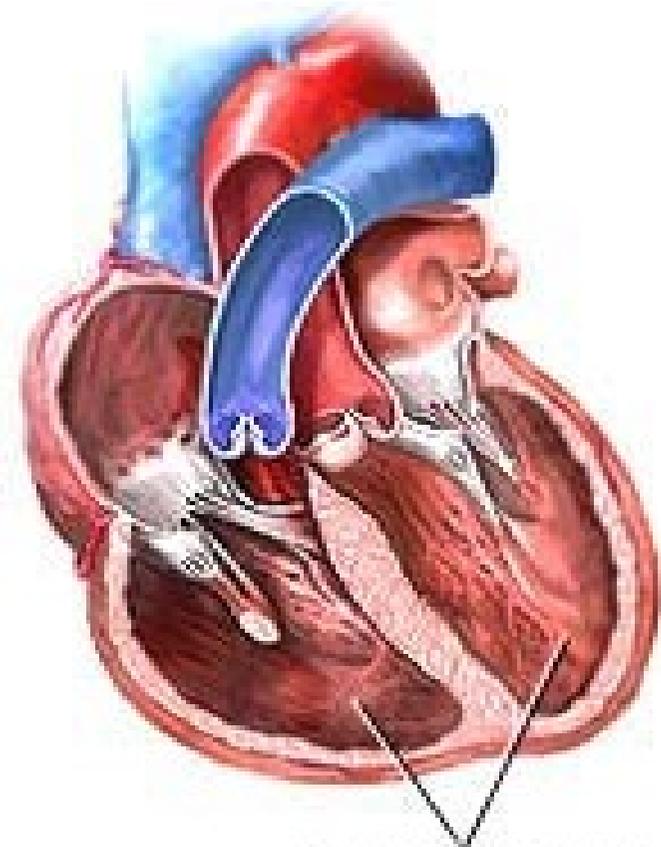
(including peripartum cardiomyopathy and alcoholic cardiomyopathy), which involves dilation or enlargement of the heart's ventricles and is usually accompanied by an increase in cardiac mass. This often affects young people.

Dilated cardiomyopathy can effect organ systems such as the lungs and liver



Lung

Liver



Enlarged left and right ventricles

2. Hypertrophic cardiomyopathy, which involves an abnormal growth of muscle fibres in the heart muscle, usually in the left ventricle. In this case, the volume of the left ventricle is normal or reduced,

but the additional muscle fibres prevent the chamber from relaxing completely after contraction (diastole), making it a ***diastolic dysfunction***. This is usually considered a genetic disorder.

3. Restrictive cardiomyopathy, which means the heart muscle cannot adequately relax after contraction, making it unable to fill completely with blood. This condition is distinguished from some forms of hypertrophic cardiomyopathy because the left ventricle is frequently normal sized.

This cardiomyopathy is more common in the tropics than other forms of cardiomyopathy

Nutritional and Natural medicines

Carnitine

Taurine

Zinc (testosterone)

CoEnzyme Q10

Mixed tocopherols

Magnesium

Black walnut

Hawthorne

Cloves

There are an ever-increasing number of studies indicating an association between **high testosterone and low cardiovascular disease** rates in men.

Testosterone is a **muscle-building hormone** with many testosterone-receptor sites in the heart.

Various studies have shown that the **weakening of the heart** muscle can sometimes be attributed to testosterone deficiency.

Testosterone is not only responsible for maintaining heart muscle protein synthesis but a promoter of coronary artery dilation and helps to maintain healthy cholesterol levels.

In the majority of patient's symptoms and ECG measurements improve when **low testosterone levels** are corrected.

One study showed that blood flow to the heart improved 68.8% in those receiving **testosterone therapy**.

In China doctors are successfully treating **angina** with testosterone therapy.

Effects of low testosterone on cardiovascular disease

1. **Cholesterol**, fibrinogen, triglycerides and insulin levels increase.
2. **Coronary artery** elasticity diminishes.

3. Blood pressure rises

HGH declines (weakening heart muscle).

4. Abdominal fat increases

(increasing risk of heart disease)

Nutrients

Zinc

Fenugreek

Black walnut

HEART FAILURE

Definition

Heart failure, also called congestive heart failure, is a disorder in which the heart loses its ability to pump blood efficiently. The term "heart failure" should not be confused with cardiac arrest, a situation in which the heart actually stops beating.

Causes, incidence, and risk factors

Heart failure is almost always a chronic, long-term condition, although it can sometimes develop suddenly. This condition may affect the right side, the left side, or both sides of the heart.

As the heart's pumping action is lost, blood may back up into other areas of the body, including:

The liver

The gastrointestinal tract and extremities (right-sided heart failure)

The lungs (left-sided heart failure)

With **heart failure**, many organs don't receive enough oxygen and nutrients, which damages them and reduces their ability to function properly. Most areas of the body can be affected when both sides of the heart fail.

The most common causes of **heart failure** are hypertension (high blood pressure) and coronary artery disease (for example, you have had a heart attack). Other structural or functional causes of heart failure include the following:

- **Valvular heart disease**
- **Congenital heart disease**
- **Dilated cardiomyopathy**
- **Lung disease**
- **Heart tumour**

Heart failure becomes more common with advancing age. You are also at increased risk for developing heart failure if you are overweight, have diabetes, smoke cigarettes, abuse alcohol, or use cocaine.

Symptoms

Weight gain

Swelling of feet and ankles

Swelling of the abdomen

Pronounced neck veins

Loss of appetite, indigestion

Nausea and vomiting

**Shortness of breath with activity, or
after lying down for a while**

Normal foot



Foot with edema



ADAM.



Edema or swelling
of the leg, ankle
and foot

ADAM.

Difficulty sleeping

Fatigue, weakness, faintness

**Sensation of feeling the heart beat
(palpitations)**

Irregular or rapid pulse

**Decreased alertness or
concentration**

Cough

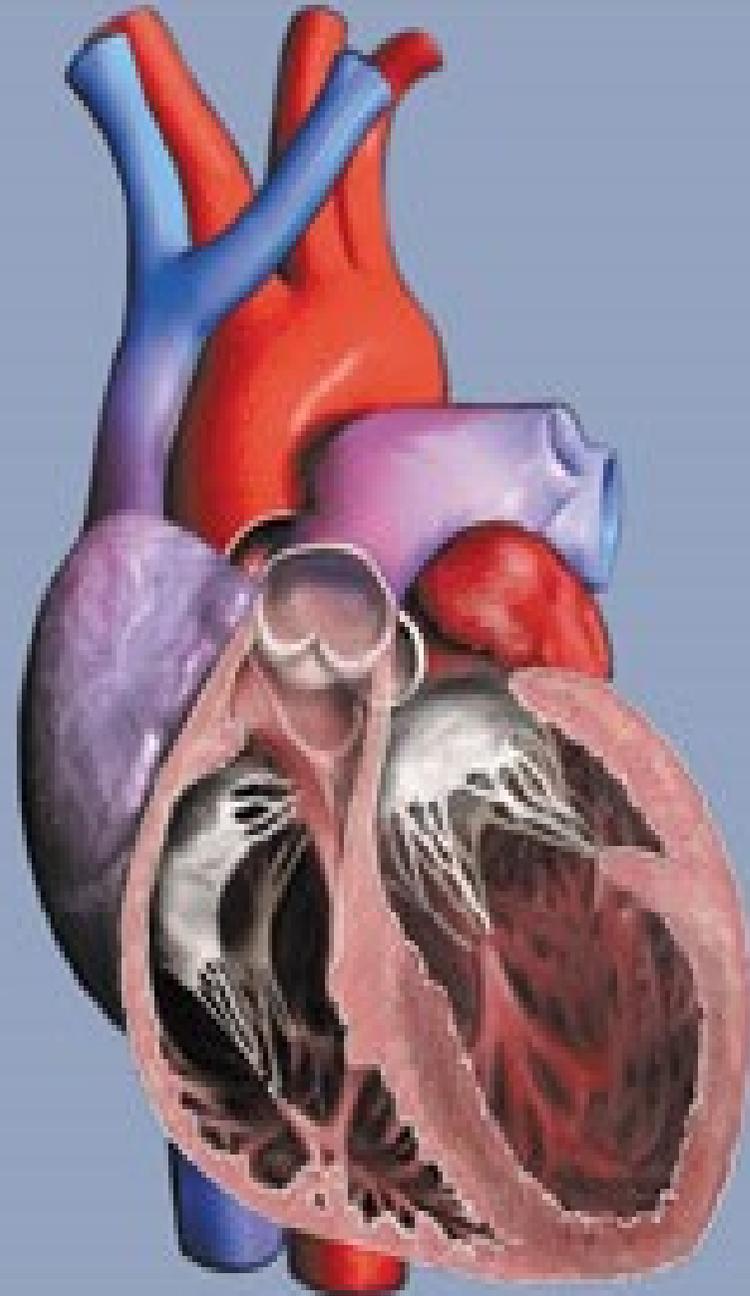
Decreased urine production

Need to urinate at night

**Infants may sweat during feeding
(or other exertion).**

Some patients with **heart failure
have no symptoms. In these
people, the symptoms may
develop only with these
conditions:**

**Infections with
high fever
Anemia
Abnormal heart
rhythm
(arrhythmias)
Hyperthyroidism
Kidney disease**



Signs and tests

A physical examination may reveal either an irregular or a rapid heartbeat. There may be distended neck veins, enlarged liver, swelling of the limbs (peripheral oedema), and signs of fluid around the lungs (pleural effusion).

Listening to the chest with a stethoscope may reveal lung crackles or abnormal heart be normal, elevated or low.

An enlargement of the heart may be seen on several tests, including the following:

Echocardiogram

Heart catheterization

Chest x-ray

Chest CT scan

Cardiac MRI

Nuclear heart scans

ECG, which may also show arrhythmias

Stay active. For example, walk or ride a stationary bicycle. **DO NOT** exercise on days that your weight has gone up from fluid retention or you are not feeling well.
Lose weight if you are overweight.

Get enough rest, including after exercise, eating, or other activities. This allows your heart to rest as well. Keep your feet elevated to decrease swelling.

Nutritional and Natural medicines

Carnitine

Taurine

Magnesium

Zinc

Thiamine pp

CoQ10

Homocysteine

factors

Hawthorn

Black walnut

DEEP VEIN THROMBOSIS



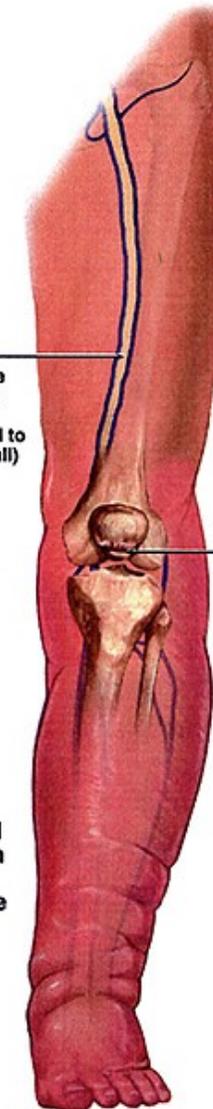
Deep Vein Thrombosis

Blood flow
to the heart
and lungs



Normal leg

Venous clot
untreatable with medicine
(too large to be effective)
and untreatable with
surgery (densely adhered to
the interior of the vein wall)



Healed
fracture of
the patella

Swelling and
inflammation
below the
blockage site

Ms. Davis' Leg

Deep vein thrombosis (DVT) is a common but elusive illness that can result in suffering and death if not recognized and treated effectively. Death can occur when the venous thrombi break off and form pulmonary emboli, which pass to and obstruct the arteries of the lung.

DVT and pulmonary embolism PE,
most often complicate the course
of sick, hospitalized patients but
may also affect ambulatory and
otherwise healthy persons.

Since **venous thrombosis** is difficult to recognize clinically, these hospitalized cases probably represent the tip of the iceberg. Unfortunately, the death rate from PE and DVT is substantial, and the probability of survival of affected individuals is decreased when compared with unaffected ones.

Epidemiology

Women are a prime target for PE, being affected more often than men. It is estimated that the number of female patients who die from pulmonary embolism complications in one year, exceeds the number of women who die from breast cancer each year in the USA.

In instances when **DVT and PE develop as complications of a surgical or medical illness, in addition to the mortality risk, hospitalization is prolonged and healthcare costs are increased.**

Etiology of PE/DVT

The main hypothesis on etiology of PE/DVT is that most patients who suffer idiopathic PE/DVT (e.g., no associated cancer) have a genetic predisposition, which remains subclinical until an additional stress occurs (e.g., immobilization during a long period).

Currently, **only 25%** of these underlying inherited hyper-coagulable states are identified. The most common of them is resistance of Factor V to inactivation by activated Protein C (it is due to a single aminoacid mutation in Factor V- known as Factor V Leiden).

Hypercoagulable States Associated

With Venous Thrombosis

Mutation in Factor V gene

Mutation in Protein C gene

Protein S deficiency

Antithrombin III deficiency

Antiphospholipid antibodies

**Elevated concentration of Factor
VIII**

Pathogenesis of venous thromboembolism

Venous thrombi consist mostly of red cell, a few platelets, and leucocyte component held together with fibrin. The disorder usually starts as a pure thrombotic process, and inflammation occurs secondary to the presence of the thrombus.

The thrombi often break off and migrate to the lungs causing PE. The formation, development, and dissolution of thrombi depend on the balance between the effects of thrombogenic stimuli and a variety of protective mechanisms.

Three mechanisms are involved in the pathogenesis of venous thrombosis, they are:

1. venous stasis,
2. injury to the venous wall,
3. hyper-coagulable states.

Venous stasis predisposes the patients to venous thrombosis mainly by impairing the clearance of activated coagulation factors from the local where thrombus is formed, and vascular damage contributes to the genesis of venous thrombosis through either direct trauma , or activation of endothelial cells by cytokines.

Risk factors

There are some clinical conditions that predispose patients to DVT. In those circumstances prophylaxis should be done in the way to reduce complications, specially PE. These conditions are directly or indirectly involved with those pathogenic mechanisms described.

Risk Factors for Venous Thromboembolism (VT)

Age > 60 y

Extensive surgery

Previous VT

Marked immobility, pre or postoperative

Major orthopedic surgery

Fracture of pelvis, femur or tibia

Malignancies

Hypercoagulable states

Obesity

Pregnancy

Estrogen use

Venulitis

Postoperative sepsis

Heart failure

Inflammatory bowel disease

Sepsis Myocardial infarction

Signs and symptoms

DVT is often first noticed as an insidious, progressive, annoying “pulling sensation” at the insertion of the lower calf muscle into the posterior portion of the lower leg. This feeling can then become more pronounced and accompanied by warmth, swelling, and erythema.

Tenderness may be present along the course of the involved veins, and a cord may be palpable. Other signs include: increased tissue turgor, distension of superficial veins, proeminent venous collaterals. “Homans’ sign” (increase resistance or pain during dorsi-flexion of the foot) is unreliable and non-specific.

Nutritional and Natural medicines

Magnesium

Mixed tocopherols

Omega 3 (EPA/DHA)

DHA

Phosphatidylcholine

Garlic

Ginger

Onions

VARICOSE VEINS



Varicose veins are enlarged veins that can be flesh coloured, dark purple or blue. They often look like cords and appear twisted and bulging. They are swollen and raised above the surface of the skin. Varicose veins are commonly found on the backs of the calves or on the inside of the leg.

During pregnancy, varicose veins called hemorrhoids can form in the vagina or around the anus. Spider veins are similar to varicose veins, but they are smaller. They are often red or blue and are closer to the surface of the skin than varicose veins.

They can look like tree branches or **spider webs** with their short jagged lines. Spider veins can be found on the legs and face. They can cover either a very small or very large area of skin.

What causes varicose veins and spider veins?

The heart pumps blood filled with oxygen and nutrients to the whole body. Arteries carry blood from the heart towards the body parts. Veins carry oxygen-poor blood from the body back to the heart.

The squeezing of **leg muscles** pumps blood back to the heart from the lower body. Veins have valves that act as one-way flaps. These valves prevent the blood from flowing backwards as it moves up the legs. If the one-way valves become weak, blood can leak back into the vein and collect there.

How common are abnormal leg veins?

About 50 to 55% of American and UK women and 40 to 45% of American and UK men suffer from some form of vein problem. Varicose veins affect 1 out of 2 people age 50 and older.

Who usually has varicose veins and spider veins?

Many factors increase a person's chances of developing varicose or spider veins.

These include:

Increasing Age

Having family members with vein problems or being born with weak vein valves

Hormonal changes. These occur during puberty, pregnancy, and menopause.

Taking birth control pills and other medicines containing estrogen and progesterone also increase the risk of varicose or spider veins

Pregnancy. During pregnancy there is a huge increase in the amount of blood in the body. This can cause veins to enlarge. The expanding uterus also puts pressure on the veins. Varicose veins usually improve within 3 months after delivery. A growing number of abnormal veins usually appear with each additional pregnancy

Obesity, leg injury, prolonged standing and other things that weaken vein valves

Sun exposure, which can cause spider veins on the cheeks or nose of a fair-skinned person

Why do varicose veins and spider veins usually appear in the legs?

The force of gravity, the pressure of body weight, and the task of carrying blood from the bottom of the body up to the heart make legs the primary location for varicose and spider veins.

Compared with other veins in the body, **leg veins** have the toughest job of carrying blood back to the heart. They endure the most pressure. This pressure can be stronger than the veins' one-way valves.

Are varicose veins and spider veins painful or dangerous?

Spider veins usually do not need medical treatment. But varicose veins usually enlarge and worsen over time. Severe varicose veins can cause health problems.

These include:

Severe venous insufficiency. This severe pooling of blood in the veins slows the return of blood to the heart. This condition can cause blood clots and severe infections. Blood clots can be very dangerous because they can move from leg veins and travel to the lungs.

Blood clots in the lungs are life-threatening because they can block the heart and lungs from functioning.

Sores or skin ulcers can occur on skin tissue around varicose veins.

Ongoing irritation, swelling and painful rashes of the legs.

What are the signs of varicose veins?

Some common symptoms of varicose veins include:

Aching pain

Easily tired legs

Leg heaviness

Swelling in the legs

Darkening of the skin (in severe cases)

Numbness in the legs

Itching or irritated rash in the legs

How to prevent varicose veins and spider veins?

Not all varicose and spider veins can be prevented. But some things can reduce your chances of getting new varicose and spider veins. These same things can help ease discomfort from the ones you already have:

Wear Sunscreen to protect your skin from the sun and to limit spider veins on the face.

Exercise regularly to improve your leg strength, circulation, and vein strength. Focus on exercises that work your legs, such as walking or running.

Control your weight to avoid placing too much pressure on your legs.

Do not cross your legs when sitting.

Elevate your legs when resting as much as possible.

Do not stand or sit for **long periods of time**. If you must stand for a long time, shift your weight from one leg to the other every few minutes. If you must sit for long periods of time, stand up and move around or take a short walk every 30 minutes.

**Wear elastic support stockings
and avoid tight clothing that
constricts your waist, groin, or
legs.**

Eat a low-salt diet rich in high-fibre foods. **Eating fibre reduces the chances of constipation which can contribute to varicose veins. High fibre foods include fresh fruits and vegetables and whole grains, like bran. Eating too much salt can cause you to retain water or swell.**

Some available treatments include:

Sclerotherapy

Laser surgery

Surgery

Nutritional and Natural medicines

**Homocysteine
factors (i.e.
Vitamin C, P-5-P)**

**Horsechestnut
Hawthorne
Collagen**



RAYNAUD'S SYNDROME



Raynaud's syndrome or phenomenon, is a disorder of blood circulation in the fingers. This condition aggravate with cold exposure. Exposure to cold abnormally reduces blood circulation causing the fingers to become pale, waxy-white or purple. Sometimes called "white finger", "wax finger" or "dead finger."

Typical attacks occur with:
tingling and slight loss of feeling
or numbness in the fingers,
blanching or whitening of the
fingers, usually without affecting
the thumb, and
pain, sometimes with redness,
which accompanies the return of
blood circulation generally after 30
minutes to two hours.

How do you live with Raynaud's phenomenon?

Precautions can be taken to reduce the number and intensity of attacks of white finger. These precautions include the following:

Protect the body from cold temperatures.

Avoid immersing unprotected hands in cold water.

Protect the hand from injury.

Avoid tobacco since nicotine sometimes causes poor blood circulation in the fingers.

Dress completely for cold weather by wearing gloves, overcoat, hat and scarf.

Nutritional and Natural medicines

**OPC's (Grapeseed extract)
Borage**

CHILBLAINS



A chilblain is a small, red swelling on the skin which can be very itchy and gradually becomes very painful. Chilblains usually occur on the smaller toes but can occur on the finger, face and the nose. They occur due to an abnormal reaction of the body to cold.

APPEARANCE

A chilblain will usually appear as a red, swollen lesion.

They can dry out leaving cracks in the skin, which expose the skin to infection.

SYMPTOMS

The lesion becomes increasingly painful.

The lesion becomes very itchy.

Patients may suffer from a burning sensation on their feet.

In extreme cases the surface of the skin may break and an infection may develop.

CAUSES

A chilblain is an abnormal reaction to cold.

Elderly people with a poor circulation are at a greater risk.

Young adults who work outdoors or in cold conditions, such as butchers, are also at risk.

If the skin is chilled and then followed by too rapid warming such as a gas fire, a chilblain may develop.

Damp living conditions can also be a contributing factor.

The sudden onset of very cold water on the skin can also lead to a chilblain.

Other contributing factors include dietary, hormonal imbalance and people who suffer from anemia.

WHAT YOU CAN DO

Keep legs and body warm, especially if you have poor circulation.

Leg warmers and thick woollen socks may be of benefit.

Nutritional and Natural Medicines

**OPC's (Grapeseed extract
Borage oil**

**Nitric oxide factors (Arginine,
Zinc, Vitamin C)**