Prof. Sydney Bush
DOpt PhD
The terms vitamin C and ascorbate and ascorbic acid and monodehydroascorbate (oxidised vitamin C) are loosely interchangeable.
Biographical details...


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CardioRetinometry ® eec (Reg US Pat+) Hearteries® AntiCoronary Clinics (UK) Ltd
55yrs Optometry,
52 yrs in Contact lens practice. Various lens patents.
Discovered double national average of Glaucoma in Hull 1979
1st to introduce frequent contact lens replacement 1984.

Why should we be interested in the retina for statins, or anything else? It is the most important square inch of the body.

Prof. William Havener states in Chapter 1 of his Synopsis of Ophthalmology (abbreviated)

“I will challenge every clinician that, using the combined resources of all the universities, their libraries and their professors, that I, with my square inch of the body, will diagnose more diseases than they, using any other square inch of the body of their choosing - except the other eye.”
My Response to Prof. Havener is –
Give me homozygous twins and I, using nothing more than CardioRetinometry® will evaluate. . .

The effects of chlorine in the public water supply.

The effects of Fluoride,

The effects of margarines

The effects of polyunsaturated oils,

The effects of Ginkgo Biloba.

The effects of vitamin C/ etc etc.

And in older twins, the effects of statins.
“...Chronic unbalanced Circadian Atheroma is advanced as the principal aetiological factor in CHD. It is diagnosable from the retinal atheroma and any subject in the Wong presentation would (benefit) from therapy ---I have many hundreds of such images ---often belonging to people with low to normal cholesterol when statins are irrelevant . . . My invitations to GPs to cooperate are ignored.”
It should be noted that by 2004 I was 100% confident that there was the closest possible correspondence between Coronary and Retinal atheroma as a result of my observations of atheroma and patients histories. It was not until two years later that I saw the Michelson, Morganroth, Nichols and MacVaugh paper and then the Tedeschi-Reiner et al paper establishing the link.
I need to inform you now that - because CardioRetinometry® and Vitamin C are still suppressed by Official Pharmaco-Medicine – from the NLM peer reviewed journals of Optometry and Medicine (apart from little read ‘Rapid Responses’ in the British Medical Journal) you have not been able to read about them.
My job today is to fill the gaps created in your knowledge by vested interests, and send you away with information that will — if applied — extend the lives of each and every one of you, and make the journey and expense worth while. I shall do that immediately.
A great deal of what you will learn has been withheld from you especially if you are a medical physician trained in a Western Medical School.
It is impossible to acquaint you with the many ways in which essential knowledge for the practise of medicine has been suppressed. We simply don’t yet know them all. Many have however become very obvious such as the way the journals are patrolled and editors sacked who publish anything detrimental to pharmacy profits. Citations are obviously sparse
Unfortunately many physicians are completely unaware of the war that exists between Pharmaceutical interests and the public. The more we learn, the more some our faith in physicians is damaged. That renders a huge disservice to honest doctors to many of whom I am grateful for helping to keep me alive.
An example of how you and they, have been kept in ignorance and disinformed is in some of the most widely used text books for medical students which, from the very outset of their careers, destroy the young doctors’ ability to practise good medicine.
This may sound harsh until we consider that **SCURVY** related diseases probably account, directly or indirectly, for over 70% of DEATHS, and to give examples of the continuing deceit, let us immediately acknowledge that . . .
Every one of those 70% of DEATH CERTIFICATES should state e.g.,

“SCURVY- manifesting as coronary thrombosis,”
or
“SCURVY manifesting as stroke,”
or aortic aneurysm, or septicaemia, or viral pneumonia, or meningitis or fifty other diseases. . .
Death certificates don’t say this possibly because e.g., two standard textbooks (similar to many others) Guyton and Hall’s, *Medical Physiology* and Baines and Dominiczak’s *Medical Biochemistry*, in their combined 1,744 pages, mention scurvy only three times; vitamin C much the same; and the only injectable form of vitamin C - sodium ascorbate - not at all.
Yet it was with injected sodium ascorbate, that Dr. Frederick R. Klenner, in 1949, CURED 59 of sixty cases of Polio who all WALKED out of his hospital.

All the others treated by his colleagues either died or were paralysed for life.
Doesn’t all this strongly suggest that if the medical course were properly constituted, free of the influence of pharmaceutical patronage, it would include several textbooks on vitamin C alone, with another dedicated to vitamin E.
The next slides will start to shock you into a realisation of the corruption and fraud in the current teaching of Medicine.

More shocks will follow.
### Dr Robert Cathcart’s Table from His Famous 1981 Paper:
**Vitamin C, Titrating to Bowel Tolerance, Anascorbemia and Acute Induced Scurvy; Medical Hypotheses. 1981 Nov;7(11):1359-76.**

**VITAMIN C, TITRATING TO BOWEL TOLERANCE, ANASCORBEMIA, AND ACUTE INDUCED SCURVY**

**TABLE I - USUAL BOWEL TOLERANCE DOSES**

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>GRAMS ASCORBIC ACID PER 24 HOURS</th>
<th>NUMBER OF DOSES PER 24 HOURS</th>
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<tr>
<td>normal</td>
<td>4 - 15</td>
<td>4 - 6</td>
</tr>
<tr>
<td>mild cold</td>
<td>30 - 60</td>
<td>6 - 10</td>
</tr>
<tr>
<td>severe cold</td>
<td>60 - 100+</td>
<td>8 - 15</td>
</tr>
<tr>
<td>influenza</td>
<td>100 - 150</td>
<td>8 - 20</td>
</tr>
<tr>
<td>ECHO, Coxsackievirus</td>
<td>100 - 150</td>
<td>8 - 20</td>
</tr>
<tr>
<td>mononucleosis</td>
<td>150 - 200+</td>
<td>12 - 25</td>
</tr>
<tr>
<td>viral pneumonia</td>
<td>100 - 200+</td>
<td>12 - 25</td>
</tr>
<tr>
<td>hay fever, asthma</td>
<td>15 - 50</td>
<td>4 - 8</td>
</tr>
<tr>
<td>Environmental and food allergy</td>
<td>0.5 - 50</td>
<td>4 - 8</td>
</tr>
<tr>
<td>burn, injury, surgery</td>
<td>25 - 150+</td>
<td>6 - 20</td>
</tr>
<tr>
<td>Anxiety and other</td>
<td></td>
<td></td>
</tr>
<tr>
<td>mild stresses</td>
<td>15 - 25</td>
<td>4 - 6</td>
</tr>
<tr>
<td>cancer</td>
<td>15 - 100</td>
<td>4 - 15</td>
</tr>
<tr>
<td>ankylosing spondylitis</td>
<td>15 - 100</td>
<td>4 - 15</td>
</tr>
<tr>
<td>Reiter's syndrome</td>
<td>15 - 60</td>
<td>4 - 10</td>
</tr>
<tr>
<td>acute anterior uveitis</td>
<td>30 - 100</td>
<td>4 - 15</td>
</tr>
<tr>
<td>rheumatoid arthritis</td>
<td>15 - 100</td>
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</tr>
<tr>
<td>bacterial infections</td>
<td>30 - 200+</td>
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</tr>
<tr>
<td>infectious hepatitis</td>
<td>30 - 100</td>
<td>6 - 15</td>
</tr>
<tr>
<td>candidiasis</td>
<td>15 - 200+</td>
<td>6 - 25</td>
</tr>
<tr>
<td>CONDITION</td>
<td>GRAMS ASCORBIC ACID PER 24 HOURS</td>
<td>NUMBER OF DOSES PER 24 HOURS</td>
</tr>
<tr>
<td>----------------------------------------</td>
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</tr>
</tbody>
</table>
• Typical disinformation warfare by the Pharmaceutical industry against the public.
• More Pharmacy
• Disinformation warfare:
• Newspapers play this game.

Typical Daily Mail article defining scurvy as “rare” and vitamins a waste of money.
CardioRetinometry® is now ready as a completely new scientific diagnostic tool ignored and suppressed to date. It will empty hospital cardiology wards.

The generic name of the new science is “Metabolic Retinology”

It is a completely new system of preventive medicine
Every one therefore, of the conditions described, predisposes to those that are not listed, the long term consequences of chronic deficiency.
And the short term pathologies of the listed conditions will predispose to the eventual myocardial infarction or stroke apparently completely unrelated to the listed disease.
Which, as you now start to join the dots, can be seen to explain how Influenza shapes up into a coronary thrombosis. The overtaxed, high revving heart, and its coronary circulation, weakened by the same influenzal infection, chronic occult scurvy that predisposed to the hepatiits, or the influenzal infection, or bacterial infection, ends in sudden thrombosis.
Perhaps it is still unclear as to how one leads to the other.
It is easier to understand if you consider that I myself and each and every one of you here, has a degree of coronary atheroma.

If the atheroma is at the level of – and here I would guess – my Grade 1, or more, and is chronic the sequence of events leading to the thrombosis is well defined.
Atheroma must be understood to be a lesion of intraluminal plaque, first and foremost dedicated to the reinforcement of the arterial endothelial lining.
At this point I should add that the world’s authority on the pathogenesis of thrombosis is Dr. Matthias Rath MD and I should acknowledge that knowing much less than he, I can only appear to be an incompetent usurper of his pathophysiologic ground, a relative ignoramus, and should really remain silent to remove all doubt.
However, he isn’t here so it falls to me to elucidate how atheroma, oxidised lipoprotein alpha, (special to Homo Sapiens) a surrogate for vitamin C, invaded by calcium, platelets macrophages and other plasma constituents, is attached to and permeates the endothelium, maintaining the system watertight at all levels of blood pressure.
As such, the plaque must be defended against colonisation by opportunistic bacteria, which would be a failed example of inflammation causing heart attacks.
The plaque, in order to be defended against such colonisation, is invaded by new capillaries. These provide the ready access by phagocytic polymorphonuclear white cells in the plasma, needed to seek and destroy foreign organisms.
These vessels exist, by definition, in chronic sub-optimal occult scurvy, wherever vitamin C deficiency has predisposed to weak collagen, and the necessity for the deposition of the surrogate Lp(a) – the plaque!
Again – by definition – in the condition of occult scurvy due to chronic sub-optimal plasma ascorbate – the new fragile vessels (neovascularisation) are vulnerable to haemorrhage. In the skin such fragility leads to petechiae, purpura, ecchymoses, and – easy bruising as subdermal haemorrhages occur.
The heart is beating non-stop and sixty pulse waves per second expand and contract the arteries. Weakness in the vessel walls is found by the turbulence, expansion and pressure surge, manifesting as intraluminal haemorrhage, between arterial wall and plaque. Pressure from pooling and oedema, breaks the embrittled plaque, forming thromboses.
Prethrombotic events resulting therefore, from chronic occult scurvy, are life shortening, and demand a means of evaluating degrees of occult scurvy
Official medicine treats scurvy as like pregnancy. No intermediate degrees are recognised. You either have it or you are healthy.
Professor Steve Hickey PhD and Dr Hilary Roberts PhD, did me the great honour of devoting the chapter of their book on “Quantifiable biomarkers for scurvy” to CardioRetinometry® in 2004.

It seems that (summarised) it is the “third technique – it is new – and it provides for the quantification of the effect of vitamin C on blood vessels in the retina.”
They suggest that the first method of evaluation of vitamin C needs is Dr Cathcart’s Bowel Tolerance.

The second is the assay of the vitamin C in red blood cells – NOT – as chosen by the physicians, the white cells, which have their own special pumps for extracting ascorbate from plasma.
CardioRetinometry® for the first time, allows direct observation and evaluation of the effects of every kind of nutrient, medication and toxin or allergen on the vascular endothelium. Scurvy detected as never before and quantifiable over any period of time!
CardioRetinometry® therefore, probably has the power to dramatically increase the Life expectancy of everyone here, no matter how well you think you supplement. No other system provides effective monitoring of arterial health either in such microscopic degree, so safely, so frequently, or so cheaply.
CardioRetinometry® can therefore be expected to provide almost continuous readout of endothelial response depending on the resolution of the fundus camera, and those able to resolve corpuscles are expected to be best of all.
And this brings us to the kernel of this talk.
STATINS:

CardioRetinometry® can therefore be expected to provide clear information of the comparative efficiency of statins vis-à-vis ascorbate.
Because Vitamin C reduces intraluminal plaque, as shown by thousands of ‘before and after’ retinal photomicrographs,
• It is impossible to tell
• just by looking at them,
• Which atherolysis was achieved
• by statin and which
• by ascorbate.
• And so, because they all look alike, it is pointless differentiating.

• Vitamin C and Statins act via the same biochemical pathway exactly!

• This is what we see.
But first – it must be noted that, according to my reliable good friends Michelson, Morganroth, Nichols and MacVaugh (Arch. Int. Med. Vol. 139, Oct 1979) Retina is a near perfect surrogate indicator of coronary heart disease (CHD). So Physicians can estimate coronary disease from retina and vice versa.
Retinal Arteriolar Changes as an Indicator of Coronary Artery Disease

Eric L. McConnell, MD; Joel Morgaroity, MD; Charles W. Nichols, MD; Horace MacVaug, MD

Funduscopic examination was performed in 70 non-diabetic, non-hypertensive patients without valvular heart disease undergoing coronary angiography for evaluation of chest pain syndromes to determine if retinal arteriolar changes could reliably predict presence of coronary artery disease. Retinal arteriolar changes were graded with respect to light reflex, vessel caliber, arteriovenous crossing defects, and changes tortuously without knowledge of angiographic findings. Each coronary vessel was graded with respect to its most occlusive lesion by angiography; coronary index was derived for each patient without knowledge of eye findings. Abnormal light reflex changes were the most sensitive indicators of presence and extent of coronary artery disease. Abnormal vessel tortuosity and decreased caliber were less sensitive but more specific; their presence also suggested more extensive coronary lesions. The loss of retinal arteriolar lesions may indicate presence of coronary artery disease and may correlate with extent of lesions in selected patients.

(Arch Intern Med 139:1139-1141, 1979)

The early recognition of coronary artery disease has important implications both for the care of individual patients and for the evaluation of specific interventions that might alter the natural history for large populations at risk. Epidemiologic studies previously have identified major risk factors associated with atherosclerotic cardiovascular disease, including hypertension, hyperlipidemia, and cigarette smoking. As early as 1917, clinopathologic studies suggested an association between small vessel retinal arteriolar and large vessel (atherosclerotic) cerebral vascular lesions. Later, Wagener and Keith and Scheie described specific retinal arteriolar changes and considered these to be the sequelae of long-standing or severe systemic hypertension. Subsequently, changes in the conjunctival microvasculature were demonstrated to occur in patients with clinically and angiographically confirmed atherosclerotic coronary artery lesions. Previous studies, however, have not attempted to correlate retinal arteriolar changes with the presence or extent of coronary artery disease in a non-diabetic, non-hypertensive population.

MATERIALS AND METHODS

Funduscopic examinations were performed (by C.W.) on 70 patients undergoing coronary angiography for the evaluation and management of chest pain syndromes. Fifty-two were men; the mean age (±SD) for all patients was 48 ± 16 years, with no significant difference between the male and female patients. Funduscopy was performed within two weeks of coronary angiography except in nine patients in whom the procedure was done during an outpatient evaluation within two months after coronary angiography. All patients were no older than 60 years of age and all were without systemic hypertension or diabetes mellitus by history, physical examination, or laboratory determinations. No patients had sickle cell disease, anemia (hemoglobin level < 120 g/dl), or other systemic or retinal disorders known to affect arteriolar findings.

Funduscopic findings (Figure) were graded by the ophthalmologist (C.W.) with respect to arteriolar light reflex changes, vessel tortuosity, arteriolar vessel caliber, and arteriovenous crossing defects as follows:

- **Light Reflex Changes**
  - Grade (G) I: Normal or thin-walled arterioles
  - GI: Minimal light reflex
  - GII: Increased, approaching whole width of arteriolar wall
  - GIII: Color change, copper wiring
  - GIV: Obliteration of vessel wall, silver wiring

- **Vessel Caliber**
  - Normal
  - Mild arteriolar narrowing
  - Moderate narrowing
  - Considerable narrowing

- **Arteriovenous Crossing Defects**
  - Normal
  - Mild: Indication only, "humping" or mild tapering of vessels
  - Moderate: Apparent compression of vessels
  - Marked: Apparent compression at every crossing with obliteration of vessel

The examiner was unformed about the angiographic findings, and the retinal changes were graded specifically without adjustment for age. A G I light reflex was considered abnormal.

All fundi were examined with the iris dilated with one drop of 1% tropicamide plus one drop of 2.5% phenylephrine hydrochloride and all vessels were evaluated at greater than 1 and usually at 2 disc diameters from the disc margin. Twenty patients underwent a second funduscopic examination within seven to ten days after their initial examination. A comparison of funduscopic scores demonstrated meaningful intraobserver variation in grading. In addition, 30 patients also were examined by another ophthalmologist. There was no meaningful interobserver variation in the grading of any of these retinal arteriolar changes.

Coronary angiograms were scored by the consensus of three cardiologists without knowledge of eye findings as follows:

- **Coronary Score**
  - G0: Normal or ≤ 50% stenosis of all vessels
  - G1: 50% to 99% stenosis of any vessel (other than left main coronary artery)
  - G2: ≥ 70% stenosis of any vessel or ≤ 50% occlusion of left main coronary artery
  - G3: 70% to 99% stenosis of the right coronary artery, left anterior descending, circumflex, and ≥ 50% stenosis of the left main coronary artery

Stenoses were graded with respect to internal vessel diameters. A total score of 2 or 3 was considered to indicate the presence of significant coronary disease.

Retinal Arteriolar Changes—McConnell et al
had a normal light reflex. None of the 23 patients with <G1 coronary disease, however, had >GII light reflex changes (100% specificity). Similarly, 14 of 15 patients with a normal light reflex and 13 of 14 patients with no retinal abnormalities were without significant coronary lesions; but these numbers were too small in this preliminary study to determine the usefulness of normal retinal arteriolar findings in predicting normal coronary arteries.

It was also apparent from our data that, although there were only 18 female patients in this series, they accounted for 14 of the 23 patients with normal coronary arteries and for five of the nine patients with abnormal light reflex changes and insignificant coronary lesions. These data suggest, therefore, that funduscopic findings may be more reliable in predicting the presence of coronary artery disease than predicting the absence of coronary disease,
The data suggest that, in selected patients, abnormal funduscopic findings reflect the presence and extent of coronary artery disease. Retinopathy was evaluated with respect to light reflex changes, vessel wall caliber, vessel tortuosity, and arteriovenous crossing defects. Even minimal light reflex changes were found to be very sensitive although not specific indicators of the presence of coronary artery disease.

An abnormal light reflex identified 46 of 47 patients with coronary artery disease (98% sensitivity). Although an abnormal light reflex (≥ GI) was not highly specific (61%) for the presence of coronary artery disease, a more abnormal light reflex (≥ GII, 78% specificity; ≥ GII, 100% specificity) was specific and, in addition, predicted more extensive underlying coronary artery disease. It is important to emphasize that a GI light reflex represents a minimal abnormality and that in routine clinical practice a nonophthalmologist would probably not detect less than
But according to my reliable good friends Michelson, Morganroth, Nichols and MacVaugh (Arch. Int. Med. Vol. 139, Oct 1979) Retina is a near perfect surrogate indicator of coronary heart disease (CHD). So physicians can estimate (in reverse) Retinal Arterial Disease via the coronaries.

Eye Findings in Patients Without Significant Coronary Disease (23 Patients)

**Light Reflex Changes.**—Only 14 of the 23 patients without significant coronary disease (61% specificity) had a normal light reflex; none, however, had $> G2$ light reflex changes (100% specificity). Of the 15 patients with a normal light reflex, 14 (94%) had no significant coronary artery disease.

**Vessel Tortuosity.**—Twenty-two of these 23 patients had normal vessel tortuosity (96% specificity). Of the 60 patients with normal tortuosity in this series of 70 patients, however, only 27 had insignificant coronary disease.

**Vessel Caliber.**—Of the 23 patients with $< G1$ coronary artery disease, 20 had normal caliber (87% specificity).
Comparison of Light Reflex Changes and Extent of Coronary Artery Disease*

<table>
<thead>
<tr>
<th>Coronary Score</th>
<th>0</th>
<th>0-I</th>
<th>I</th>
<th>I-II</th>
<th>II</th>
<th>II-III</th>
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<tr>
<td>0</td>
<td>11</td>
<td>3</td>
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<tr>
<td>1½</td>
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<td>2½</td>
<td>0</td>
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<td>1</td>
<td>2</td>
<td>8</td>
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<td>0</td>
<td>1</td>
<td>0</td>
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</table>

* *N = 70 patients; coefficient of correlation r = .60, P < .0001.*

had < G1 coronary disease.
Since we can get no further cooperation from the cardiologists we rely on common sense and the information in their papers re their grading of arterial disease.

REMEMBER

49% blockage is their Grade ZERO!
Hypertension Origin?

Artery

Cholesterol at 49% blockage

In this vein?
Hypertension Origin?

More pink circulation returns
Hypertension Origin?

• Cholesterol Blocks
Hypertension Origin?

- Dissolves

- Note how vessels widen with increased blood-flow and at the arterio-venous crossover the artery becomes more transparent. The vessels jump about because the whole vasculature is recovering its former shape and the entire retina changes colour as the cholesterol disappears and the blood returns.
Hypertension Origin?

• Cholesterol Blocks
Hypertension Origin?
Girl
Age 10
After 1 Gram
Vit C/day
Girl
Age 9
Girl
Age 10
After 1
Gram
Vit C/day
Aim: An increase in carotid intima-media thickness (CIMT) represents an early phase of the atherosclerotic process. The aim of our study was to evaluate whether a reduction in CIMT could be seen with 1-year treatment with rosuvastatin (10 mg/day). Methods and Results: Forty-five patients with hypercholesterolemia and asymptomatic carotid atherosclerosis on baseline carotid ultrasound investigation (CUI) were examined with repeat CUI after 1 year of treatment (rosuvastatin 10 mg/day). Demographic and lifestyle data were collected. A physical examination was performed, and fasting venous blood samples were obtained. Total cholesterol, low-density lipoprotein cholesterol and triglycerides decreased significantly (p < 0.001), while high-density lipoprotein cholesterol increased significantly (p < 0.001) during the intervention. The mean decreases in the IMT of the right and left common carotid arteries (CCAs) were 0.29 and 0.26 mm, respectively (p < 0.05 for each). Age and lipid profile parameters were significant predictors of change in CIMT in linear regression analyses after adjustment for established atherosclerosis risk factors. Conclusions: One-year treatment with rosuvastatin in hypercholesterolemic adults with evidence of subclinical atherosclerosis significantly reduced the CIMT of both CCAs and improved the lipid and lipoprotein levels.
Can we not expect ascorbate to perform even better and without risk of adverse drug reactions?
• Can we not confidently expect that ‘Big Pharma’ has – in fact – done the research with vitamin C, and is keeping quiet about it?
In the USA people using the LifeStream® home cholesterol monitor found that vitamin C was as effective as statins.
Can we not confidently expect that ‘Big Pharma’ has again here – done the research with vitamin C - and is keeping quiet about it?
If you were Big Pharma, wouldn’t it be sensible for you to know what threats your business faces?
The power to change the National Health Service, the elimination of almost all degenerative cardiovascular disease - and medicine as you know it today.
Professor Steve Hickey and Dr Hilary Roberts PhD., of Manchester Metropolitan University in their book "Ridiculous Dietary Allowance," detailing 100 farcical errors in the formulation of the RDA for vitamin C did me a great honour.
Ridiculous Dietary Allowance

An open challenge to the RDA for vitamin C

Dr Steve Hickey
&
Dr Hilary Roberts
They stated re the RDA for Vitamin C - that “the Public has been actively misled” and go on to say about the new science that it "represents a new technique for the estimation of Vitamin C requirements"
What has this to do with Statins?
It means the end of Statins
The reason for that is the shared biochemical pathway by which statins work in reducing plasma cholesterol.
But that is not all – it gets a bit complicated here
Atheroma – Arterial disease i.e. intraluminal plaque that obstructs our coronary arteries in probably everybody here
Is caused by cholesterol but NOT your High or Low density cholesterol.
It is caused by VERY low density cholesterol – Lipoprotein alpha [Lp(a)] barely found in other animals.
And this is a Risk
‘MARKER’
not a Risk
‘FACTOR’
Which means that when you **HAVE** high Lp(a) **you HAVE** heart disease!
What do statins do? They lower cholesterol! But NOT always Lp(a). Atorvastatin has the greatest effect.
So that’s good isn’t it?

NO! It Isn’t
What people are not told is that the Lp(a) cholesterol is deposited at critical points where the wear in the vessels is greatest.
On the septa of retinal arteriolar bifurcations in the eye I would call it Hollenhorst Microplaque.
the plaque is serving to keep the system watertight!!!

Hypothesis: lipoprotein(a) is a surrogate for ascorbate
Rath M, Pauling L.
And people are not told that the greatest danger can actually come from the veins - and particularly in the eye!
A Venous Pressure Change

Vein pushed up to arch higher as the cholesterol blocks entry to larger vein

Cholesterol block dissolving, blood-flow restored. pressure falls – arch falls.
Blockage of the veins can cause instant and permanent blindness
These images, still rejected for publication in peer reviewed journals, could not be of greater significance to everyone here!
They represent the missing link explaining why statins fail to provide life extension.
So why does this mean the end of statins?
I think you all fully understand now Because
1. Statins they act via the same HMG3 CoA Reductase enzyme inhibition pathway that Vitamin C acts through in reducing plasma cholesterol!
2. The pharmaco-medical profession has doubtless researched and confirmed that at any moment world-wide loss of confidence in statins can be expected at any moment when the public learns the truth about vitamin C..
Physicians cannot prescribe vitamin C ending so many diseases, without ending the practice of medicine in the West as it is exists now.
And at this point I am delighted to be able to tell you that we have exceeded 13 years of at times dramatic reversals of retinal atherosclerosis corresponding with, as my medical colleagues say, 26 years cardiovascular life extension.
Thank you for your attention.

Any Questions.