



CardioRetinometry® ® EEC. (Reg. US. Pat+):

Slayer of Statins

Sydney J Bush DOpt., PhD.

The terms vitamin C and ascorbate and ascorbic acid and monodehydroascorbate (oxidised vitamin C) are loosely interchangeable.

Biographical details...

Sydney J Bush DOpt., PhD.hc. (2004)

2nd MB Newcastle. Reverted to Optometry

1954 DOpt. Institute of Optical Science London

Snr. Rsch Scntst. NCHVAMedCentre. Chicago

Director UK Institute of CardioRetinometry® & Canadian Institute of

CardioRetinometry®

Distinguished Professor; Chief of Optometry

and CardioRetinometry Cosmopolitan University. Mo. USA. (2008) Correspondence:

bush@cosmopoplitanuniversity.ac

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.

Discovered reversibility of retinal arterial disease 1999. Named the new science of Nutritional Cardio Retinal Atherolysis CardioRetinometry(Reg) in Dec 1999.



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.



NOV 26th 2004 BMJ. Rapid Response to Wong

". . . 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, "log/2013



NOV 26th 2004 BMJ. Rapid Response to Wong

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. 27/09/2013



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.



2

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.



Doessn't all this strongly suggest that if the medical course were properly constituted, free of the influence of pharmceutical 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

	GRAMS ASCORBIC ACID	NUMBER OF DOSES
CONDITION	PER 24 HOURS	PER 24 24 HOURS
normal	4 - 15	4 - 6
mild cold	30 - 60	6 - 10
severe cold	60 - 100+	8 - 15
influenza	100 - 150	8 - 20
ECHO, Coxsackievir	8 - 20	
mononucleosis		12 - 25
viral pneumonia	100 - 200+	12 - 25
hay fever, asthma	15 - 50	4 - 8
Environmental and		
food allergy	0.5 - 50	4 - 8
burn, injury, surgery	y 25 - 150+	6 - 20
Anxiety and other		
mild stresses	15 - 25	4 - 6
cancer	15 - 100	4 - 15
ankylosing spondyli	itis 15 - 100	4 -15
Reiter's syndrome		4 - 10
acute anterior uveiti	s 30 - 100	4 - 15
rheumatoid arthritis	15 - 100	4 - 15
bacterial infections	30 - 200+	10 - 25
		6 - 15
infectious hepatitis candidiasis	15 - 200+	6 - 25



GRAMS A	SCORBIC ACID	NUMBER OF DOSES
CONDITION PER	R 24 HOURS	PER 24 24 HOURS
normal	4 - 15	4 - 6
mild cold	30 - 60	6 - 10
severe cold	60 - 100+	8 - 15
influenza	100 - 150	8 - 20
ECHO, Coxsackievirus	100 - 150	8 - 20
mononucleosis	150 - 200+	12 - 25
viral peumonia	100 - 200+	12 - 25
hay fever, asthma	15 - 50	4 - 8
Environmental and		
food allergy	0.5 - 50	4 - 8
burn, injury, surgery	25 - 150+	6 - 20
Anxiety and other		
mild stresses	15 - 25	4 - 6
cancer	15 - 100	4 - 15
ankylosing spondylitis	15 - 100	4 -15
Reiter's syndrome	15 - 60	4 - 10
acute anterior uveitis	30 - 100	4 - 15
rheumatoid arthritis	15 - 100	4 - 15
	30 - 200+	10 - 25
infectious hepatitis	30 - 100	6 - 15
candidiasis	15 - 200+	6 - 25



Vitamin C'is powerless' in the battle against colds

TAKING vitamin C to combat colds is a waste of time, scientists said

FRUIT and vegetables do not improve the survival chances of women with breast cancer, scientists have found. A study of more than 3,000 sufferers showed that

even boosting consumption way beyond normal guidelines and cutting fat intake did not help them live longer.

They were just as likely to die or suffer a recur-rence of breast cancer as those on a regular five-a-day' diet. In both groups during the study period, cancer returned in about 17 per cent of cases and 10 per cent of the women died.

Professor Marcia Stefanick, from Stanford University School of Medicine in California, who led the study, said: 'I was really surprised and, frankly, a little disappointed by the results.' But she said a healthy diet was important even

if a 'super-healthy' one wasn't.

the world, revealed the pills had little benefit. It found that those taking a daily dose of vit-min C is likely to benefit only those taking a daily dose of vitamin C were just two per centless likely to catch a cold. Their symptoms would clear up only marginally more quickly.

The researchers, from Finland and the Australian National University in Canberra, said this would equate to the average person suffering a cold for 11 days a year instead of 12. I seem research.

'You've just got to stick it out'



even a glass or two of orange juice do little to dry up the sniffles.

However, taking them will not do any harm - and simple belief that they work could

per year, they said.

The study also showed that starting to take the vitamin at the first sign of symptoms did little to hasten recovery.

It did find, however, that the young benefited marathon promise the first sign of the fi

The businessman who wants to give £1billion to charity

Typical disinformation warfare by the Pharmaceutical industry against the public.

Get your unfair bank charges back. With interest.

Why car

If you've been hit by unfair bank charges anytime in the last 6 years, you could be in line for a refund.

Call 0845 271 4004

27/09/2013





- More Pharmacy
- Disinformation warfare:
- Newspapers play this game. **Typical Daily Mail** article defining scurvy as "rare" and vitamins a waste of moneyous

O

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 prediposed 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.



0

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 then he, I can only appear to be an incompetent usurper of his pathophysiological 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!



O

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. Michelson, MD; Joel Morganroth, MD; Charles W. Nichols, MD; Horace MacVaugh III, MD

· Funduscopic examination was performed in 70 nondiabetic, nonhypertensive 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 vessel tortuosity 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. Thus, funduscopic examination demonstrating specific 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 I 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, hyperlipoproteinemia, and cigarette smoking.

As early as 1917, clinicopathological studies suggested an association between small vessel retina (arteriosclerotic) and large vessel (atherosclerotic) cerebral vascular lesions.2.3 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.4-6 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 nondiabetic, nonhypertensive population.

MATERIALS AND METHODS

Funduscopic examinations were performed (by C.W.N.) 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 ± 7 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 < 12.0 g/dL), or other systemic or retinal disorders known to affect

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

Vessel Caliber

Light Reflex Changes

Grade (G) 0	Normal thin-walled arterioles	Normal
GI	Minimally increased light reflex	Mild arteriolar narrowing
GII	Increased, approach- ing whole width of arteriolar wall	Moderate narrow- ing
GIII	Color change, copper wiring	Considerable nar- rowing
GIV	Obliteration of vessel wall, silver wiring	
Arteriove	enous Crossing Defects	Vessel Tortuosity
Normal	None	Normal
Mild	Deflection only, "hump- ing," or mild tapering of venule	Mildly increased
Moderate	Apparent compression	Moderately in-
	of venule	creased
Marked	Apparent compression	Considerably in-

obliteration of venule The examiner was uninformed about the angiographic findings, and the retinal changes were graded specifically without adjustment for age. A ≥ GI light reflex was considered abnormal.

at every crossing with

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



Normal to < 50% stenosis of all vessels ≥ 50% but < 70% stenosis of one vessel (other than left main coronary artery) ≥ 70% stenosis of one vessel or ≥ 50% occlusion of left main coronary artery

Each vessel was graded with respect to the most occlusive lesion, and a total corenary score was obtained by adding individual vessel scores to a maximum of grade 4: ≥ 70% 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 ≥ G1 was considered to indicate the presence of significant coronary disease.



Accepted for publication May 7, 1979.

From the Cardiovascular Section, Department of Medicine (Drs Michelson, and Morganroth), Department of Ophthalmology (Dr Nichols) and the Cardiothoracic Surgery Section (Dr MacVaugh), Department of Surgery, University of Pennsylvania School of Medicine, Philadelphia.

Reprint requests to Lankenau Hospital, 2221 Medical Science Bldg, City Line and Lancaster Avenues, Philadelphia, PA 19151 (Dr Morganroth).

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,

COMMENT

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

25 of these 32, however, had ≥ G2 coronary artery lesions

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 > GII 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 calibor (87% specificity)

Comparison	of Light	Reflex	Changes	and	Extent	of
	_		y Disease			

1	Coronary	1	Grade of Light Reflex Changes					45
	Score	al.	9-1	1	1-11	N II	11-111	IIIVIV
4	0	11	3	3	1	5	0./	0 0 0
- andre	1/2	0	0	0	0	0	0	000
	1 100	70	0	1	2	2	1	000
	11/2		0	0	2	0	0	000
4	2	0	0			8	4	000
	21/2	0	0	1	2	8	0	000
	3	0	0	2	4	25	83	000
	31/2	0	0	0	0	0100	91/1	000
	4	0	0	0	0	1 1	0	000

"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!

Artery

Cholesterol at 49% blockage In this vein?



More pink circulation returns



•Cholesterol Blocks

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



•Cholesterol Blocks





Girl Age 9



Girl Age 10 After 1 Gram Vit C/ day

Girl Age 9



Girl Age 10 After 1 Gram Vit C/ day

Pharmacology. 2010 Jan 21;85(2):63-67. One-Year Treatment with Rosuvastatin Reduces Intima-Media Thickness in 45 Hypercholesterolemic Subjects with Asymptomatic Carotid Artery Disease. Riccioni G, Vitulano N, Mancini B, Zanasi A, D'Orazio N.

- Cardiology Unit, San Camillo de Lellis Hospital, Manfredonia, Italy.
- 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: Fortyfive 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 是如此2013 Copyright © 2010 S. Karger AG, Basel.

 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!!!

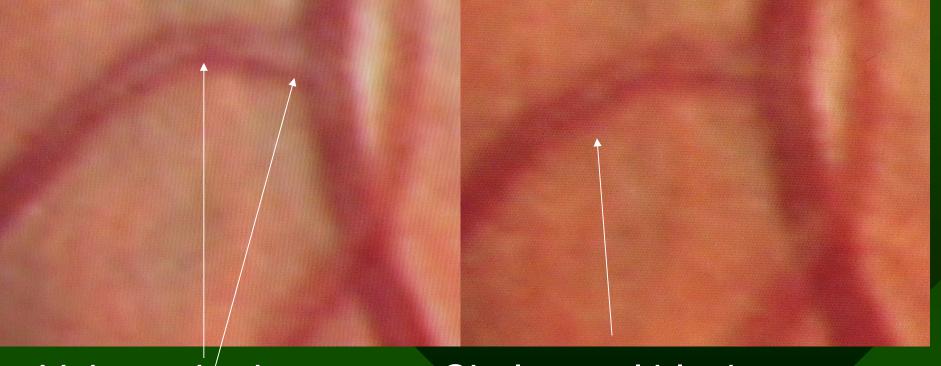
Hypothesis: lipoprotein(a) is a surrogate for ascorbate Rath M, **Pauling L.**Proc Natl Acad Sci U S A. 1990 Aug;87(16):6204-7.



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.

27/09/2013

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?



\mathcal{D}

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 leaarns 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.



