Brief Introduction to Your Architecture of Time

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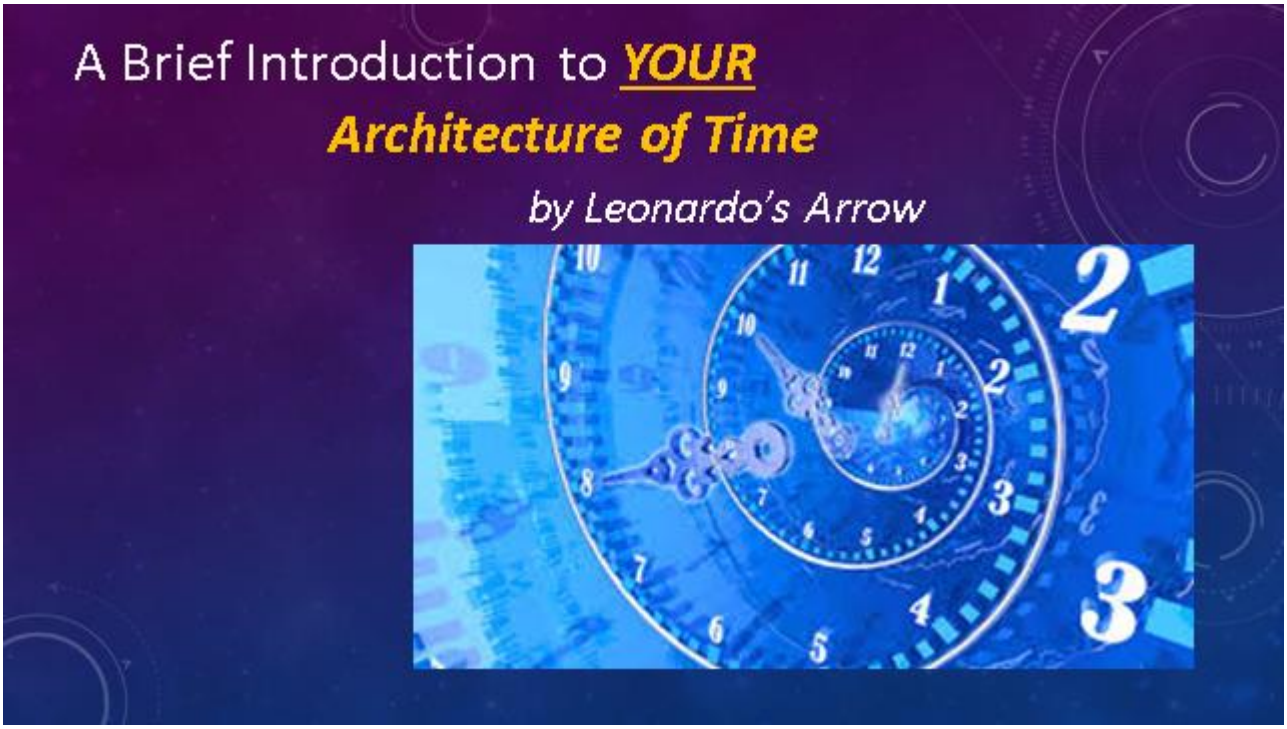


A special number connects ***you*** to the architecture of time. **Phi** is a ubiquitous constant that is fundamental to all life forms.[1] For example, our developmental milestones—birth, menarche, menopause, life expectancy, and upper limit of lifespan—are exponentially connected to it.[2]

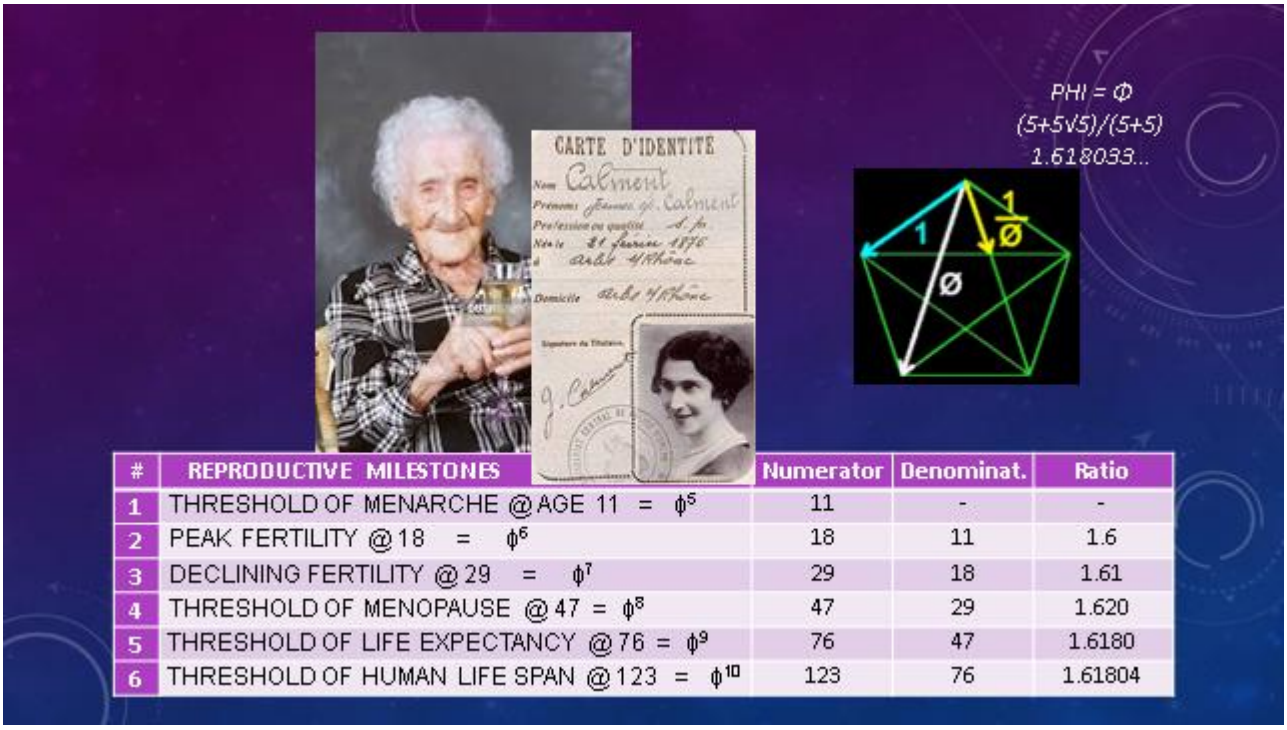
Biomarkers can serve as numerical representations of health and fitness. Medical examples include age, vital signs, body mass index, lab tests, and measurable images. Extremes are incompatible with longevity, and the ratio of most cutoffs converges on phi. In other words, the **ratio of normal limits** for vital biometrics such as blood pressure, heart rate, respiratory rate, body mass index, blood glucose,

and cholesterol—is consistently **phi ~ 1.6**, a remarkable relationship that could be used to calculate vascular risk.[3]

Carotid arteries can easily and noninvasively be measured by ultrasound. An ultrasound study of a diverse population over forty found that arterial plaque tends to thicken exponentially—by a factor of phi per decade.[4] A phi-based biometric index of vascular risk was developed in 2010.[5] It uses key biometrics to calculate ***cardiometabolic age (CMA)—***a useful surrogate for vascular risk. After forty, CMA closely matches the extent and progression of atherosclerosis. See **Appendix 1** for an example of a CMA' calculations for the co-author.

Footnotes

[1]Liu, Y., 2018, *Is the golden ratio a universal constant for self-replication?* PLOS ONE, July 16; v. 13(7).
https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0200601



#	REPRODUCTIVE MILESTONES	Numerator	Denominat.	Ratio
1	THRESHOLD OF MENARCHE @ AGE 11 = ϕ^5	11	-	-
2	PEAK FERTILITY @ 18 = ϕ^6	18	11	1.6
3	DECLINING FERTILITY @ 29 = ϕ^7	29	18	1.61
4	THRESHOLD OF MENOPAUSE @ 47 = ϕ^8	47	29	1.620
5	THRESHOLD OF LIFE EXPECTANCY @ 76 = ϕ^9	76	47	1.6180
6	THRESHOLD OF HUMAN LIFE SPAN @ 123 = ϕ^{10}	123	76	1.61804

[2] On a logarithmic scale, reproductive milestones M tend to line up as follows: Early menarche at 11, peak fertility at 18, declining fertility at 29, early menopause at 47, life expectancy at 76, and maximum lifespan at 122 ½, based on the

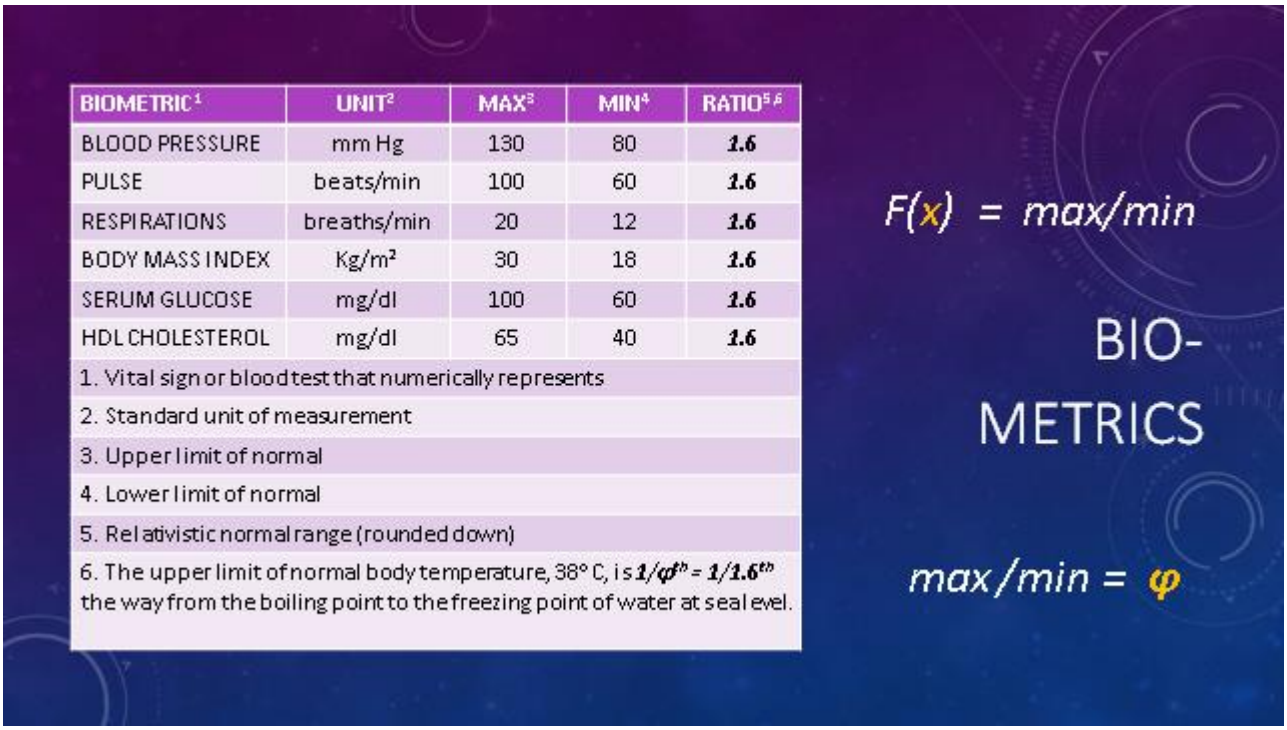
following equation, $F(x) \sim n \log_x M_n$, where n corresponds to serial numbers 5 through 10 and x represents the biometric constant phi. Grip strength, blood pressure, and plaque thickness follow the same general idea.

Iconaru. E. I., et al., 2018, Hand grip strength as a physical biomarker of aging from the perspective of a Fibonacci mathematical modeling. *BMC Geriatrics*, Nov 29; v. 18(1), p. 296. https://www.ncbi.nlm.nih.gov/m/pubmed/30497405

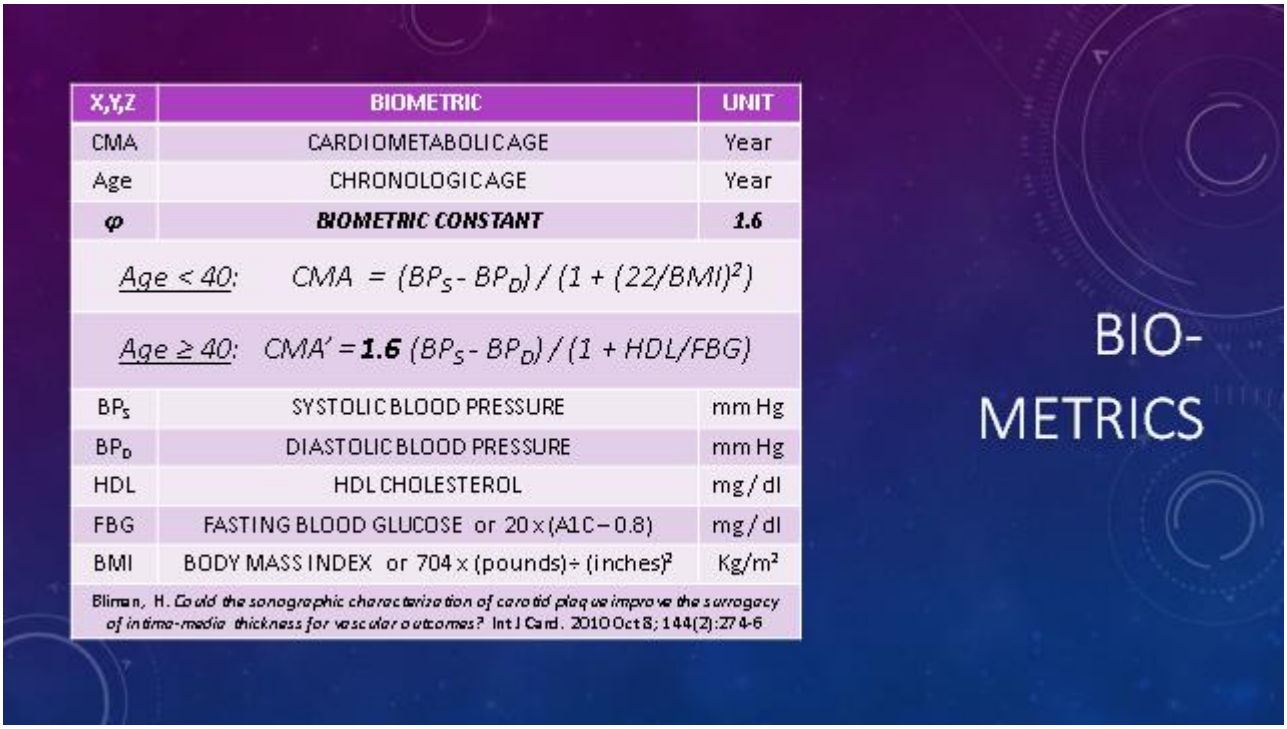
[3]Selzer, R. H., et al., 1994, Evaluation of computerized edge tracking for quantifying intima-media thickness of the common carotid artery from B-mode ultrasound. *Atherosclerosis*, Nov; v. 111(1), p. 1-11. https://www.ncbi.nlm.nih.gov/pubmed/7840805

[4]Dempsey, R. J., Diana, A. L., and Moore, R. W., 1990 , Thickness of carotid artery atherosclerotic plaque and ischemic risk. ISSN: 0148-396X , 0148-396X , 1524-4040. https://www.ncbi.nlm.nih.gov/pubmed/2234325

[5]Bliman, H., 2010, Could the sonographic characterization of carotid plaque improve the surrogacy of intima-media thickness for vascular outcomes? *International Journal of Cardiology*, Oct 8; v. 144(2), p. 274-276. https://www.sciencedirect.com/journal/international-journal-of-cardiology/vol/144/issue/2



Extremes are incompatible with longevity, and the ratio of most cutoffs converges on phi.



X,Y,Z	BIOMETRIC	UNIT
CMA	CARDIOMETABOLIC AGE	Year
Age	CHRONOLOGIC AGE	Year
φ	***BIOMETRIC CONSTANT***	***1.6***
Age < 40:	$CMA = (BP_S - BP_D) / (1 + (22/BMI)^2)$	
Age ≥ 40:	$CMA' = \mathbf{1.6}\ (BP_S - BP_D) / (1 + HDL/FBG)$	
BP_S	SYSTOLIC BLOOD PRESSURE	mm Hg
BP_D	DIASTOLIC BLOOD PRESSURE	mm Hg
HDL	HDL CHOLESTEROL	mg / dl
FBG	FASTING BLOOD GLUCOSE or 20 x (A1C – 0.8)	mg / dl
BMI	BODY MASS INDEX or 704 x (pounds) ÷ (inches)^2	Kg/m^2

Bliman, H. *Could the sonographic characterization of carotid plaque improve the surrogacy of intima-media thickness for vascular outcomes?* Int J Card. 2010 Oct 8; 144(2):274-6

The following is a brief introduction to CMA—Cardiometabolic Age.

The table above gives a couple of medical applications that the author has used to guide his management of vascular disease. Successful applications in a wide variety of fields have been reported,[6] and in 2011, Dr. David Shechtman won the Nobel Prize in Chemistry for his discovery of quasicrystals − crystals that defy symmetry by a factor of phi per quarter turn.[7]

Cardiometabolic Age (CMA) is a composite index of vascular risk. It was developed in 2007 as a risk assessment tool for a college-age population, but the following year, it was adapted for an older population, using a separate, but similar equation, CMA'.[8] Separate equations were needed because the context and meaning of "vascular risk" is very different for young and old.[9] Under forty, CMA represents fitness, while over forty, CMA' is a surrogate for disease (hardening of the arteries by the build-up of plaque). A composite index can improve the signal to noise ratio of vital biometrics, and units of time can prove that "premature aging" is a better surrogate for vascular events than vascular risk. As a result, CMA and CMA', in relation to chronologic age, could be a practical way to track the

trajectory of vascular health.[10] Practical applications in other fields support this principle, that… "The blueprint for life is to grow by phi per quarter turn, because most deviations and mutations are simply unsustainable."

Continued Foornotes

[6]Livio, Mario (2003) [2002]. The Golden Ratio: The Story of Phi, the World's Most Astonishing Number (First trade paperback ed.). New York City: *Broadway Books*. ISBN 0-7679-0816-3

[7]Schechtman, David. The Nobel Prize in Chemistry 2011". Nobelprize.org. Retrieved 2011-10-06

[8]Bliman, H. 2010, Could the sonographic characterization of carotid plaque improve the surrogacy of intima-media thickness for vascular outcomes? *International Journal of Cardiology*, Oct 8, v. 144(2), p.274-276. https://www.sciencedirect.com/journal/international-journal-of-cardiology/vol/144/issue/2

[9]Under age forty, CMA equals pulse pressure divided by the sum of one plus the ratio of a thousand divided by the product of pulse and body mass index; over age forty, CMA' equals the product of pulse pressure and phi divided by the sum of one plus the ratio of HDL cholesterol to glucose. Both CMA and CMA' are expressed in units of years. As a result, when compared to chronologic age, the ratio of pulse pressure divided by the sum of one plus the ratio of two important biomarkers, is directly related to the rate of vascular aging. The separation of CMA and CMA' at a cutoff of forty is not arbitrary; it was chosen with the help of phi, since the upper and lower limit of clinical utility occurs when their respective signals are equipotent, $1/\phi$ the way from life expectancy to the age of menarche, $(76 - 18) / 1.618 = 40$.

[10]**Appendix 1** shows a CMA' calculation based on coauthor Lorence Collins' resting blood pressure, HDL cholesterol, and fasting blood glucose, 0and confirms his trajectory of "graceful aging" (at age 87 ½, as of December 7, 2018) using the accompanying snapshots.

What is your trajectory?

A Brief Introduction to the Timestamp of Risk

In the developed world, vascular disease is the leading cause of death, and life expectancy is closely connected to vascular risk.[11] Since risk is a measure of frequency, and frequency is the inverse of time, **vascular age** could be used to predict vascular events.[12,13] For example, if a vascular event is defined as a heart attack, stroke, or preemptive intervention, then the **timestamp** of vascular age could identify sentinel milestones in the life of an artery. Milestones are not limited to outcomes; they also include the timestamps of modifiable risk factors such as smoking, diabetes, hypercholesterolemia, obesity, and a lack of exercise. The connection of risk factors to vascular outcomes is complex and nonlinear, but a sentinel timestamp can integrate the timing and magnitude of each component of vascular risk in a meaningful way. For example, a fifty pack-year history of smoking might shave seven years off the life expectancy of a seventy-year-old man; a forty-year history of uncontrolled diabetes might shave eight years off the life expectancy of seventy-year-old woman; and in a younger population, a long history of morbid obesity, lack of exercise, and drug abuse could have more dire consequences. These time differentials (with respect to chronologic age) are sentinel timestamps that demonstrate **accelerated vascular aging**. In all three cases, the rising integral of wear and tear predicts a reciprocal drop in life expectancy.

Seven numbers connect arteries to accelerated vascular aging. They are chronologic age, systolic blood pressure, diastolic blood pressure, heart rate, cholesterol, glucose, and body mass index. The connection of each of these risk factors to premature aging and each other is complex and nonlinear. But the ratio of upper to lower norms for all, except chronologic age, is exactly **1.6**. This0 property made it possible to combine risk factors in an equation that predicts the variable trajectory of vascular aging. Different units of measure were cancelled out by coefficients that standardize pressure to years and heart rate to body mass-index. Two different equations are needed, depending on age and available blood tests. The numerators of vascular age are proportional to a representative difference between systolic and diastolic pressures (averaged over three readings). Under age forty, the denominator is one plus the inverse product of heart rate and standardized body mass index. Over age forty, the denominator is one plus the

ratio of good cholesterol to glucose. Such **vascular age calculations** can track vascular fitness, uncover disease, and guide treatment.

Continued Footnotes

[11]Timmis, A., Townsend, N., Gale, C., Grobbee, R., Maniadakis, N., Flather, M., Wilkins, E., Wright, L., Vos, R., Bax, J., Blum, M., Pinto, F., Vardas, P., 2018, European Society of Cardiology: Cardiovascular Disease Statistics 2017.; ESC Scientific Document Group. *European Heart Journal*, February 14, v. 39(7), p. 508-579.

[12]Andersen, P. K., 2017, Life years lost among patients with a given disease. *Statistics in Medicine*, September 30, v. 36(22), p. 3573-3582, doi: 10.1002/sim.7357. Epub 2017 June 5.

[13]Mäkinen, V., P., Forsblom, C., Thorn, L., M., Wadén, J., Kaski, K., Ala-Korpela, M., Groop, P., H., 2009, Network of vascular diseases, death and biochemical characteristics in a set of 4,197 patients with type 1 diabetes (the FinnDiane Study). *Cardiovascular Diabetology*, October 6, v.8, p. 54. doi: 10.1186/1475-2840-8-54.

When poetry reconciles science and faith, could resonant rhymes reveal your architecture of time!

Appendix 1

Example

$CMA' = 1.6\ (BP_S - BP_D) / (1 + HDL/FBG)$

$CMA' = 1.6\ (136 - 69) / (1 + 47/101)$

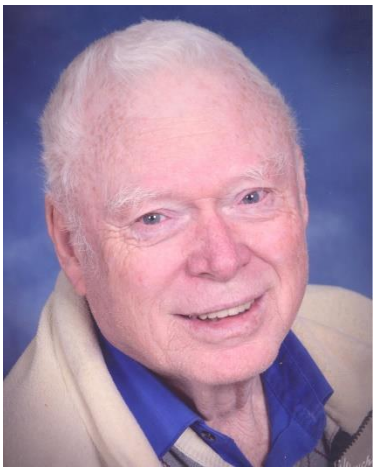
$CMA' = 1.6\ (67) / (1 + .47)$

$CMA' = 1.6\ (67 / 1.47)$

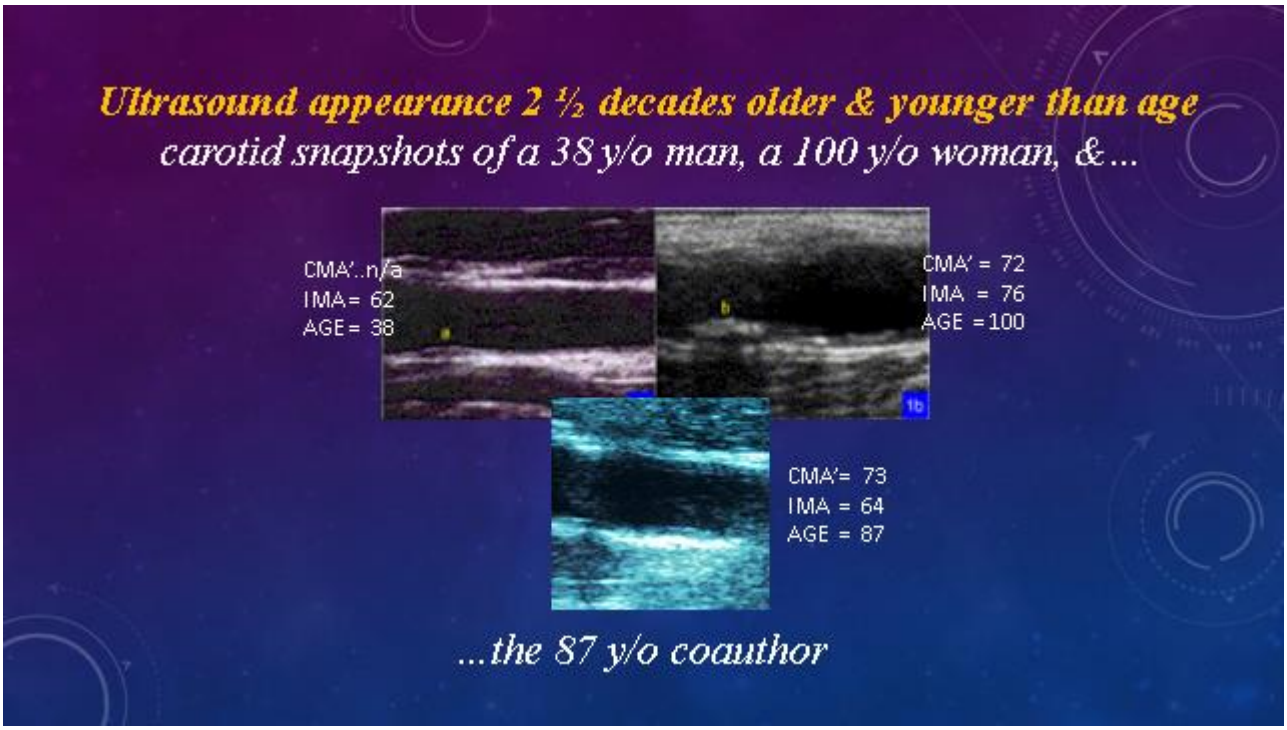
$CMA' = 1.6 \times 45.47$

Cardiometabolic age = 72.9 years

Chronological age = 87 years



Lorence G. Collins



Appendix 2. On the left is an ultrasound image of a carotid artery of a man whose chronological age is 38 years but who has plaque (linear white areas) on the wall of his artery that is equivalent for a person who is 62 years old. (Being under 40 years, the CMA' is not applicable.) On the right is an ultrasound image of an old woman whose CMA' equals 72 and whose chronological age is 100 years old but still has minimal plaque on the walls of her carotid artery that is equivalent for a person who is 76 years old. For Lorence Collins the CMA' equals 73, his chronological age is 87 years, but he has minimal plaque on the wall of his carotid artery that is equivalent for a person who is 64 years old. See **Appendix 1** for calculation of CMA' of the coauthor.