

# THE AFIB REPORT

*Your Premier Information Resource for Lone Atrial Fibrillation!*

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**16<sup>th</sup> YEAR**



*Summer is certainly heating up here in Northern Arizona and so are plans for starting the long anticipated afibbers website revamp. Not long after each of you receive this issue of the newsletter we will be embarking on the process to modernize the website, and it is our aim while transforming the underlying code to a thoroughly modern up-to-date technology, to make it even more user-friendly and accessible to a wider audience in the process. There are many now who might not be able to find us with the current limitations of our search engine recognition (SEO) capabilities.*

*In any event, as a one-man band, I needed to reach out for real expertise in this area and, thankfully, one of our own frequent forum members and SEO expert, Travis Van Slooten, has kindly thrown his hat into the ring to help steer the technical details of the reconstruction effort.*

*Trying to juggle the demands of the AFIB Report and the website redesign at the same time remains an unknown challenge at this point. Though every effort will be made to get all six issues out this year, please keep in mind that exact times and dates for publication may vary, and the primary priority the next few months will be completion and posting of the website make over. Once the website work is complete, we envision being able to morph much of the newsletter content into an easier to digest online blog and multimedia format, with news updates and more concise study summaries with links sent to each subscriber as we post them. Those are the goals, and we look forward to completing this long overdue task as soon as is practical*

*Obviously, this work will cost a fair amount of money as well, and while one very generous donation has already been received, we would appreciate and value any additional donations that any of our readers might wish to contribute via our donation link here: <http://www.afibbers.org/donations.htm>. This website/newsletter resource is for us all, and we thank those of you who are able, for any assistance that is comfortable to make.*

*This summer issue leads off with a generous reprint authorized by world-renowned natural medicine MD from Harvard Medical School, Dr. Jonathan V. Wright, from his own 'Nutrition and Healing' newsletter on the timely topic: 'Are you getting too much of a good thing with your Magnesium intake?' Most afibbers can use more, not less, magnesium in their daily diet, but for a certain number of us the more typical recommended dosage can prove counter-productive and Dr. Wright kindly shares with us just how to recognize if, and when, that might be the case and what to do about it.*

*Our next topic is from an editorial by Drs. T. Jared Bunch and John Day of the famed Intermountain Medical Center in Utah with an important finding copasetic with our own philosophy here of marshaling all treatment modalities that are most promising for control of AFIB, and bring them to bare earlier, rather than later, in the disease process. This conclusion, from their look at what might be a new metric defining the possible 'tipping point' of remodeling progression, beyond which any further reverse remodeling might not be possible even after regaining NSR. Hence, their strong urging not to procrastinate too long in one's efforts to restore long-term NSR by all the best means at our disposal.*

*Several follow up research papers from around the world have now reinforced the seminal paper from Texas Cardiac Arrhythmia Institute from 2012 that proposed four distinct shapes comprising Left Atrial Appendage Morphology and its relationship to stroke risk. A new editorial from Drs. Jorge Romero et al. from UCLA Medical Center, Montefiore Medical Center and Texas Cardiac Arrhythmia Institute, further*

refine and simplify the LAA Morphology and possible stroke risk association, indicating a strong rationale for including LAA morphology assessment as an adjunctive tool to help better sort out which of those at low risk based on CHADS-VASc scores who might nevertheless still need to consider OAC therapy, in distinction from those that might never need begin, or can safely stop, OAC drugs.

Our last study summary is a 'Short Take' update that revisits a key issue we have looked at before over the past two years highlighting new insights into AFIB's relationship with obesity, indicating that the association between AFIB and obesity is not just that the two conditions share mutual co-morbidities, but rather that obesity itself is associated with a distinct AFIB atrial substrate. Thus, indicating a more direct potential impact on the development of AFIB.

We wrap up this summer issue of the newsletter with a **Part 2** installment of the '**Dietary Enlightenment**' series by our own Jackie Burgess, in which Jackie gives a detailed look at many of the complex issues and controversies surrounding GMO's (Genetically Modified Organisms) that seem to be with us to stay now in growing sectors of our food supply. Thank you Jackie for sharing this information.

Regardless of the various opinions folks may have about GMOs, being well informed on all sides of such a topic is a good thing. I also feel that most of us will agree that it makes little sense to not at least require accurate labeling of GMO-containing food products. Strong efforts are being made in some circles now to remove GMO labeling from our foods.

**Wishing plenty of NSR for All!**  
**Shannon**

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## **Magnesium: Are you getting too much of a good thing?** **Nutrition and Healing - 2014 Special edition newsletter -** **Dr. Jonathan V. Wright**

As an introduction to this timely piece, we owe a debit of gratitude to world-renowned natural and nutritional-based medicine MD from Harvard Medical School, Dr. Jonathan V. Wright who is director of the large Tahoma Clinic in the Seattle area of Washington, and whom I've had the pleasure to get to know at a number of Bio-identical Hormone Replacement Therapy (BHRT) Conferences I have worked at over the past number of years.

Dr. Wright is well aware of our website and *The AFIB Report* and supports our focus on sharing insights into combining the best of both natural and allopathic methodologies, and thus, offering a truly integrative approach toward AFIB care. Over the years, I've come to much admire and respect Dr. Wright's passion, dedication and many contributions toward better overall health care, which often starts by identifying and replacing what is missing in endogenous human physiology, biochemistry and nutritional status as expressed by various disease states.

Dr. Wright was one of the early pioneers in addressing vital nutrient, mineral and bio-identical hormone repletion as keys to long term health, including his ongoing efforts to highlight the importance in insuring adequate magnesium levels as a significant component for proper functioning of well over 300 core biochemical reactions in the body.

As such, and due also to magnesium's essential role in aiding heart rhythm function, it's only apropos that we turn here to Dr. Wright to learn how to recognize when a select number among us are taking too much supplemental oral magnesium for our own good. Thus, without further adieu, let's enjoy this reprint from a special edition of Dr. Wright's own popular newsletter, **Nutrition and Healing**, on the topic of '**Magnesium: Are you getting too much of a good thing?**'

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## Clinical Tips #154

Magnesium is an essential nutrient with hundreds of known functions in the body. From maintaining and improving energy levels to building bone density, to treating migraines - there are numerous health benefits to supplementing with this nutrient. However, there is a hidden health hazard that most people don't know about.

The most commonly recommended dosage for (oral) magnesium supplements is 200mg to 600mg and occasionally as high as 1,000mg a day. For most of us, 200mg to 600mg is sufficient. But there's a possible hidden side effect that can occur – and it can happen even at lower doses.

### Magnesium-induced Magnesium Deficiency

In the late 1970s, I worked with a professor of chemistry from a major university. His knowledge and use of vitamins and minerals was well ahead of the university's medical school. When he first arrived, he brought with him an excellent diet plan and for the time, an exceptionally comprehensive list of vitamins and mineral supplements. He'd been following the diet, exercising, and taking the supplements for several years, but still complained that he was "all tired out!"

So we did some tests. Surprisingly, nearly all his mineral levels were lower than usual. Low, even compared to individuals who had not been supplementing with minerals. Some of his other nutrient levels were also lower than expected. After several negative tests for more-usual causes of nutrient mal-absorption (low stomach acid, insufficient digestive enzymes, allergy) we discovered that his 500 milligrams-a-day dose of magnesium was the culprit.

### You May Not Have Liked Milk of Magnesia ... This Could Be Why

Remember Milk of Magnesia? If you ever took it as a child, it's probably not a fond memory! The large quantities of magnesium in this product are quite irritating to the bowel and will usually make you "go". Some of us are much more sensitive to this side effect of magnesium than others. The professor was one of the sensitive ones. But as he observed: "I don't have diarrhea or even loose bowels!"

There's a prototypically British term that describes the professor's situation: '**Gastrointestinal Hurry**'. The term was coined by my friend and colleague, Dr. Stephen Davies, then of London, now of Queensland, Australia. By this he meant a sufficient speeding-up of the bowel to adversely affect nutrient absorption, but not enough to really be noticeable. To document gastrointestinal hurry, he had individuals measure their intestinal transit time while taking varying quantities of magnesium ... and then measure again when not taking the magnesium doses.

Intestinal transit time, although not mentioned in the index of two major textbooks of gastroenterology I just checked, is generally understood to be the term that describes the length of time food takes to "transit" from the entrance to the exit of the gastrointestinal tract. Although estimates vary, a reasonable range for 'normal' transit time appears to vary from a minimum of 12-14 hours to 20-24 hours. Transit time is easily measured by eating beets or corn or swallowing charcoal tablets and observing how long it takes these or other markers to emerge.

The chemistry professor found that his transit time was approximately 18 hours without his usual 500 milligrams-a-day dose of magnesium. With the magnesium, his transit time dropped to 9 to 10 hours without loose bowels; clearly a case of '*gastrointestinal hurry*'. He had a classic case of magnesium-induced magnesium (and other nutrient) deficiency. He later found that the largest amount of magnesium he could take each day without causing this 'hurry' was 250 milligrams.

Most of us can take more magnesium each day without risking gastrointestinal hurry or magnesium-induced magnesium deficiency. However, it's easy enough to discover if your magnesium supplement is

causing 'hurry'. I'd recommend checking, especially if your supplements contain more than a total of 500 to 600 milligrams of magnesium daily.

**Excerpted with permission from "Nutrition & Healing Newsletter" ([www.wrightnewsletter.com](http://www.wrightnewsletter.com)), by Dr. Jonathan V. Wright, Medical Director of Tahoma Clinic in Tukwila, Washington,**

**Editors comments:** Once again, I want to express our appreciation to Dr Wright for allowing us to reprint this important, and until recently, little understood and discussed caveat with regard to magnesium dosing for select individuals. Granted, the majority of afibbers seem to require more, rather than less magnesium intake to achieve more optimal intracellular magnesium sufficiency.

And yet this less common, but certainly not unheard of anomaly in magnesium dose requirement for some individuals certainly applies to some afibbers as well. Thus, anyone who is embarking on a dedicated long-term magnesium repletion protocol for overall better health, and specifically in an effort to help quiet a jumpy heart, should be aware of the signs and symptoms of this 'gastrointestinal hurry' leading possibly to magnesium-induced deficiency of not only magnesium itself, but possibly other vital nutrients as well.

And keep in mind that this form of 'hurry' is not revealed by bowel intolerance, but rather the more subtle measures of a somewhat reduced gastrointestinal transit time that can result in less assimilation and storage of key nutrients from our diet and supplements over time. This can lead to a whole host of symptoms ranging from fatigue and tiredness to depression and many more such manifestations.

The take home message is that it is no longer enough to just use shy of bowel intolerance as the main marker for having achieved a healthy daily level of magnesium dosing. Especially those who may be feeling worse, rather than better, some months after starting a magnesium dosing program, or 'The Strategy' protocol, should test themselves for the possibility of needing less (if so, try around 250mg/day) rather than the more typical 500mg to 600mg/day of magnesium intake normally recommended.

## **Adverse Remodeling of The Left Atrium (LA) in Patients with AFIB: When is the Tipping Point in Which Structural Changes Become Permanent? (Editorial Comment - Journal of Cardiovascular EP)**

SALT LAKE CITY, UTAH. More insights from Dr. T. Jared Bunch and Director Dr. John Day, of the large AFIB research center at Intermountain Medical Center Heart Institute in Utah, whose very active investigations we have featured quite a few times in recent years in *The AFIB Report*. They now share an editorial comment I found particularly relevant for our group, and indeed for all afibbers. The topic is: when is it too late to bring about successful reverse atrial remodeling of an existing fibrotic substrate?

In the intro of this editorial, these two leading EPs noted: "A tipping point is a critical point in an ongoing event or situation that leads to a new irreversible change". "Understanding a tipping point at which the effect of disease processes that provoke AFIB result in permanent irreversible substrate changes to the atrium is of critical importance." "If lifestyle (or other natural [editor]) changes and pharmacologic therapies are to be used, they need to be introduced well *before* the tipping point is reached to allow maximal benefit, and in some cases reverse the natural history of AFIB."

That description of what a tipping point is, and the implication of knowing how, and when, to intervene with both appropriate and timely therapies, underscores the key philosophy and goal of our newsletter and website's educational and patient support efforts to a 'T'. And that is; finding the best collective methods possible to put the genie back in the bottle for good!

While the precise tipping point of irreversible change to the atrium is not known, in general, therapeutic interventions of all kinds for AFIB tend to work better when begun earlier, rather than later, in the natural history of progression of this challenging and frustrating condition. The authors noted that within their large medical system they can measure deleterious changes in outcomes after AFIB ablation with delays in achieving sustained NSR of as little as 6 months from the initial diagnosis of AFIB!<sup>1</sup>

The authors also affirm what has been reported elsewhere as well, that structural atrial reverse remodeling is indeed possible when the effort begins early enough, and led by restoration of NSR. However, it appears that there is a definite limit beyond which any reversal of unwanted atrial structural remodeling in the form of fibrosis and scarring is very unlikely to happen, even after re-establishing long term sinus rhythm. Thus, a key issue is determining where that tipping point is, and most importantly beginning any, and all, appropriate therapies as early in this progressive process as possible after diagnosis ... within reason.

Such research findings underscore our own long 16 year anecdotal experience with afibbers from all over the world via our forum and this newsletter, that procrastinating too long in adopting all the best treatment options when the need for each arises, is counterproductive in helping to restore normal sinus rhythm (NSR) as expeditiously and persistently as possible.

Left atrial remodeling is the adaptation of cardiac myocytes over time due to exposure to stressors such as tachycardia and/or volume/pressure overload. Electrical remodeling, which typically occurs first, can appear as early as 30 minutes after exposure to rate-related stress and tend to reverse almost immediately once the tachycardia stress is removed.

Structural remodeling changes occur relatively quickly too, though more slowly than electrical changes. Slow zones of conduction and fibrosis start to occur at around five weeks of AFIB within the left atrium, and as AFIB stress exposure continues, atrial collagen synthesis and degradation increase with dynamic remodeling including hypertrophy of atrial myocytes in an effort to adapt to the added AFIB stress burden, with the net result being added scar tissue and functional stiffening of the LA.

The authors noted as well that as this adaptive process starts to fail, progressive dilatation or expansion of the LA occurs along with thinning of the atrial wall and fibrosis as well as patches of focal and regional cellular apoptosis or death increase.

These progressive processes, and various methods for possibly minimizing or reversing them, are factors we have explored for years on our forum and in our various *'conference room'* focused topics.

We still don't know for sure where the magic tipping point lies, after which there is little chance of return to a more healthy heart via extensive reverse remodeling. Nevertheless, the authors emphasize that what does seem clear is that any possible reversible remodeling likely involves changing the mechanisms of reactive fibrosis that respond to inflammation, biochemical stresses and hormonal stimulation by increasing perivascular and interstitial collagen deposition.

And it's equally apparent from all that we have learned to-date, that in spite of both MRI-based and mechanistic cellular data, each showing that reverse remodeling is possible after restoring NSR, it is also very apparent that some degree of fibrosis will persist even in the setting of extensive reverse remodeling, and thus provides a longstanding substrate towards further arrhythmia genesis and maintenance.

In other words, the longer we allow our hearts to rock and roll, even with what may seem to be relatively asymptomatic AFIB, or other atrial arrhythmias, the odds of being able to make a later stage significant reverse remodeling of long standing atrial structural changes become increasingly dubious. Once again,

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<sup>1</sup> Bunch TJ, et al: Increasing time between first diagnosis of AFIB and CA adversely effects long-term outcomes. Heart Rhythm 2013;10:1257-1262.

the evidence veritably shouts to us ... 'don't beat around the bush too long when it comes to doing everything in our power to re-establish consistent NSR'!

### **A tipping point clue...?**

We still need more studies further clarifying our understanding of AFIB-related atrial remodeling in order to most accurately determine by what time, and by which therapies, we might best avoid or minimize the long-term deleterious impact of this lousy disease.

Nevertheless, progress is being made and in a study by Takahashi et al,<sup>2</sup> in the same 2015 issue of the Journal of Cardiac Electrophysiology, in which also appeared this editorial comment by Drs. Bunch and Day, this Japanese team investigating the impact of atrial remodeling on pulmonary vein (PV) ostium and left atrial substrate, and how that might impact ablation procedures that require long-term PV isolation uncovered what seems to be a interesting piece of this puzzle.

In studying 75 subjects (50 of whom underwent AFIB ablation) using a 320-slice CT scan, the average increase in LA thickness of measured segments was around 13% greater in the AFIB patients compared to those without AFIB. And this atrial thickening in the afibbers reflects likely atrial myocyte hypertrophy (expanded growth), intracellular fibrosis and inflammation/edema.

Not surprisingly, this atrial thickening was greatest in persistent AFIB patients compared to the paroxysmal AFIB group, with this finding being reflective of progressive remodeling of the disease due to ongoing atrial tachyarrhythmia. In addition to AFIB/tachycardia as a driver of this progressive remodeling, hypertension was the one demographic variable that was most associated with left atrial thickness due to exposing the LA to chronic pressure overload stress.

What these Japanese researches found was that this LA thickening was present with all degrees of LA enlargement or dilatation. Next, as fibrosis continued to develop and became more severe, left atrial thickening interestingly decreased! This finding strongly suggests that the process of adaptive atrial thickening that occurs in the earlier stages of remodeling as the heart tries to adapt to AFIB stresses, and often hypertension pressure overload stress as well, eventually starts to fail and this loss of adaptive thickening does seem to represent the tipping point after which no reverse remodeling is likely, or even possible, beyond this point of progression.

The bottom line here, is that 'time equals atrial tissue' when it comes to initiating and following through with effective therapies for AFIB. And thus, it's clear that treatment can never get started too early and, indeed, many of the stressors that contribute to atrial remodeling and that, in turn, leads to more AFIB genesis often start early in life as well.

In conclusion, the two authors of this timely editorial stated that as EPs, they typically see patients only well after they have developed AFIB and after various degrees of structural disease progression have occurred. In these patients, life-style changes can still make a real difference as many can yet benefit from reverse remodeling to a significant degree. However, they underscored once again that ... "when rhythm control strategies are adopted, they should be started as early as practical in the disease process to minimize total exposure and accumulation of AFIB burden on the heart. This new CT analysis of the atrium provides added understanding of the disease process and gives a metric that can be considered a measure of substrate reversibility reserve".

***Bunch, TJ and Day, J. Jour. of Cardiovascular EP, Editorial Comment. 06/2015 Vol. 26.pp 606-607***

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<sup>2</sup> Takahashi K, et al, Relation between left atrial wall thickness in patients with atrial fibrillation and intracardiac characteristics & ATP-provoked dormant pulmonary vein conduction. J. Cardiovascular Electrophysiology 2009;20:461-465.

**Editor's Comment:** The central message that has evolved since the inception of *The AFIB Report* has increasingly reflected this same fundamental recognition and recommendation now shared about addressing AFIB treatment options in this new editorial from Drs. Bunch and Day.

In the early to middle years of our forum and newsletter investigation efforts, the discussion was often focused on avoiding any invasive treatment as long as possible by adopting whatever combination of natural therapies along with anti-arrhythmic or rate control drugs was deemed necessary, in an effort to beat back AFIB sufficiently enough to at least lessen its presence in our lives to a significant degree.

But as ablation technology, understanding and skill, especially among the most experienced operators, continued to mature and our understanding of the underlying disease process, if not yet the core origins, also continued to evolve, we have expanded our focus as well to include a truly more holistic approach in which all of the best tools available to us are explored and supported in the proper sequence of escalation and collaboration of modalities.

These include key life-style and cardiac risk factor reducing methods along with dietary, supplemental, and stress reduction improvements and all adopted as early as possible after diagnosis. And all combined with appropriate exercise, plus including an expert ablation process when, and if, necessary.

The important message echoed by this Utah editorial is that the earlier each phase of these treatment options are adopted the better. We recommend all afibbers adopt this truly integrative approach and add each modality at the appropriate time in their individual journey toward restoring long term NSR as effectively and quickly as possible.

No longer is avoiding an ablation at all cost a primary goal for the wise afibber. Our overall recommended pathway, when followed with dedication, insures that those who are able to quiet their AFIB for the long term sufficiently well via natural and life-style risk factor reducing and/or pharmacological methods, will find that out fairly early in the process. Much sooner now the savvy afibber can determine when, and if, they will need to include a more invasive ablation step as the best remaining option. From our long collective experience, the inclusion of each of these best options at the right time will result in the best overall odds of achieving solid success as early and reliably as possible.

'Time truly is atrial tissue', as the authors noted, and the sooner all of these key methods are considered and adopted in sequence, and as needed, the earlier and more robust will be our individual success at restoring lasting NSR; that most important goal worth achieving.

## **Left Atrial Appendage (LAA) Morphology and Physiology: "The Missing Piece of the Puzzle"**

Bronx, NY. Los Angeles, CA. Austin, TX. In 2012, an important study of research proposing a relationship between four distinct LAA morphological shapes and an associated risk of stroke or TIA in afibbers was first published by DiBiase, et al, at Texas Cardiac Arrhythmia Institute.

In a most recent in-depth editorial review of all subsequent studies examining this relationship from an upcoming issue in the *Journal of Cardiovascular Electrophysiology*, Drs. Jorge Romero, et al, propose considering adding LAA Morphological classification as an adjunctive measure to the current CHA<sub>2</sub>DS<sub>2</sub>-VASc stroke risk score system, and particularly for lower risk patients in whom the currently accepted stroke/TIA risk scores of 0 for men and 1 for women leave some ambiguity as to which patients with this low risk scores can safely forgo therapy with oral anticoagulation in given circumstances, from those who might yet warrant treatment when LAA morphology indicates a higher risk for potential embolic events, even with all other cardiovascular risks accounted for and those risk scores remaining low.

The originally proposed LAA morphological classification includes 4 overall shapes; with Chicken Wing being both the most prevalent from the various study groups investigated (found in approx. ... 50% of

AFIB patients), and the most benign in terms of associated embolic risk. That leaves Windssock, Cactus and Cauliflower as the final three LAA shape classifications with a respectively increasing risk of stroke or TIA.

However, recent research has clarified that for all practical purposes, the dividing line between a safely low effective ischemic risk, and a possible increased need to consider OAC (oral anticoagulation) therapy, even with those who have low scores on traditional CHA2DS2-VASc risk factors, is between Chicken Wing on the benign side and Non-Chicken Wing on the heightened risk side of the coin.

The current stroke risk system, first adopted as the new defacto standard in Europe in 2012, and in the US in 2014, has been heralded as a universal improvement over the prior CHADS2 system.

Nevertheless, it is not a perfect system and still strokes happen even in AFIB patients with low CHA2DS2-VASc risk scores. In fact, in a recent large Taiwanese study, Chao, et al,<sup>3</sup> evaluating AFIB patients including only those males with a risk score of 1 and females with a score of 2 who were not on OAC or anti-platelet drugs, among 12,935 male AFIB patients with a risk score of 1, a total of 1,858 (14.4%) of those men experienced ischemic stroke during a follow-up of 5.2 years, for an annual stroke rate of 2.75%.

For female afibbers and a risk score of 2, 14.9% had an ischemic stroke with an annual rate of 2.55%. Ischemic stroke risk increased in this study from 1.91% for women with hypertension to 3.34% for those 65 to 74 years of age. It is remarkable, indeed, that men and women afibbers from such a large cohort with a CHA2DS2-VASc net effective risk score of 1 (excluding sex as a risk factor) had an annual stroke rate of 2.75% and 2.55%, respectively! This rate is significantly higher than risk of intracranial hemorrhage (0.23% to 0.5%) reported in trials of the new NOAC (novel oral anticoagulant) drugs.

Even a CHA2DS2-VASc score of 0 is no promise of an ischemic stroke free survival for afibbers, contrary to what the original paper on the new risk factor standard implied. In a paper by Kimura, et al<sup>4</sup> the riskiest LAA morphology of 'Cauliflower' was found more commonly in patients with stroke and low CHADS2 score. And 26.7% of the patients included in this analysis who had experienced a prior ischemic stroke, had a CHA2DS2-VASc score of 0.

In the past, and even today, many cardiologist no doubt felt that these patients in these low risk ranges were well protected just on aspirin, something we now know is not true. The point being, that while the recognized stroke risk factor scores have mostly been used in clinical practice for initiating OAC therapy, they are mostly focused on well-known risk factors for ischemic stroke and none of these risk factors include or assess the LAA itself where the vast majority of AFIB related thrombus are formed.

Thus, the results reported in this same issue of the Journal of Cardiovascular Electrophysiology by, Lee et al<sup>5</sup>, are important and clinically relevant, especially for patients with AFIB considered to be low risk of ischemic events based on CHADS2 or CHA2DS2-VAS ranging from 0 to 2. In this class of 'low risk' afibbers, the authors report that ... "the presence of a Non-Chicken Wing LAA morphology dramatically increases the risk of thromboembolic events".

With roughly 50% of the AFIB population having a more benign Chicken Wing LAA shape, possibly at least 12% of afibbers with an otherwise low CHA2DS2-VASc score might benefit from further stratification by assessing LAA morphology when making a well thought-out OAC decision in these patients, while always balancing the risks of ischemic stroke with bleeding risks.

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<sup>3</sup> Chao TF, et al. *Should atrial fibrillation patients with 1 additional risk factor of the CHA2DS2-VASc score (beyond sex) receive oral anticoagulation?* JACC. 2015;65:635-42.

<sup>4</sup> Kimura T, et al. *Anatomical characteristics of left atrial appendage in cardiogenic stroke and low CHADS2 scores.* Heart Rhythm Journal. 2013;10:921-5.

<sup>5</sup> Lee JM, et al. *Why is Left Atrial Appendage Morphology Related to Strokes? – An Analysis of the Flow Velocity and Orifice Size of the LAA.* J Cardiovasc Electrophysiology. 2015;26: <http://dx.doi.org/10.1111/jce.12710> .



One golden opportunity to perform such an LAA shape assessment is during work up for catheter ablation where either a pre-ablation CT scan, MRI or perhaps 3D-TEE could be used to kill two birds with one stone and gather LAA morphological data while collecting the pre-ablation imaging needed.

**Romero, J. et al. Left Atrial Appendage Morphology and Physiology: 'The Missing Piece of the Puzzle'. J. Cardiovascular EP. 2015 <http://dx.doi.org/10.1111/jce.12746> .**

**Editors Comment:** The bottom-line take home message from this interesting evolution of our understanding of LAA morphology and its association with stroke risk, is that LAA shape-related stroke risk is clearly divided now between the relatively more benign Chicken Wing morphology and all Non-Chicken Wing shapes that collectively carry a significantly higher risk of stroke, and thus should be considered as an adjunctive factor added to overall stroke risk factor decisions in active afibbers. This is especially true for those with low CHA<sub>2</sub>DS<sub>2</sub>-VASc scores of 0 to 1 in which some ambiguity might remain when deciding for, or against, initiating or continuing OAC therapy.

Patients with Chicken Wing LAA show significantly smaller LAA volume and faster LAA flow velocity compared to those with Non-Chicken Wing LAA. Thus, Chicken Wing LAA's with little to no pectinate trabeculations within the LAA ... which itself is an added independent risk factor for stroke ... is key to identify those afibbers with lower risks for stroke.

To be sure, large additional randomized studies further defining and confirming this relationship will have to be done before formal approval and adoption of this new potential 'missing piece of the stroke scoring puzzle' sees wider use. But savvy afibbers, such as our readers, who happen to fit this bill and may have a low risk score, but wish to insure assuming the lowest possible risk for stroke, can now add LAA morphological identification to their stroke evaluation deliberation with their EP or Cardiologist. And those with clearly a higher risk of stroke due to having one of the riskier Non-Chicken Wing LAA shapes, can perhaps now make better and safer all around decisions for themselves be it for deciding for, or against, OAC therapy.

## **Short Take**

### **Atrial Fibrillation and Obesity**

PARIS, FRANCE. Yet another interesting and timely editorial, this time from Dr. Stephane Hatem MD PhD, at the Institute of Cardio-metabolism and Nutrition, Dept. of Cardiology, Assistance Publique-Hopitaux de Paris, on the relationship between obesity and AFIB. As a central, fully addressable life-style risk factor for AFIB, obesity has been shown conclusively in recent years not only to be generally associated with this arrhythmia, but in recent issues of this newsletter we've seen how directly attacking obesity by supervised aggressive weight loss programs can have a huge impact on reducing over all AFIB burden for over-weight afibbers, often in dramatic fashion as long as the weight loss not fluctuate.

Reporting on the results of another study by Mahajan, et al.<sup>6</sup> addressing electrophysiological and structural remodeling of the atria due to sustained obesity, Dr Hatem underscores that obesity is a systemic condition characterized by low-grade inflammation, increased renin-angiotensin-aldosterone-system expression and endothelin pathway activity, as well as higher levels of transforming growth factor (TGF)- $\beta$ 1, that may be a precursor for developing atrial fibrosis.

"These factors are coupled to signaling pathways involved in hypertrophy and fibrosis of the myocardium, and therefore support the possibility that obesity could be another distinct independent risk factor for AFIB", noted Dr Hatem. How this connection may work is complex, but appears to be the result of fatty infiltration of the abundant adipose tissue contiguous to the epicardial (on the outside of the

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<sup>6</sup> Mahajan R, et al. Electrophysiological, electromechanical and structural remodeling of the atria as consequences of sustained obesity. JACC 2015;66:1-11

heart) adipose tissue, into the atrial myocardium. Envision streaks of interstitial fatty tissue invading and spreading in between otherwise contiguous, consecutive atrial muscle cells. Which in turn, can result in less stable electrical conduction from one myocyte to another that then can disrupt stable heart rhythm conduction through normally would have been a uniform atrial myocardium tissue layer.

Indeed, the authors of the study in question found that this fatty infiltration from the epicardial area of the heart predominates in the posterior wall of the left atrium, at and near, the junction of the pulmonary veins and thus generates low and heterogeneous voltages that can lead to arrhythmogenic substrate around the perimeter of these low voltage areas. This fatty infiltration can even result in conduction block and depressed excitability and, in addition, a milieu of inflammatory cytokines, chemokines, and growth factors can then freely diffuse into the adjacent myocardium and modify its structure and functional characteristics. Thus, leading to more proliferation of structural atrial fibrotic changes that help sustain the arrhythmogenic substrate.

Two important confirmations result from this observation that obesity can play a more direct roll in contributing to AFIB genesis. First, improvement in cardiac imaging will help improve detection of myocardial fat deposits in both obese and metabolic syndrome patients. Secondly, aggressive weight loss programs for over-weight afibbers can have a very beneficial effect on atrial remodeling ... so long as the person has not waited so long to get serious about weight loss that they have crossed the tipping point beyond which little to no reverse remodeling is likely to occur at all.

This scenario only further underlines our well-honed message too, to not procrastinate in doing everything in our power to improve our own health and diets, not only for the more general overall physical health benefits, but to directly impact the course of one's AFIB burden and history in a very positive manner.

**Hatem, S. 'Atrial Fibrillation and Obesity: Not Just a Coincidence' JACC 2015:**  
<http://dx.doi.org/10.1016/j.jacc.2015.04.057>

**Editors comment:** Not much to add to this common sense, but nonetheless, important confirmation that AFIB and obesity, as well as metabolic syndrome and the constellation of life-style risk factors, have a more direct bearing on the course of our AFIB than we once may have thought. This underlying message is no surprise to our readers, once again, that the sooner we grab the bull by the horns and do everything in our power to address the very changeable factors in our lives that may be contributing to, if not causing our arrhythmia, makes all the sense in the world. We may not all be fully successful in taming AFIB via our efforts, but dedicating ourselves to better overall health can only help in our cause even when an expert ablation may also be required in the case of more advanced disease.

## **Dietary Enlightenment - Part 2**

### **GMO (Genetically Modified Organism) Facts**

***We can easily forgive a child who is afraid of the dark; the real tragedy of life is when adults are afraid of the light ... Plato***

The GMO topic is broadly complex and multi-faceted. This overview covers important, basic facts. Hopefully, motivated readers will continue independent research for benefit of personal health and that of family, pets, farm animals and the environment. It's maybe **that** critical. Almost daily, new findings are reported. As always, my mission is to create awareness, so this is an alert for those who really want to know about GMO's – the risks, the facts, the science.

Healthy regards, *Jackie Burgess*

## Introduction

Adverse health conditions can result from GMO food consumption. This could be relevant to afibbers because of the direct impact on the gastrointestinal (GI) tract since GI issues are frequently reported as part of a prelude to an AFIB event. Also noted, are connections to immune activation and allergies. Since 70 percent of our immune system function either resides in, or depends on, the gut and its optimal functioning to expel and kill foreign invaders, it makes sense not only to be concerned, but highly aware. Children may be the most vulnerable to the potential dangers of GMOs. In some studies test animals and farm animals show significant consequences of consuming GM foods.

*"Since so little research has been done on the safety of GM foods, it is not possible to rank its risks. Unlike the others, GM crops persist in the environment and may continue to pose risks to health for centuries. In addition, transfer of transgenes to gut bacteria may present long-term chronic exposure since the foreign protein may continue to be produced inside our bodies after we no longer consume the GM food."*<sup>[1]</sup>

A deeper scientific understanding about the genetic implications of these modified plants and foods is addressed in the book by Professor of Medicine, Cell Biologist, Epigeneticist Bruce H. Lipton PhD, *The Biology of Belief* (2007).

*"Epigenetics is the science of how environmental signals select, modify and regulate gene activity. This new awareness reveals that our genes are constantly being remodeled in response to life experiences... which again emphasizes that our perceptions of life shape our biology."*

*"Already there is a study that shows that when humans digest genetically modified foods, the artificially-created genes transfer into and alter the character of the beneficial bacteria in the intestine. (Heritage 2004; Netherwood, et al, 2004) Similarly, gene transfer among genetically-engineered agricultural crops and surrounding native species has given rise to highly-resistant species deemed superweeds."*

*"Now that we are aware of this inter- and intra-species gene transfer mechanism, the dangers of genetic engineering become apparent. For example, tinkering with the genes of a tomato may not stop at that tomato, but could alter the entire biosphere in ways that we cannot foresee."*

*"When we eat food, the micro-RNA from the food is picked up by the digestive system and not broken down. The micro-RNA is taken into our own body intact, and now what they've found is that the micro-RNA from food ends up in our own cells - like in our liver and other cells in our body - and these micro-RNAs still have the same function: they change our genetics and they change the readout of our genome. And the significance is profound: it says when we eat genetically modified food we're eating a new class of micro-RNAs that have never been in the world before, and yet these micro-RNAs are picked up by our biology and adjust our own genetics, so in a sense the old story "we are what we eat" actually now has a biochemical foundation"*

Jeffrey M. Smith is the primary information source quoted in this report because of his vast experience and credibility. Jeffrey is a highly-respected and well-known leading researcher and consumer advocate, author of *Seeds of Deception: Exposing Industry and Government Lies About the Safety of the Genetically Engineered Foods You're Eating* and the award-winning film, *Genetic Roulette: The Documented Health Risks of Genetically Engineered Foods*.

Jeffrey counsels leaders from every continent, campaigned to end the use of genetically-engineered bovine growth hormone, influenced the first state laws in the United States regulating GMOs and is an admired keynote speaker around the globe. He has lectured in nearly 40 countries and has been quoted by world leaders and hundreds of media outlets. He is also a popular guest on influential radio and television programs.

Former National Institutes of Health neuroscientist, the late Candace Pert, described Jeffrey as "the leading world expert in the understanding and communication of the health issues surrounding genetically modified foods."

There are many other credible sources confirming the observations and science. For background info on GMOs, I suggest starting with Jeff's talk, "*Don't Put That in Your Mouth*" from 2007.

<http://tinyurl.com/ovb439s> Much more has occurred since. IRT's website blog offers news on current developments, the latest on GM labeling laws and trade agreement conflicts that impact US consumers. <http://tinyurl.com/kpmdkvb>

Those who consume exclusively fresh or frozen certified organic foods and beverages, choose organic supplements and enjoy organic garden produce from certified organic seeds will find this report reinforcing of their good habits. For adults and their children who do not eat "unprocessed organic" and eat meals away from home, eat packaged foods and don't read labels, let this serve as an awareness alert on the potential health risks of consuming Genetically Modified Organisms (GMOs) or Genetically Engineered foods (GE).

### **So what exactly are GMO's?**

Clarification: **hybridized** is not the same as GMO. Wheat is hybridized or bred (by sexual reproduction techniques) to enhance various desirable qualities such as wheat bred to enhance traits that make bread products lighter, fluffier, use less flour, taste better, have different mouth-feel or for farmers who want a wheat that has higher yield per acre or is more drought tolerant.

A GMO is the result of a laboratory process where genes from the DNA of one species are extracted and artificially forced into the genes of an unrelated plant or animal. The foreign genes may come from bacteria, viruses, insects, animals or even humans. Because this involves the transfer of genes, GMOs are also known as "transgenic" organisms. Genetic Engineering (GE) or Genetic Modification (GM) is the same process and works in several ways to cause harm:

1. **Seeds** are treated with the herbicide glyphosate, aka Roundup by Monsanto, so that when the plants grow, they are genetically engineered not to die when sprayed with Roundup herbicide; thus, are called "Roundup Ready" (RR) seeds and include soy, corn, cotton, canola, sugar beets, Hawaiian papaya, alfalfa and small squash - yellow crookneck and zucchini. This means that plants of RR seeds store a portion of Roundup in the food portion we (or animals) eat which means we get a dose of Monsanto's herbicide with every bite.<sup>[1]</sup> Newly approved are the GM non-browning apples and non-bruising potatoes. Now, Monsanto wants to introduce Roundup-ready wheat. Many more are coming.

A [report](#) published March 20, 2015, *The Lancet Oncology* medical journal, the International Agency for Research on Cancer (IARC; Lyon, France) announced findings after a meeting of 17 oncology experts from 11 countries met to review the available scientific research exploring the connection between the organophosphate pesticides, glyphosate, tetrachlorvinphos, parathion, malathion and diazinon and various human cancers. These assessments will be published as volume 112 of the IARC Monographs. <http://tinyurl.com/p5yk7lv>

"Consumers have the right to know how their food is grown and whether their food dollars are driving the use of a probable carcinogen." — Ken Cook, Environmental Working Group. According to IARC, glyphosate is used in more than 750 different herbicide products and during spraying has been detected in the air, in water and in food. The panel of experts concluded that "limited evidence" exists to show the herbicide can cause non-Hodgkins lymphoma in humans and additional "convincing evidence" that it can cause other forms of cancer in both rats and mice. Researchers noted that glyphosate has been found in the blood and urine of agricultural workers, showing the chemical has been absorbed by the bodies of those who work with it the most. <http://tinyurl.com/q8khefa> <http://tinyurl.com/q90ludj>

An interesting side-note on the glyphosate component in Roundup: It has broad-spectrum antibiotic properties and is highly selective as it kills the beneficial gut bacteria but not the E. coli, salmonella or botulism bacteria. This causes big bacterial imbalance (dysbiosis) problems in poultry and cattle and the epidemic of botulism because it's now found everywhere, when formerly was typically held in check by good bacteria that keep out invaders so more gut issues or complaints are being seen. Example: the good bacteria, bifidus, helps suppress inflammation so many health problems can be related to

inadequate protective (good) gut bacteria, overgrowth of bad gut bacteria, leaky gut, autoimmune disease, food allergies, autism, cancer and more.<sup>[1]</sup>

**2. Plants** are engineered by inserting *Bacillus thuringiensis* (Bt toxin) to produce their own toxic insecticide by killing insects that feed on the plant, fruit or leaves. This kills the bugs by bursting their stomachs and making holes in their intestines. The Bt toxin is a natural soil bacteria often used by organic farmers to kill insects while it's active but it also washes off and biodegrades. Corn and cotton plants (themselves) are now registered pesticides as the plants do the killing.<sup>[1]</sup> When the insect's good, normal gut bacteria get out of the stomach and into the insect's blood stream, it becomes pathogenic. Transfer of transgenes to gut bacteria may present long-term chronic exposure, since the foreign protein may continue to be produced inside of us after we no longer consume the GM food.

**3.** Aside from the GMO process, the **topical spraying** of glyphosate, Roundup's active ingredient which kills beneficial gut bacteria, is also used as an herbicide to prepare crops for harvest so is yet another source of exposure to this toxic chemical considered to be an **endocrine disruptor**. This is a particular problem now since the US Environmental Protection Agency increased the allowable residues on fruits, vegetables and grains by as much as 30 times. It's said the acceptable level now is about a million times more than the original level linked to damage from Roundup. Fruits, vegetables and grains are being sprayed to dry down crops to make harvest easy.

The spray doesn't just stay on the surface. It has a surfactant, which allows the Roundup/glyphosate to penetrate inside; and then it gets stored in the fast-growing sections of the crop. As harvest time gets closer, that's the only place it goes, to the food portion, which we eat and includes crops of wheat, rye, barley and lentils. But these aren't GMO's. Now, it's going to be used for citrus and sweet potatoes before harvesting.<sup>[1]</sup>

Back to the endocrine disruption (hormones)...glyphosate disrupts aromatase (enzyme) which is the basis for estrogen and testosterone production. In animals fed Roundup, the females had more testosterone and the males had more estrogen; an inverse of the natural balance. They are feminizing the males and virilizing the females, which over the past decade and more has been found to be a growing concern in humans at this point in terms of hormone levels.

Another big danger with Roundup is that glyphosate was originally patented as a broad-spectrum **chelator**, meaning it binds with trace minerals making them unavailable for digestion and assimilation. That means the food that's genetically engineered, Roundup-ready, is drenched with Roundup and becomes nutrient deficient.

The animals eating these RR crops become nutrient-deficient. We eat the Roundup residues. It can then further chelate within us and block the availability of certain nutrients. Trace minerals are like keys in locks to certain metabolic pathways; and many diseases are found to have associated nutrient deficiencies. So, by eating large quantities of Roundup-drenched food, we may be interfering with metabolic pathways that, of course, can affect health. It's known to interfere with calcium, cobalt, vitamin B12, manganese, **magnesium** and so on.<sup>[1]</sup>

**SAFETY CLAIMS 'Busted'...** The EPA claimed the Bt toxin is safe because it only interacts with certain insects and not with humans or mammals. But, in the *Journal of Applied Toxicology* (2012), this was overturned when they took Bt toxin from a GM corn plant, applied it to human cells and it poked small holes in the human cells causing leakage. (The author wrote: *Like in insect cells*).<sup>[1]</sup>

While the EPA and biotech industry have also said the Bt toxin is completely destroyed in the stomach and it becomes ineffective, this finding was overturned in 2011 by Sherbrooke University Hospital (Quebec, Canada) in a study using blood from pregnant women that found: *This is the first study to reveal the presence of circulating PAGMF in women with and without pregnancy, paving the way for a new field in reproductive toxicology including nutrition and utero-placental toxicities.*<sup>[2]</sup>

The Bt toxin was in the blood of 93% of the pregnant women and about two thirds of the non-pregnant women. With the pregnant women, the Bt toxin was found in fetal cord blood, leading then to speculation that with the hole-poking toxin in the blood of humans and unborn fetuses, it might end up in the brain because the blood brain barrier is not yet well developed and that could underlie developmental problems of the child's brain such as Autism.<sup>[1]</sup>

**One Claim of Scientific Consensus on GMO Safety Is Patently FALSE**<sup>[4]</sup> “The 1992 FDA policy statement, which was invalid when it was made, has been repeatedly rebutted and refuted and yet that policy statement is *still* the sole purported legal basis for the presence of GE foods in the US. The FDA knew the legal criteria of their policy had not been fulfilled and today there's even more evidence of conflict and disagreement within the scientific community.

In fact, on January 24, a statement signed by 300 scientists, researchers, physicians and scholars was published in the peer-reviewed journal Environmental Sciences Europe, asserting that *there is no scientific consensus* on the safety of GMOs. Moreover, the paper titled “No Scientific Consensus on GMO Safety,” states that the claim of scientific consensus on GMO safety is in actuality “an artificial construct that has been falsely perpetuated.” The paper also notes that such a claim “is misleading and misrepresents or outright ignores the currently available scientific evidence and the broad diversity of scientific opinions among scientists on this issue.”

In addition, the FDA still does not possess any evidence demonstrating safety because they do not even do scientific reviews. And even if they *did*, a large number of scientists say there's still no evidence demonstrating that GE foods are safe while a number of independent studies have raised serious health concerns. **Quite clearly, there is NOT a “general recognition of safety among scientists,” and these foods are on the market illegally. They were illegal when they were first introduced in 1992, and they continue to be illegal now in 2015.**

“One of the nation's most important and most venerable consumer safety statutes, consumer protection laws, has been blatantly and intentionally violated by the agency that's supposed to uphold it, and they've been getting away with it all these years,” says Steven Druker. “It's time to really pull the rug out from under the whole thing, because the evidence is overwhelming. All people have to do is just read it.” [In this article, attorney, Steven Druker, author of [Altered Genes, Twisted Truth](#), exposes scientific fraud, collusion and continues the fascinating story of how GMOs came into being and have been allowed to permeate our food supply through illegal means and without legally required safety testing.]<sup>[4]</sup>

The pervasiveness of GMOs in animal feed, also makes it a challenge for organic producers to secure uncontaminated organic feed. This is a challenge that will continue to grow if GMO crops are allowed to continue their land cover growth as a result of limited supply, increasing the risk of irreversible cross contamination.<sup>[1]</sup>

**What are other sources of GMOs?** Currently, commercialized GM crops in the U.S. include soy (94%), cotton (90%), canola (90%), sugar beets (95%), corn (88%), Hawaiian papaya (more than 50%), zucchini and yellow squash (over 24,000 acres). Learn more on sources of GMOs – follow the extensive links at IRT website. Products derived from the forgoing list, include oils from all four, soy protein, soy lecithin, cornstarch, corn syrup and high fructose corn syrup among others – refer to this link: <http://responsibletechnology.org/gmo-basics/gmos-in-food>

**Note: a glaring loophole occurs** where the GMO label is not applied to some soy food products or animals raised on GMOs. Thus, is responsible for large amounts of unnoticed GMO exposure. I.e., meat, eggs, dairy, soy lecithin in supplements and chocolate.

Learn more at these reference links and at the expanded report to be posted soon at the AF forum which includes more reactions from GM food consumption and the latest developments.

## REFERENCES

[1] Jeffrey M. Smith – Quotes from **The Gluten Summit 2013** plus: <http://www.responsibletechnology.org/about>, [responsibletechnology.org](http://responsibletechnology.org) [nongmoshoppingguide.com](http://nongmoshoppingguide.com) [geneticroullettemovie.com](http://geneticroullettemovie.com) <http://responsibletechnology.org/docs/141.pdf>

**Composite Book Review—Genetic Roulette: The Documented Health Risks of GE Foods**

<http://tinyurl.com/oupenfb>

[2] [Reprod Toxicol](http://www.reprod.toxicol). 2011 May;31(4):528-33. Epub 2011 Feb 18. [Aris A, Leblanc S](http://www.ncbi.nlm.nih.gov/pubmed/21511111). **Maternal and fetal exposure to pesticides associated to genetically modified foods in Eastern Townships of Quebec, Canada.**

<http://tinyurl.com/l5d53wv>

[3] **Bought** (movie) <http://free.boughtmovie.net/> - segment on GMOs

[4] **One Claim of Scientific Consensus on GMO Safety Is Patently FALSE** <http://tinyurl.com/pym34f8>

**Altered Genes Twisted Truth**, Steven M. Druker, Atty. <http://alteredgenestwistedtruth.com/>

<http://tinyurl.com/n6mbgvv>

[5] Food Babe <http://tinyurl.com/mf2km9k> GMOs in beer <http://tinyurl.com/ppunww9> GMO ingredient list

<http://tinyurl.com/p72q9va>

**Must view:** Video series offering expert testimony "**GMO Ticking Timebomb**" directed by Gary Null, PhD, author of **Seeds of Death**. <http://tinyurl.com/o3kp7al> as well as the full movie

**Genetic Roulette – The Gamble of Our Lives** - winner - **2012 Movie of the Year** by the Solari Report and the Top Transformational Film of 2012 by AwareGuide! <http://online.www.geneticroullettemovie.com/>

**The Hidden Agenda of Genetic Manipulation** F. William Engdahl [\[www.amazon.com\]](http://www.amazon.com)

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