

THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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16th YEAR



Welcome to the first issue of the newsletter in 2015! I am very happy to report to those subscribers who haven't visited our website forum recently, that I am now able to end my two and a half month sabbatical forced on us with my wife's, Magdalena, 95% certain kidney cancer diagnosis landing on our doorstep in early December. In early January, Magdalena underwent a successful open nephrectomy surgery at MD Anderson in Houston with top kidney cancer surgeon Dr. Christopher Wood.

I knew something good was up, when Dr. Wood came through the waiting room door immediately after her big procedure with a huge grin on his lips carrying the news that her tumor was a rare 'Lipid-poor AML' benign form. Needless to say, we both felt like winners of the Powerball Lottery! Magdalena is recovering well from her major surgery, allowing me to get back in the saddle here, though a few weeks later than usual in getting out this issue of the newsletter. Thank you all for your patience and kind expressions of support to us both during this time.

We start this New Year off with three relevant reports of long-term interest to Afibbers. First, is a well-done prospective study from Wake Forest revealing a long suspected, but until now unconfirmed, association between AFIB and subsequent risk of Myocardial Infarction (MI), comparable to the well-known connection with having an MI (heart attack) and then subsequently developing AFIB. Each year we learn more that AFIB is far from benign, especially for those Afibbers with higher risk scores.

Next, a similar large-scale and broad-scope retrospective report out of Sweden, finds that anti-coagulation in people with AFIB and a CHA₂DS₂-VASc score of 1 is unlikely to be beneficial, and quite possibly harmful. A message founder Hans Larsen has shared for years, in warning not to go too far overboard in overmedicating low risk lone afibbers. We all agree on the paramount importance of oral anti-coagulation (OAC) for higher risk patients, but the European guideline of a CHA₂DS₂-VASc score of 1 as the marker for starting OAC therapy appears to be on shaky ground, at least as a routine guideline.

Our third report looks at a timely study from Japan examining the Left Atrial Appendage Flow Velocity (LAAFV) both before, and after, successful ablation of persistent AFIB with a follow up TEE, six or more months after NSR had been sustained in a 104 patient cohort of persistent Afibbers. The results here will be of keen interest to the growing numbers of persistent, and even long standing paroxysmal AFIB members of our community here, and gives some valuable insights into who might stop OAC therapy safely, and who might still need to continue such protection or consider an LAA closure device.

My final contribution to this issue is a summary of two key topics at the recent International Symposium on LAA in Los Angeles I attended two weeks ago. Key insights were shared that we touch on here you wont want to miss.

Finally, long-time forum stalwart Jackie Burgess, has kindly jumped in to help out with one of her thorough reviews of the problems of anti-inflammatory factors in most of our diets like wheat and gluten. Jackie gives us an in-depth overview of these interesting issues, and a host of links for those wishing to improve their overall health for the better. Many thanks, Jackie!

Wishing you all only NSR!
Shannon

Atrial Fibrillation and the Risk of Myocardial Infarction

WAKE FOREST SCHOOL OF MEDICINE - WINSTON SALEM, NC. Our first review in this issue highlights a new wrinkle to the long-recognized risk factor between having an MI (myocardial infarction) and subsequent development of AFIB, with AFIB occurring in 6% to 21% of MI patients. Could the flipside of having AFIB also be a significant risk factor for subsequent MI itself, and if so to what extent?

For the first time, a large-scale prospective study investigates this suspected, but until now poorly documented association between AFIB as another independent risk factor for MI by using data from the extensive REGARDS (Reasons for Geographic and Racial Differences in Stroke) registry. A study cohort of 23,928 participants from this large US database, and all without coronary heart disease (CHD) at baseline, were enrolled between 2003 and 2007 with follow up through December 2009.

The REGARDS study goals have been published elsewhere, but briefly it was designed to investigate regional and racial disparities in stroke mortality by oversampling black and white residents of the 'stroke belt' region including North and South Carolina, Georgia, Alabama, Mississippi, Tennessee, Arkansas and Louisiana ... basically most of the southern states.

Individuals with prevalent CHD, history of MI or coronary revascularization procedures at baseline were excluded from this cohort selected for the current investigation. Thorough and extensive steps were taken for ascertainment of both MI events and AF history. A host of covariates and confounding elements were also taken into account as well to derive as accurate and relevant results as possible within the limitations of the studies structure.

The Results

Out of these **23,928** participants included in this analysis after all screening and exclusions AFIB, was present at baseline in **1631** of them: with **268** of these AFIB cases detected by screening ECG and the rest were found from medical history, or both methods. Comparing those without AFIB to those with AFIB, those with AFIB were older and less likely to be both black and men. Not surprisingly, those with AFIB generally also had more CHD risk factors like hypertension, diabetes (i.e. a higher CHADS2 score) and worse kidney function.

Out of a **median 4.5 years (6.9 years total)** of follow up, **648** MI events occurred. The age adjusted incidence rate for MI in participants with AFIB at **(12.0 [95%CI, 9.6-14.9] per 1000 person-years)**, was two times greater than the rate in those without AFIB **(6.0 [95%CI, 5.6-6.6] per 1000 person-years) (P < .001)**. No mention is made of low risk lone Afibbers and lower MI risks, though it could be inferred.

Going beyond the unadjusted cumulative incidence of MI events by baseline AFIB status, in a socio-demographically adjusted Cox proportional hazards model, AFIB was associated with a **96%** increase in MI risk compared to those with no AFIB **(HR-[hazard ratio] 1.96 [95%CI, 1.52-2.52])**. This finding compares well when AFIB was ascertained by even more restrictive methods such a detection by direct ECG and/or history of a physician diagnosis plus warfarin use **(HR 1.77 [95%CI, 1.14-2.76])**.

In addition, the association between AF and MI remained strong and significant after further adjustment for CHD risk factors and confounders in the fully adjusted model **(HR 1.70 [95%CI, 1.26-2.30])**, and when accounting for baseline heart failure, stroke events and chest pain hospitalization as time-updated variables **(HR 1.71 [95%CI, 1.27-2.30])**. Even when using death as a competing risk, it did not appreciably alter this strong association between AFIB and MI **(HR 1.65 [95%CI, 1.24-2.25])**.

Of interest, in a multivariable model similar to the one noted above, the risk of MI associated with AFIB in warfarin users was significantly less than in non-warfarin users **(HR 0.76 [95%CI, 0.29-1.94])** vs. **(HR 1.92 [95%CI, 1.42-2.60]) (P = .02 for interaction)**. And yet no statistically significant difference in MI risk was seen between AFIB aspirin users **(HR 2.13 [95%CI, 1.44-3.15])** vs. non-aspirin users with AFIB **(HR 1.36 [95%CI, 0.86-2.15])**, even though the non-significant trend was for slightly less MI associated with non-aspirin using Afibbers, counter to what most physicians used to expect.

Some may find this later association surprising; though it is consistent with other recent studies not only calling into question the value of aspirin as an AFIB-related stroke preventative, but also potentially calling into question aspirin's long-assumed role of MI prevention for the AFIB patient as well.

The lower risk of MI associated with AFIB warfarin users vs. non warfarin users suggests a potential modifying effect on MI risk from warfarin use, and this is in accord with previous reports showing that warfarin might provide an MI protective effect after acute coronary syndromes and in AFIB patients who were on warfarin for stroke prevention.

For sure, no one is suggesting giving warfarin as a stand alone front-line MI prophylactic based on these results. But those AFIB patients who already qualify for anticoagulation and who also have a higher range CHAD2S2-VASc score, might take some added solace in the likelihood that their warfarin dose may play at least some part in helping to avoid an MI. As expected, CHADS2 score >1 was associated with increase risk of MI in Afibbers in this study.

Whether or not one can presume such a potential MI risk-reducing effect in AFIB patients from the NOAC drugs, it is too early to know until further studies show conclusively that we can extend such a connection to the NOAC class of drugs.

The last significant finding from this largest US cohort study is that the risk of MI associated with AFIB differed by sex and race, but not so much by age, with women and blacks having significantly stronger associations between AFIB and their risk of an MI event, than do whites and men.

In sex-race stratified analysis, MI association with AFIB was strongest in black men (**HR 2.91 [95%,CI 1.62-5.23]**), followed by white women (**HR 2.33 [95%,CI 1.27-4.28]**), then black women (**HR 2.17 [95%,CI 1.19-3.98]**), but interestingly was non-significant for white men (**HR 0.86 [95%,CI 0.58-1.56]**).

Conclusion

This important large-scale population-based prospective study convincingly establishes an association between AFIB and an increased risk of Myocardial infarction, especially for blacks of both sexes and white women. Since only African Americans and Caucasians were recruited in the REGARDS study, we cannot extrapolate these findings to other races. Low risk Afibbers may have lowered the overall MI risk. Bottom-line, these results further underline the seriousness of AFIB as a long-term public health burden.

Soliman, EZ, et al. Atrial Fibrillation and the Risk of Myocardial Infarction. JAMA intern Med. <http://dx.doi.org/10.1001/jamainternmed.2013.11912>

Editors comments: In recent years we have seen a growing awareness of the impact of AFIB in its often two-way association with a growing list of chronic conditions connected with both genetics, aging, environmental factors and poor life style choices. In this case, we now see that AFIB can predispose to an increased risk for MI for large portions of the population, joining the long established two-way street association between an incident MI and subsequent development of AFIB.

Either one may apparently be the chicken or the egg, or perhaps more accurately, they both share common cofactors and mutual contributors in a synergistic dance towards increasing illness unless proactive steps are taken to reduce at least those underlying forces within our influence that we can control and shape to at least some degree. Such has been the day one core message of this newsletter.

Unlike the benign arrhythmia it used to so often be assumed, even by cardiologist in the past, it has become all too obvious that AFIB is a serious malady with growing associations now to dementia, Alzheimer's, heart failure and both stroke and now modest myocardial infarction risk. Emphasizing the wisdom of making reducing AFIB burden in one's life, by whatever means, a top priority.

Benefits of Anticoagulation Unlikely in AFIB Patients with a CHA2DS2-VASc Score of 1

STOCKHOLM, SWEDEN. Once again, comes another important original AFIB-related investigation from Sweden's Karolinska Institute. It is no surprise that Sweden and Scandinavia are such a hot bed of high quality population-based epidemiological medical research, what with the entire population of residents and their complete medical histories being so thoroughly documented in extensive and well-controlled national health registries.

In this case, structuring a retrospective study of unselected patients from these nationwide, cross-matched Swedish health registries to conduct a large-scale assessment of AFIB-related stroke risk among all Swedish residents who have AFIB and a CHA2DS2-VASc score of 1, and never having taken OAC drugs.

As such, the study population included all Swedish patients with a diagnosis of non-valvular AFIB over a five year period between July 1, 2005 and June 31, 2010, who had not been exposed to warfarin or other OAC drugs at any time during follow up from an initial diagnosis of AFIB.

Current accepted guidelines in the US and Europe recommend a risk-based view toward stroke prevention with OAC agents. The problem is, that large differences exist among the estimates of risk among those who are not taking OAC drugs, and especially those deemed at low risks by both the older CHADS2 scoring system and the new benchmark, CHA2DS2-VASc, that has in recent years universally superseded the less discriminating CHADS2.

For higher risk patients the practical debate is a moot one anyway, since all agree that every patient above a CHA2DS2-VASc ≥ 2 , should be on OAC drugs by definition (except in select case-by-case extenuating circumstances).

For those unfamiliar with how this acronym works, CHA2DS2-VASc is a risk number scoring system where (**C=Congestive heart failure = 1 point, H=Hypertension = 1 point, A=Age ≥ 75 yrs = 1 point, D=Diabetes = 1 point, S=Stroke/TIA = 2 points, V=Vascular = 1 point, A= Age between 65yr and 74yr = 1 point and S=Sex= 1 point for female gender**) Thus, in effect, an **age ≥ 75 equals 2 points** of added risk burden.

In the US guidelines, a CHA2DS2-VASc ≥ 2 means OAC drugs are required, while in Europe they interpret CHA2DS2-VASc ≥ 1 as the threshold for starting OAC, but in Europe they do not assign any points to female gender as an added risk, so the two thresholds (US and Europe) are basically the same in practice.

Study Methods

One of the impressive aspects of this study was the care taken to evaluate how the estimated event rate for stroke was affected by the various ways of counting these events as used in previous studies, and thereby getting an informative view of how much the 'stroke' rate was truly influenced by inclusion of other diagnosis previously, such as PE (pulmonary embolism) and TIA (transient ischemic attack).

They also evaluated how reports of acute ischemic stroke recorded as a low secondary diagnosis affected the total stroke risk estimates. And included as well analysis of quarantine periods of varying lengths (blinking periods) between the initial index diagnosis of AFIB and any recorded CVA events that were accepted. If no blinking period was used in given prior estimates, then the odds are high that these classifications would lead to an overestimation of stroke risk associated with AFIB. It is the long-term risk of stroke that is relevant regarding anticoagulation decisions.

Stroke rates were calculated as events per 100 years at risk, but were expressed as annualized percent rates for easier comprehension.

Results and Conclusions

The overall risk for ischemic stroke among AFIB patients with a CHA₂DS₂-VASc of 1 appears to be **lower** than previous studies have indicated. A finding in line with other studies and findings highlighted over the years in this newsletter. The author's concluded that these earlier findings may have led to unnecessary and potentially harmful OAC treatment of low-risk patients.

At a CHA₂DS₂-VASc of 1 for Swedish AFIB residents, the annual event rate for stroke in this large study varied between **0.5%** and **0.9%** depending on whether only ischemic strokes were counted or a more inclusive endpoint was used. The event rate dropped to a low of **0.3%** in the more restrictive Riks-Stroke registry.

This study found that divergent estimates of stroke risks associated with AFIB are partly the result of differences in prior study methods. Event rates, thought to be actual 'stroke' risk, in reality often represent generally more varied and diverse endpoints, such as including PE and TIA, when those are not technically appropriate to include if the target endpoint is true stroke. Inevitably, including more varied diagnosis under one label of 'stroke' can obviously inflate the estimated stroke risk beyond its true real world value.

The authors noted that '... no doubt PEs and TIAs should be used to alert physicians to the need to initiate OAC therapy. However, TIAs are difficult to validate, and thus remain a poor endpoint for stroke studies. In addition, primary prevention of PE among AFIB patients is not an approved indication for OAC treatment.' This study found that overall event rates in some older studies that have been widely quoted, inflate the estimated AFIB related stroke risk by **44%** if TIA, PE, systemic embolism and unspecified strokes were added to the endpoint.

European guidelines clearly spell out that a woman with a CHA₂DS₂-VASc of 1 should not be anti-coagulated on the basis of sex alone. And this study found that, indeed, women were truly low risk, with an annualized ischemic stroke rate of only **0.1%** to **0.2%**. While low risk men's ischemic stroke rate, according to the strict definition Riks-Stroke registry, was **0.5%** and jumped only marginally to **0.7%** according to the slightly more inclusive Swedish National Patient Register. But, when the endpoint was enlarged by inclusion of TIA, PE, arterial embolism and 'stroke-type-not-specified' or hemorrhagic, the annual rate for men jumped to **1.3%**. Keep in mind that an annualized stroke rate of **greater than >1%** is generally accepted as the threshold to make the overall risk/reward worth adding OAC therapy .

And yet, in spite of a European guideline CHA₂DS₂-VASc of 1 being their threshold to consider anticoagulation (when not adding 1 point for women based solely on sex alone), this study found that **almost half of the men with scores 1** were nevertheless given OAC therapy, and **22.5% of the same low risk women** cohort with a risk score of 1 were also anti-coagulated.

The bottom-line of this key study is that the risks of stroke in AFIB patients with a CHA₂DS₂-VASc of 1 is lower than previously thought, thus the authors feel the EU guideline threshold of 1 for starting OAC drugs may be unwise and that no benefit is anticipated for routine use of OAC agents in these patients.

Freiberg L, et al. Benefit of Anticoagulation Unlikely in Patients with Atrial Fibrillation and CHA₂DS₂-VASc Score of 1. JACC Vol.65 No.3 2015 <http://dx.doi.org/10.1006/j.jacc.2014.10.052>

Editors Comments: Once again, a major study confirms the long supported opinion shared by this newsletter and website, that rates of stroke for people in the low risk category, such as lone afibbers, do not merit anti-coagulation. That nearly **50% of Swedish men** and **22.5% of women** who met the low risk ranking of **1 on the CHA₂DS₂-VASc** scoring system in Europe were on OACs, without adding any points for female gender, underscores a common **disconnect** and time lag between the consensus of leading edge research and those insights making there way to front-line physician decision making.

To be sure, these risk-based stroke scoring systems are not infallible, and as well-known OAC drug researchers Drs. Daniel Singer and Michael Ezekowitz noted in a companion editorial¹ to this important study, the often dramatic consequences of ischemic stroke off OAC, or major bleeding events on OAC, underscore the need to focus on absolute rates of stroke risk while being alert to biases in studies reporting these risks, and thus more carefully consider the prediction point scores derived there from.

We likely won't make any better headway in fine-tuning for whom, and when, OAC therapy is truly warranted among these lowest risk AFIB patients until large-scale trials are completed of the NOAC drugs versus placebo (with NOAC's lower risk of intracranial bleeding compared to warfarin, as well as their modestly lower to equivalent ischemic stroke risks). In the meantime, its clear that an EU CHA2DS2-VASc of 1 doesn't warrant anticoagulation as routine therapy, not without other compelling factors tipping the scales in favor of starting an OAC drug in these otherwise low risk patients.

LAA Flow Velocity after Successful Ablation of Persistent AFIB

IBARAKI, JAPAN. This timely study attempts to uncover factors associated with either improvements, or stasis, in Left Atrial Appendage Flow Velocity (LAAFV) during Normal Sinus Rhythm (NSR) after successful catheter ablation for persistent AFIB. And has become an important topic with the increasing numbers of our readers, and AFIB ablation patients as a whole, who have had persistent AFIB ablation in recent years and need to have a good metric for when, and if, it is safe to stop OAC therapy. The question is relevant to a good many long-standing paroxysmal cases as well.

A sufficiently low LAAFV, as measured by trans-esophageal echocardiography (TEE) showing LAA emptying velocities $\leq 40\text{cm/sec}$, has a long recognized association with increased thromboembolic stroke/TIA risk in active AFIB patients. In contrast, this retrospective study included 104 Japanese patients with persistent AFIB at their institution who underwent successful RF (radio frequency) ablation, and who then maintained NSR for at least 6 months after the final ablation procedure.

The point being, to see if LAAFV had increased over this prolonged time period of at least 6 months of unbroken NSR, representing positive reverse structural remodeling of the LA and LAA during this time frame, compared to the generally low LAAFV seen in these pre-ablation persistent AFIB patients which averaged a mean LAAFV of just $29 \pm 11\text{cm/sec}$. Thus, the highest mean value was only 40cm/sec , or borderline too low to even consider stopping OAC during this group's prior persistent AFIB time frame. And the **average 29cm/sec** was far too low for safety going off OAC therapy. Note too, that some active long-term paroxysmal afibbers can also have subpar LAAFV, even in modest periods of NSR as well.

This cohort of persistent ablation patients who had baseline TEEs prior to, and during, the first ablation procedure, as well as after at least 6 months of stable NSR, were matched by a control group of **104 patients** who prior to a non-cardiac surgery at the same institution did not have a TEE, but rather a standard TTE (transthoracic-echocardiography), and none of whom had AFIB or any cardiovascular disease previously or during the study, in order to match age, gender, and body surface area between the control and active RF ablation study group for comparison, which were nearly identical by design.

The Results ...

(1) The LAAFV of persistent AFIB patients after successful ablation and at least 6 months of sustained NSR, show major overall improvement over prior lower velocities during persistent AFIB, indicating extensive reverse remodeling in most patients.

(2) In spite of the vast majority showing improvement to a safe LAAFV, roughly a quarter remained below the threshold of $\leq 40\text{cm/sec}$ for considering stopping OAC therapy, even though all improved.

¹ Singer, DE & Ezekowitz MD. *Adding Rigor to Stroke Risk Prediction in AFIB*. *JACC Vol.65 No.3 2015*
<http://dx.doi.org/10.1016/j.jacc.2014.11.013>

- (3) A CHA2DS2-VASc score of ≥ 2 and female gender were identified as independent parameters predicting low LAAFV post-ablation, though most of these women also had a risk score ≥ 2 .
- (4) Spontaneous Echo Contrast (SEC) found during AFIB, prior to ablation, was also a predictor of low LAAFV after ablation and prolonged NSR, though all SEC patients also had CHA2DS2-VASc score ≥ 2 .
- (5) 23 out of 104 patients with low LAAFV post-ablation showed statistically different Left Atrial Diameter (LAD) and mitral flow parameters compared to those 81 out of 104 patients with a post-ablation higher and safer LAAFV.

Thus, the key take home points here for EPs and patients alike, is that 22% of patients with persistent AFIB demonstrated inadequate recovery of LAAFV despite maintenance of NSR after successful RF ablation. And that this was in spite of significant improvement of LAD, LAAFV and SEC as a whole after successful ablation. Although, all of those whose LAAVF remained too low to warrant stopping OAC did have some improvement in their LAA flow velocity, just not enough to stop on-going OAC therapy.

Finally, a CHA2DS2-VASc score ≥ 2 was identified as a novel predictor of poor recovery of LAAFV after restoration of NSR long term, all of which indicates that the magnitude of reverse remodeling after successful ablation for persistent AFIB may be limited by degree of remodeling suffered before ablation.

Kusa, S, et al. Left Atrial Appendage Flow Velocity after Successful Ablation of Persistent AFIB. Am Heart J. 2015;169:211-21 <http://dx.doi.org/10.1016/j.ahj.2014.11.009>

Editors Comment: With the growing number of persistent AFIB cases being successfully converted to NSR via an expert catheter ablation process, it stands to reason more fine-tuned guidelines for determining on-going anti-coagulation status for these patients is warranted. This study is the first, to my knowledge, to indicate using a CHA2DS2-VASc score of ≥ 2 as a possible indicator for a confirming TEE exam after 6 months of NSR to evaluate whether or not those known higher risk patients can safely forgo the anti-coagulation issue. Many of these folks who cannot stop OAC safely, even after achieving NSR through elimination of arrhythmia, like those undergoing LAA isolation ablation who similarly have a too low LAAFV at 6 months TEE, might also be good candidates for an LAA exclusion procedure in place of, or in combination with, ongoing OAC therapy.

Shorts Takes from the annual International Symposium on Left Atrial Appendage (ISLAA) in Los Angeles – February 6-8, 2015

I had the pleasure to attend the ISLAA symposium again for the second year of its now three year annual history ... this time held in the Marriott at Marina Del Rey near LA airport. I greatly enjoy this stimulating and very informative conference format which uniquely combines all three major cardiovascular sub-specialties of Cardiac Surgeons (CS), Interventional Cardiologists (IC) and Electro-physiologists (EP) coming together to collaborate and share their own unique perspectives on this same vital topic of cardiovascular well being and overall good health centered around what used to be considered a small afterthought in the heart ... the left atrial appendage.

In the interest of brevity, we will take a look at highlights of two key take home issues gleaned from the large wealth of detailed insights and findings shared by a large group of renowned cardiac surgeons, EPs and ICs over two non-stop days. I was most impressed by the palpable increase in attention and effort regarding LAA issues evident in just the past year.

Famous physicians such as Drs. James Cox, Horst Seivert and John Camm, joined vibrant new voices (new to me anyway) like Drs. Basel Ramlawi head of AFIB surgery at Houston's DeBakey Heart & Vascular Center, and Tzikas Apostolos from Greece, as well as a host of frequent well-known heavy weights like Drs. Andrea Natale, DJ Lakkireddy, Saibal Kar and Vivek Reddy all four of whom were sponsors of the ISLAA ... and far too many others to list here. There certainly was no shortage of important information to convey about this one lonely little appendage, a bit shorter and slightly wider than an average thumb, so let's dive in!

- **LAA Arrhythmogenesis:** First up, Dr Sam Asirvatham from Mayo Clinic, gave an excellent opening conference overview of the anatomy and physiology of the LAA in which he reminded everyone that the posterior left atrial wall, a common target of many advance PVAI-based ablation procedures, is not true atrial myocardium tissue.

Dr James Cox, iconic creator of the original Cox-Maze surgical AFIB ablation back in 1987, was next up on the podium and when Dr Cox speaks, everyone listens. And listen they did, when he emphatically urged all the attendants to take careful note to read and accept the landmark study by Dr. Natale's St David's group from 2010 (reference: **DiBiase, L. Left Atrial Appendage: An Under-recognized Trigger Site of AFIB. *Circulation* 2010;122 (109-118) <http://dx.doi.org/10.1161/circulationaha.109.928903>**)... showing that of **987** consecutive ablation patients returning for a repeat follow-up ablation, a full **27% (266)** of them were found to have significant active triggers from the LAA.

And **8.7% (86)** of this **987** total cohort **only** had triggering in the LAA alone, with no reconnections at all in the PVs, the posterior wall or elsewhere in either right or left atriums. A significant percentage with LAA involvement that, if not addressed in the follow up ablation, will surely lead to more failure and further repeat ablations.

Dr Cox, emphasized that in his long experience in removing the LAA as a routine step in his Cox-maze procedure, these study numbers were accurate and very important to understand and to address to improve ablation success rates, as well as for better stroke reduction in afibbers. Noting that: *"No question, by including LAA isolation as part of the procedure for the appropriate patient, significant increases in success rates will result"*. A predicted outcome already demonstrated and validated in studies by both Kansas Univ. Medical Center and St David's large research center.

Dr Cox finished his appeal to this multi-specialty audience with a bold prediction that: *"Few things in medicine will bring as much benefit to health as clipping or occluding the LAA"*.

- **LAA Closure Devices:** Much was made about improvements in the new **LARIAT** and **Watchman** LAA closure systems, and big support was shown for the **ATRICLIP** surgical LAA ligation clip, which impressed me with its many positive attributes.

Everyone there is hoping for, and expecting, FDA approval for the Watchman in the coming months, even if limited at first. The *'Watchman FLX'* is the new enhanced design that will be on offer to a wider audience of patients when FDA approval is finally achieved. Watchman FLX has a rounded distal end with its PET fabric extending more distally as well. This new device can be partly recaptured and moved deeper into the LAA, if needed; has 12 'J' shaped anchors rather than 10 straight anchors, and 18 strut frame members rather than 10 as in the original device. Finally, a recessed screw head on the proximal face of the device faces the LA blood flow.

The **LARIAT** has also be improved with a larger adjustable suture replacing the fixed-size pre-tied suture that limited the use of the LARIAT by a significant number of patients that could not pass initial anatomical screening for the system when their LAA morphology would not fit the fixed suture. A larger suture should open the door, at least anatomically, to many more people.

The epicardial catheter portion of the LARIAT has also been redesigned including a braided catheter to allow more torsional control and flexibility when routing the catheter through the narrow pericardial space just above the right ventricle to the LAA. This is a big improvement making it easier for the operator to get a good fit, and safer too, helping to avoid the rare, but not unheard of, nicking of the RVOT (right ventricular outflow track) by the epicardial catheter, and mostly in the hands of inexperienced operators, now made an even smaller risk.

The **ATRICLIP** was the biggest star of the show in my view. Several surgeons extolled its relative simplicity and easy application, only requiring an experienced cardiac surgeon no more than 30 minutes to complete the entire process.

In addition, the moment the ATRICLIP clamps down on the proximal end of the LAA, you have instant permanent electrical and vascular isolation with little to no chance of ever having a leak. Also, like with the LARIAT, no hardware is left behind in the endocardial blood stream. Thus, with complete LAA exclusion immediately confirmed, the patient never again has to take an OAC drug, at least not as a LAA-based stroke prevention measure. Some people with very high CVD risk factors may require an ATRICLIP, a LARIAT or Watchman, plus ongoing OAC therapy to protect against non-LAA systemic risks of stroke or other embolic events.

ATRICLIP has been installed in 45,000+ cases; most during open heart surgery of one kind or another, but increasingly in stand alone, minimally invasive ATRICLIP installations during Mini-Maze surgical ablations. In all this time, no deaths have been attributed to the procedure and no known safety issues have been found. The only two caveats are a little more pericardial discomfort and pain that is typically felt somewhat longer than with the LARIAT, up to 3 to 4 weeks in decreasing degrees after the procedure. But the pain is generally well managed with colchicine and pain medication. After discomfort subsides, there should be no late surprises with this solid history of success, and it's a small price to pay for such a result, in my book.

All three of these proven LAA closure methods are far and away better and more advanced than the original surgical attempts at ligation via sutures or staples. These new generation approaches seem to provide far more effective and safer outcomes, for the most part, when done by experienced operators, be they EPs, ICs or surgeons.

Work is underway developing a sub-xiphoid ATRICLIP access procedure for EPs to install the clip, though it will likely be at least a few years before it's ready for prime time and has approval. This would be similar to just the epicardial phase of the LARIAT, and would avoid having to partially deflate the left lung (though I am told that is a pretty routine step with little chance of a unwanted outcome as it is).

There is so much more to share from the cutting edge of this exciting new focus in cardiovascular medicine. And we'll explore more in coming issues of relevance to an increasing number of us. But it's time now to hear from long-time afibbers forum leader, Jackie Burgess, as she kindly offered to assist me during my sabbatical with her in-depth insights on '*Enlightened Dietary Tips*' for us all!

SPECIAL REPORT

Dietary Enlightenment Tips for the New Year

by Jackie Burgess

Resolved: Start the New Year with knowledge and awareness about damaging eating habits that adversely affect our health yet remain largely unrecognized as causal by most medical practitioners. It's never too late to begin.

Introduction

In a recent presentation, a Functional Medicine practitioner talked about the importance of eating an "enlightened" diet. Exactly what does that mean?

In this case, enlightenment comes in the form of science-based evidence and clinical experiences linking a multitude of alarming detrimental health effects caused by large daily intakes of high-carbohydrate “modern” grains or seeds from grasses and other high-carb foods known to contribute to over 300 chronic ailments. Unfortunately, all too often, treatment is not based on tracing symptoms to core causes and thus patients are typically treated with Rx drugs that merely mask symptoms rather than address the etiology, allowing the damaging effects to all too often continue, even if suppressed somewhat by such symptom control medications.

This overview can hardly do justice to a topic with such broad complexities, yet it’s imperative that we all become at least aware of the wheat/grain connection underlying so many common ailments. This is not, however, just another wheat/gluten toxicity alert, but rather an expose’ on many other components in the modern-day engineered and manipulated wheat associated with major chronic, adverse health effects that result from an adaptive response to a biologically incompatible food. The high-caloric, high-glycemic content of grains which typically comprises 50% of all human calories world-wide as wheat, corn and rice then contributes to health problems such as blood sugar elevations, insulin resistance, weight gain, inflammation, autoimmunity and much more. The over-abundance of wheat/gluten-free substitute products now available adds to the high-carb burden and furthers the overall problem. Experts say: *Be grain free and gluten free, but be careful not to consume ‘Gluten Free’ substitute starchy carb junk foods.*

Neurodegenerative conditions are escalating and the connection to inflammation, the cornerstone of Alzheimer’s, MS and Parkinson’s, is now recognized as critical to prevent since there are no cures. According to *Lancet Neurology* “...more than 54% of the world’s current Alzheimer patient load could have been prevented if specific lifestyle issues would have been addressed.”^[1,3]

The gluten protein in wheat and other grains is but one factor making modern grains an unfriendly, poorly digestible food. And, contrary to common belief, it’s not mainly or only a Celiac or intestinal ailment for a genetic few, since research proves wheat can harm everyone’s intestines.^[4] The opiate effect of wheat’s peptide, gliadin (a gluten component), is a powerful appetite stimulant (exorphin) that drives addictive cravings. Five new groups of non-gluten proteins, distinctly different from gluten proteins, are found to cause inflammatory damage in Celiacs. Rice and corn are found problematic and another 400 gluten proteins in various grains are even more inflammatory than gliadin, the original type of gluten protein discovered in 1952.^[8]

Another major grain culprit, lectins, (invisible thorns that protect plants), even more damaging than gluten and found in many common foods including wheat and legumes, are known to promote the formation of fatty streaks, mature arterial atherosclerotic plaque and chronic, low-level inflammation from inflammatory cytokine production. “...Wheat Germ Agglutinin (WGA) is a lectin glycoprotein shown in clinical studies to cause damage to gut lining, joints, kidney, pancreas and the brain since it is able to cross the blood-brain barrier as research identifies WGA as a neurotoxic substance. WGA is also “cardiotoxic” to the lining of arteries, leading to invisible inflammatory damage, atherosclerosis and is well known to promote platelet aggregation/clumping and clot formation. Potentially inflammatory lectin-containing food groups are listed in the extended version of this report on the website forum.

“Most people are aware of food reactions with obvious symptoms of gas, bloating, diarrhea, constipation, headache, fatigue, swollen joints, water retention, hives and psoriasis. Some symptoms resolve quickly after eliminating the offending food family, but other symptoms may take 6 – 12 months to resolve. And If you are genetically intolerant, you may never be able to consume those food groups safely.”^[9]

Fortunately, two outstanding, easy-reading books help guide the way to awareness on the critical need for “enlightened” eating habits. ***Wheat Belly***, authored by Integrative Cardiologist, William Davis, and his plan ***Wheatlessness: A 21st Century Health Strategy***^[2] along with Neurologist, David Perlmutter’s clinical observations in ***Grain Brain: The Surprising Truth about Wheat, Carbs, and Sugar--Your Brain’s Silent Killers***^[3] detail the damaging health effects from consuming “modern-day” wheat and subsequently,

high-carb diets. Quotes from both books follow along with several from the 29 leading experts invited to share their research and clinical experiences on the detrimental health effects of grains at the **2013 Gluten Summit: A Grain of Truth**^[1] whose mission is to educate the public and medical professionals about the historical decline of the U. S. population's health over the past 50 years, and can be done.

A large body of scientific literature documents that by totally eliminating the offending foods, symptoms of various ailments might potentially be stabilized or prevented from occurring. A few from the extensive list includes the obvious links to obesity, diabetes, heart disease (including arrhythmia, ectopies and a number of other CVD malady's), cancer, dementia and depression, arthritis, osteoporosis and most other autoimmune conditions. The list further expands to include many lesser-known associated risk factors between wheat and modern ills which number over 200 from documented case studies^[4].

We who have been touched by Atrial Fibrillation know the importance of avoiding and managing factors contributing to silent inflammation, chronic nerve irritation, acidic tissue pH, GI distress and related chronic conditions such as diabetes, hypertension, GERD (gastro-esophageal-reflux disease), IBS (irritable bowel syndrome), H. pylori, gut dysbiosis, food reactions and so on. Therefore, this informational alert on the myriad of ailments associated with various grain reactions and the consequences of the resulting high blood glucose is critically important to ensure ongoing good health and calm hearts. A high-carb diet quickly uses up stores of magnesium and potassium essential for normal heart electrical conduction.

Expert Observations

Mark Hyman, MD *"... wheat products, not just gluten (along with sugar in all its forms, are the major contributor to so many modern ailments and is why there are now 30% more obese than undernourished in the world; and why, globally, chronic lifestyle and dietary-driven disease kills more than twice as many people as infectious disease. These non-communicable chronic diseases will cost our global economy \$47 trillion over the next 20 years.*

The history of wheat parallels the history of chronic disease and obesity across the world. Supermarkets today contain walls of wheat and corn disguised in literally hundreds of thousands of different food-like (FrankenFoods) products. Each American now consumes about 55 pounds of wheat flour every year....not just the amount but also the hidden components of wheat that drive weight gain and disease. This is not the wheat your great-grandmother used. It is FrankenWheat – a scientifically-engineered food product developed in the last 50 years. The man who engineered this modern wheat won the Nobel Prize – it promised to feed millions of starving around the world. Well, it has, and it has made them fat and sick."^[5]

Virtually every Summit presenter mentions wheat's addictive property mechanisms. Dr. Hyman says it best: *"This new modern wheat may look like wheat, but it is different in three important ways that all drive obesity, diabetes, heart disease, cancer, dementia and more. It contains:*

1. **A Super Starch** – amylopectin A that is super fattening.
2. **A form of Super Gluten** that is super-inflammatory.
3. **Forms of a Super Drug** that is super-addictive and makes you crave and eat more."^[5]

William Davis, MD (Wheat Belly) *"Dr. Davis views wheat consumption as a widespread societal problem responsible for an incredible amount of illness, obesity and suffering and he advocates removal from the diet all foods made from wheat in an effort to reduce blood sugar-- based on the fact that wheat products increase blood sugar more than nearly any other food."*^[1]

To clarify, Dr. Davis uses the term "wheat" in this context to mean **all grains** but he emphasizes, the problem is not just a grain issue, but also the metabolic effects resulting from eating starchy carbs that metabolize to glucose which then do damage to the body via Advanced Glycation End Products (AGEs) that 'caramelize' cells in addition to various other grain protein components such as gluten, gliaden plus plant and dairy lectins^[4] that cause detrimental systemic reactions and neurological damage. *He states "... if you have high blood sugar, pre-diabetes or diabetes, you can't control cardiovascular risk which*

means you'll have a heart attack, need stents, and bypass. It may take 10 or 20 years, but you'll lose control over cardiovascular risk and coronary plaque.”^[1]

Dr. Davis comments on the disparity of deluding the public into thinking that whole wheat grains are wholesome and healthy when “...in reality, 2 slices of whole wheat bread increases blood glucose levels more than 2 tablespoons of sugar. This was known by nutritionists 30 years ago, yet the ADA advises diabetics to reduce fat and include 45 – 60 grams of carbs, preferably “healthy whole grains” in each meal for 135 – 180 grams of carbs/day not including snacks.”^[2] He doesn't recommend eating gluten-free high carb foods as that just increases the glycemic load and resulting damage. In 2011, the exploding availability of GF foods was noted to add \$6 billion a year in sales for these junk products.

He notes.... “The blood test Hemoglobin A1C can be used to gauge the ongoing rate of AGE formation and serves as a simple index of glycated hemoglobin reflecting to what degree you are glycosylating body proteins beyond hemoglobin. The higher the A1C, the more you are glycosylating the protein in the lenses of your eyes, kidney tissue, arteries, skin, etc. It's a measure of ongoing aging. The higher the A1C, the faster you are aging. At 5% or less, you are aging at a normal rate; over 5%, you are moving at a faster rate taking you closer to the great nursing home in the sky. Wheat-free is anti-aging, says Davis.”^[1]

Dr. Davis points out important discrepancies in *The China Study* (T. Colin Campbell, PhD) which failed to note the high correlation of cardiovascular disease and wheat consumption. The correlation of “foods that increase blood sugar the most, also trigger insulin the most, followed by the most vigorous stimulation of fatty liver, visceral fat deposits, increased VLDL/triglycerides and small LDL particle production.”^[2] He advises to look at LDL particles rather than LDL cholesterol.

David Perlmutter, MD, FACN, ABHM (*Grain Brain*) puts the focus on neurological disorders resulting from the detrimental effect of high glucose/carb intake from grains (lifestyle) and the connection to Alzheimer's for which there is no treatment or cure. He says that's about 2.6 million people that didn't have to get in that situation in the first place. He notes many are now recognizing the relationship between Alzheimer's and dysregulation of blood sugar to the extent that some are calling Alzheimer's “Diabetes Type III” because of this profound relationship.”^[1,3]

An NEJM 2013 study demonstrated even mild elevations of blood sugar just a bit over 100 already correlate with risk of dementia. It's known that elevated A1C directly correlates with shrinkage of the hippocampus (the brain's memory center) and that this goes back to the effect of inflammation and the glycation process which 1) increases production of free radicals (chemicals) that damage everything in sight and 2) increases the inflammatory process which is devastating for the human brain.^[1,3]

Because we can control inflammation, his advice is to cut back on carbs since excessive carbs are the killers due to the free-radical production which damages our DNA, our fat, our protein and every part of the body. His recommendation is to eat an extremely low carbohydrate/high-fat diet. Healthy cell membranes require healthful fat intake and says that this notion of eating high fat is going to cause heart disease could not be further from the truth as shown by the NEJM study 2008.^[1,3] As for wheat and the gluten components, he discusses each in great detail in ***Grain Brain*** saying gluten is basically a protein we are not designed to accept. Neurological manifestations of gluten reactivity are a scientific fact and gluten sensitivity can be a neurologic problem without any involvement of the gut whatsoever, but the factor of cross-reactivity with other types of proteins can be a strong influence.^[1,3] Dr. Perlmutter sees a lot of patients with headaches that resolve with a wheat/gluten-free diet. ***Grain Brain*** is a must-read for those interesting in exploring the subject in more depth, along with more information at his website.

Mark Houston, MD, MS, ABAARM, FACP, GAHA, FASH,^[6] a cardiologist with a Master's degree in Nutrition and Metabolic Medicine, says that gluten sensitivity can impact your heart and cardiovascular (CV) system. A point he emphasizes is the influence of autoimmunity on the development of CV disease. He says: “The blood vessel has an infinite number of insults, but only three finite responses to the insults which are **inflammation, oxidative stress and vascular autoimmune disease**. Autoimmune disease is one of the newest and more important factors leading to endothelial dysfunction, peripheral vascular disease, coronary heart disease, heart failure and other types of cardiovascular illnesses in this country and worldwide.”^[1]

A report in the medical journal *Circulation* shows antibodies to trans-glutaminase, a marker for celiac disease, also can affect the endothelium or inner lining of blood vessels...a classic example of how molecular mimicry works. If a gluten sensitive person eats gluten, they make antibodies to the gluten which the body sees as a foreign protein/object or invader so this is one of the infinite insults. The body responds to the insult in those three finite ways: inflammation, oxidative stress and autoimmune dysfunction. So the observation is: the body is just doing what it's supposed to do...responding to the invader whatever it may be... but the bystander (the blood vessel) suffers from the result and the three processes now within the arteries were initially directed at the gluten. This can be one of the mechanisms causing idiopathic cardiomyopathy now shown to be reversible with a gluten-free diet in those who are sensitive.^[1]

This may extend to other cardiovascular diseases including coronary heart disease, myocardial infarction, congestive heart failure, cardiac arrhythmias, and probably others that haven't yet been investigated.^[1]

Dr. Houston recommends the DASH-2 Diet which is high potassium, low sodium, high magnesium, lots of vegetables, some fruit depending on your glycemic index, minimal to no grains, minimal to no dairy and lots of fiber...plus resistance and aerobic exercise. He finds by a prospective clinical trial that close to 70% of patients following nutrition and exercise have a reduction in either CV disease or other diseases.^[1,6] See references for a list of his outstanding books.

Aristo, Vojdani, PhD, MSc, MT^[7] a neurobiologist in the field of immunology research (40 years) who focuses on the role of environment and environmental factors, such as toxic chemicals, infections, dietary proteins and peptides in complex diseases, how mercury or aluminum might be affecting our brain, how food sensitivity such as gluten might be affecting us by complex diseases. Dr. Vojdani is CEO and Technical Director of Immunosciences Lab, Chief Scientific Advisor at Cyrex Laboratories and has published over 120 peer-reviewed articles in scientific journals. Dr. Vojdani identified for the Summit reliable testing for gluten and other sensitivities involved with autoimmunity. Welcome news is that the salivary antibody test for gluten sensitivity and celiac disease detection at an early stage is reliable and should be done in children sooner rather than later so preventive measures can begin before damage is done to the intestinal villi. By age one, children can be tested (by saliva) for antibodies. Abundant reports of interest by Dr. Vojdani can be found at Immuno-science website. Start with this report in *Townsend Letter, Oct. 2014 Blood-Brain Barrier Damage and Neuro-autoimmunity*
<http://tinyurl.com/ojcyf2d>

Unfortunately, space is limited and this overview has barely scratched the surface of this very extensive topic, but hopefully serves as incentive for independent research.

Take away message: Modern grains are not health foods. Gluten found in many grains is not digestible by humans. If you are treating for any of the multiple, chronic ailment symptoms listed or mentioned in the online reference link, and if you haven't been advised at the very least to go on a grain-free diet, that would be the initial starting point. Ideally, the appropriate tests as offered by Dr. Vojdani's labs would be done first to rule out genetic connections to various antibodies in grains, dairy and other reactive foods or other food allergy type reactions. Note that often it takes 6 to 12 months before significant symptom improvement is noticed with a grain-free diet that includes sprouted wheat as well so don't get discouraged if results take time, and avoid indiscretions during this period which will set you back. No way around it. Knowledge is Power.

Note: An expanded version of this report (same title) is posted at the Afibbers Forum along with an extended resource list for expert quotes including that of well-known insulin and leptin-resistance pioneer, Ron Rosedale, MD, (*Insulin and Its Metabolic Effects (1999)*). Dr. Rosedale had helped patients recognize the detrimental effects of consuming high-glycemic foods many years before publication of his Insulin report and book, *The Rosedale Diet* (© 2004) based on his clinical experiences and review of over 10,000 medical journal reports.

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