THE AFIB REPORT

Your Premier Information Resource for Lone Atrial Fibrillation!

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In this issue we continue Patrick Chambers fascinating review of the vital role of magnesium and potassium, not only in the prevention of atrial fibrillation, but indeed in the maintenance of overall good health. It is ironic that a quarter of the US population is deficient in magnesium and that over 50,000 annual deaths from coronary heart disease are directly attributable to a magnesium deficiency. Supplementation is pretty well a must for most of us in order to get the recommended 400 mg/day of elemental magnesium. Make sure to use the right supplement though; the common form of magnesium oxide is next to useless.

Also in this issue we welcome Jackie Burgess as a new guest author. Her article "Digestive Wellness – What You Need to Know" is an essential read for anyone striving for optimum digestion – an important component in afib management.

The evaluation of responses from our 5th LAF survey (LAFS V) is proceeding well and we now bring you the first results. The overall sample size of 341 lone afibbers makes, I believe, LAFS V the largest survey of its kind ever done. Thus the findings can be regarded with considerable confidence that they are representative of the overall population of lone afibbers. Among our first observations:

- *The average (median) age at diagnosis for all types of afib (adrenergic, mixed, vagal and permanent) is 48 years with a range of 8 to 80 years. Women tend to be somewhat older at diagnosis, particular in the vagal type. Well over 50% of all afibbers were between the ages of 40 and 55 years when first diagnosed.*

- *50% of all afibbers experienced 3 or fewer episodes during the first year. However, the number of episodes was 2 to 4 times higher among afibbers who took antiarrhythmics or beta-blockers regularly during the first year than among those who did not use drugs.*

- *About 20% of all afibbers were females. This varied according to type though. Women were most likely to have the mixed variety (27.8% female component) and least likely to have the vagal variety (14.4% female component).*

In the next installment of the survey evaluation we will look at general health status and episode characteristics and severity.

Just a reminder - if you haven't already done so, don't forget to get your copy of my recent book "Lone Atrial Fibrillation: Towards A Cure" at www.afibbers.org/lafbook.htm - it provides a wealth of information on dealing with LAF.

Enjoy!

Wishing you lots of sinus rhythm,
Hans Larsen

Evaluation of Survey Results

Demographics
A total of 341 afibbers (40 adrenergic, 108 mixed, 146 vagal, and 47 permanent) have now answered questions regarding their present age, their age at diagnosis, and their gender. A total of 9 afibbers with underlying heart disease also participated in the survey. Their responses, however, will be treated separately.

Present Age
The present age of participants was as follows:

- Adrenergic 55.0 (mean) 53 (median) range: 25-83 years
- Mixed 54.5 (mean) 56 (median) range: 31-73 years
- Vagal 53.0 (mean) 54 (median) range: 19-74 years
- Permanent 57.7 (mean) 57 (median) range: 34-82 years
- Total paroxysmal 53.8 (mean) 54 (median) range: 19-83 years
- All participants 54.3 (mean) 55 (median) range: 19-83 years

The difference in present age between the paroxysmal group and the permanent group was statistically significant as was the difference in age between vagal and permanent afibbers.

Age at Diagnosis
The average age at diagnosis was as follows:

- Adrenergic 49.6 (mean) 48 (median) range: 22-80 years
- Mixed 46.3 (mean) 48 (median) range: 17-71 years
- Vagal 46.6 (mean) 48 (median) range: 14-73 years
- Permanent 48.1 (mean) 48 (median) range: 8-75 years
- Total paroxysmal 46.9 (mean) 48 (median) range: 14-80 years
- All participants 47.0 (mean) 48 (median) range: 8-80 years

There were no statistically significant differences in age at diagnosis. The finding that the average age at diagnosis among 341 lone afibbers is 47 years (median 48 years) should hopefully put a serious dent in the myth that lone atrial fibrillation is an “old age” disease. It clearly is not – quite the contrary, it tends to strike men and women at their most productive age. As a matter of fact, well over 50% of all afibbers were between the ages of 40 and 55 years when first diagnosed and over 16% were at or below the age of 35 when first diagnosed. Only 6.5% were 65 years of age or older when first diagnosed.

Years of Afib
The average number of years that the survey respondents had experienced LAF was as follows:

- Adrenergic 5.4 (mean) 3 (median) range: 1-15 years
- Mixed 8.2 (mean) 7 (median) range: 1-39 years
- Vagal 6.5 (mean) 4 (median) range: 1-40 years
- Permanent 9.0 (mean) 5 (median) range: 1-65 years
- Total paroxysmal 7.0 (mean) 5 (median) range: 1-40 years
- All participants 7.3 (mean) 5 (median) range: 1-65 years

Although the years of afib and the present age of respondents are not really that indicative of anything other than as a measure of how long the afibber had suffered with afib before finding www.afibbers.org and participating in the survey, it is interesting to note that it is indeed possible to live with LAF for a very long time – up to 40 years for paroxysmal afibbers and 65 years for permanent. As a matter of fact, 25% of paroxysmal afibbers had lived with afib for 10 years or more. I am not sure whether this is good news or bad. Living with this condition for 65

years sure seems like a very, very long time indeed! But, unless we come up with a solution that is exactly what some of us may just have to do.

Number of Episodes During First Year
This question was answered by 141 afibbers (15 adrenergic, 51 mixed, 67 vagal and 8 permanent).

- Adrenergic 4.7 (mean) 3.0 (median) range: 1-18
- Mixed 15.5 (mean) 3.0 (median) range: 1-225
- Vagal 15.3 (mean) 4.0 (median) range: 1-365
- Permanent 13.0 (mean) 7.5 (median) range: 1-52
- Total paroxysmal 14.2 (mean) 3.0 (median) range: 1-365
- All participants 14.1 (mean) 3.0 (median) range: 1-365

Clearly there is a vast variation in the number of episodes during the first year of afib. However, 50% of all afibbers experienced 3 or fewer episodes during the first year while 68% had 6 or fewer. There was no statistically significant difference between the number of first-year episodes for the various types of afib.

There was, however, a very significant correlation between the number of episodes experienced during the first year and the use of pharmaceutical drugs during the first year. Sixty-seven respondents (49%) had not taken any drugs (antiarrhythmics or beta-blockers) while the remaining 71 respondents (51%) had taken drugs. The non-drug takers experienced an average (mean) number of episodes of 4.7 during the year (median =2) while drug takers experienced an average (mean) number of episodes of 13.9 (median =6). This difference was highly significant ($p<0.0001$). The detrimental effects of drugs applied to all types of paroxysmal afib. There were too few permanent responses to draw a valid conclusion.

- Adrenergic 27% no drugs mean 1.8 episodes (median 2) range: 1-3
- Adrenergic 73% drugs mean 5.7 episodes (median 4) range: 1-18

- Mixed 63% no drugs mean 4.6 episodes (median 2) range: 1-50
- Mixed 37% drugs mean 18 episodes (median 8) range: 1-72

- Vagal 44% no drugs mean 5.5 episodes (median 2) range: 1-57
- Vagal 56% drugs mean 13.5 episodes (median 5) range: 1-52

All the differences in episode frequency between drug and non-drug users were statistically highly significant.

It is clear that taking antiarrhythmics or beta-blockers during the first year of afib is detrimental. This could well be because the first choice in drugs is often digoxin or sotalol or a beta-blocker. All are directly detrimental to vagal afibbers and of dubious, if any, value for mixed and adrenergic afibbers. So based on this statistically highly significant finding the conclusion is to stay away from antiarrhythmics and beta-blockers for at least the first year of your afib career unless your heart rate goes so high during an episode that you need to slow it down by taking a calcium channel blocker or a beta-blocker during the actual episode only.

Conversion to Permanent Afib
This was clearly a difficult question and only 15 permanent afibbers provided a complete or partial answer. The majority (73%) had developed permanent afib from the paroxysmal form while the remaining 27% (4 respondents) had been diagnosed with permanent afib when the condition was first discovered. Only 7 of the 11 respondents who had progressed to permanent were aware of what kind of paroxysmal afib they had. The majority (57%) believed they had progressed from mixed while the remaining 43% (3 respondents) believed they had progressed from vagal. The small sample size clearly makes any conclusions on this aspect very tenuous to say the least. However, it does appear that vagal afibbers can indeed become permanent. It took an average 3 years (range 1-8 years) for the condition to turn permanent, but again these numbers are based on a very small sample size (11 respondents) so should be taken with a large grain of salt.

Gender Differences
The majority (80.4%) of all afibbers were male. There was a statistically non-significant trend for female afibbers to be diagnosed somewhat later than males.

- Adrenergic (17.5% female) Median age at diagnosis – male=49 female=47
- Mixed (27.8% female) Median age at diagnosis – male=46 female=53
- Vagal (14.4% female) Median age at diagnosis – male=46 female=54
- Permanent (19.1%female) Median age at diagnosis – male=47 female=60
- All afibbers (19.6%female) Median age at diagnosis – male=53 female=47

Only the 8-year difference in median age at diagnosis between male and female vagal afibbers was statistically significant ($p<0.02$). Is it possible that the delay in females developing vagal afib could be due to a protective effect of estrogen? Tantalizing possibility, but as far as I know, pure speculation on my part.

Magnesium & Potassium in Lone Atrial Fibrillation – Part II

by Patrick Chambers, MD

MAGNESIUM AND HORMONES

Cyclic AMP/cGMP and Taurine
Cyclic AMP (cAMP) and cyclic GMP (cGMP) act as "second messengers" for certain hormones. They function as kind of localized intracellular hormones. Insulin and catecholamines function via receptors (beta) on target cell membranes, which involve cAMP. On the other hand, cholinergic receptor activity (predominantly the parasympathetic nervous system or PNS) involves cGMP. Adenylate cyclase (for cAMP production) requires Mg, whereas guanylate cyclase (for cGMP production) requires Ca(13). Consequently, in Mg deficiency the intracellular cAMP/cGMP ratio, normally between 10 and 100 to 1, is reversed(52). This may partially explain the insulin receptor resistance (low cAMP) seen in impaired glucose tolerance associated with Mg deficiency. cGMP also mediates the effects of ANP in target cells, i.e., enhanced natriuresis(31). This may be another reason why VMAF episodes (enhanced cholinergic receptor activity) and/or LAF episodes due to Mg deficiency, both associated with high cGMP, seem to revert to NSR (normal sinus rhythm) more spontaneously than do adrenergically mediated episodes(95).

Interestingly, hyperinsulinism tends to maintain this reduction in cAMP(13). Jean Durlach, M.D., Editor-in-Chief, *Magnesium Research,* President of the International Society for the Development of Research on Magnesium, and author of Magnesium in Clinical Practice, suggests that taurine may affect this turnaround (less cGMP and more cAMP). Catecholamines and insulin favor cellular influx of taurine. Taurine is a powerful membrane stabilizer. It also chelates Ca, a Mg antagonist, facilitates maintenance of intracellular K and opposes the undesirable cellular effects of insulin and catecholamines(13). Taurine plays an important role in Mg deficiency. Ingestion of monosodium glutamate (MSG) can lead to taurine deficiency, since glutamate competes with cysteine (required to make taurine) for cellular uptake(46).

Glutamate and Aspartate
Glutamate and aspartate are neurotransmitters. Specifically glutamate determines HR through the SA node. More glutamate translates to higher vagal tone. MSG is also a common trigger of AF and premature atrial complexes (PACs) for many LAFers(72). NMDA (N-Methyl-D-Aspartate) receptors are located on neurons and are associated with the Ca channel(42). When glutamate or aspartate (aspartame in NutraSweet is metabolized to aspartate) attaches to the NMDA receptor, it triggers the flow of sodium (Na) and calcium (Ca) ions into the neuron, and an outflow of potassium (K), firing the neuron. ATP pumps are required to return the ions and restore the resting state. The Ca channel is blocked by magnesium. This helps maintain membrane potentials near resting value. If the repolarized or resting state cannot be maintained, e.g., hypoglycemia, defective pump (as in Mg deficiency), then the neuron fires and the channels open. This pump failure gradually allows excessive calcium/sodium build up inside the cell, which is eventually lethal(43,44). Furthermore, ATP pumps

are required not only to return the ions but also to remove the glutamate and return it to the neuron (neuronal reuptake). Glutamate is then converted into glutamine, another process that requires ATP. That is a total of three separate ATP and Mg requiring steps. Free radicals further impede this(45). Mg has a circadian excretory rhythm, with lower blood levels and higher excretion occurring at night(56,92)). During this period, extracellular Mg is at its diurnal nadir and vagal tone is at its zenith. Undoubtedly the compounding effects of concomitant Mg deficiency are a powerful stimulant for nighttime VMAF. Add the excitatory properties of glutamate to the recipe and the soil becomes very fertile for LAF. For these reasons, MSG and even mild gastroesophageal reflux disease (GERD) make dinner out a risky proposition for many LAFers, especially VMAFers (see discussion on GERD and postprandial reactive hypoglycemia below).

MAGNESIUM AND THE DEFECTIVE SUBSTRATE

Autonomic Nervous System

Mg is required for activity by the cholinesterase enzymes(13). One of these, acetyl cholinesterase degrades acetylcholine, the neurotransmitter substance for the PNS and for the first part of the sympathetic nervous system (SNS), specifically the nicotinic receptors of the SNS. In fact, deficiency of magnesium and excess calcium both increase the release of acetylcholine. Deficiency of either magnesium or calcium prolongs the effect of acetylcholine(58). Mg deficiency translates to enhanced vagal tone further augmented by too much or too little Ca.

Catecholamine-O-methyltransferase (COMT) and monoamine oxidase (MAO) catabolize (break down) norepinephrine (NE), the neurotransmitter for the rest of the SNS. However, unlike acetylcholine but like glutamate, neuronal reuptake of discharged norepinephrine is a major mechanism for terminating sympathetic neurotransmission (see glutamate discussion above). MAO catabolizes this NE, while COMT is more active in catabolizing extracellular circulating (humoral) catecholamines secreted by the adrenal gland(30). Both are part of the SNS. COMT requires Mg as a cofactor(28,29). Neuronal reuptake also requires ATP (and Mg). Low Mg translates to higher sympathetic tone(105). These enzymatic shortfalls might produce an exaggerated response of either the PNS or the SNS at transition or crossover points, a time when many VMAF episodes arise, e.g., lying down or bending over. The neurotransmitter substance or hormone secreted on each occasion is not degraded or removed, resulting in a prolonged over response. For example, cocaine blocks dopamine reuptake, leaving more dopamine in the synaptic cleft, which results in over stimulation of the D2 receptors (causing schizophrenic episodes)(106).

Sexual activity triggers some episodes for many afibbers(72). In addition to MAO breakdown of dopamine within neurons (neuronal reuptake) COMT breaks down circulating dopamine, an important hormone produced at this time. The dopamine no doubt triggers automaticity (associated with beta-1 receptors) in ectopic foci with a resulting increase in PACs (see EP discussion below)(107). The over responding vagus causes a shortening of the AERP. Mg deficiency in this scenario (independent of K) may be causative in bedtime episodes and even some more typically adrenergic episodes.

GERD

The “alkaline tide” precedes the start of any meal. This is caused by gastric cell secretion of H and Cl into the lumen for digestion of food and simultaneous extrusion of K and HCO3 into the blood. This more alkaline blood causes bicarbonaturia (HCO3 in urine) to lower this pH (blood pH is tightly controlled between 7.35 and 7.45). Unfortunately, K(54) as well as Mg(104) are cations lost in the urine (kaliuria and magnesuria respectively) along with the anion HCO3. This lowers blood K, although not necessarily below lower limit of normal. Furthermore, there is evidence that high vagal tone may sustain basal gastric acid hypersecretion in some persons and temporary hypersecretion during stress in others(49). Some cases of GERD (gastroesophageal reflux disease) and non-ulcer dyspepsia (NUD) probably result in transiently low K via the constant steady alkaline state (in plasma) that accompanies the slightly hyperacidic state (in the stomach). The K/H pump also rectifies this increase in blood pH. H goes into the blood and K comes into the cells. Again this requires cardiac muscle cells to maintain their intracellular K concentration against a greater gradient. Also, greater concentration of K within renal tubule cells contributes to increased renal secretion of K into urine. Normally the concentration of K within heart muscle cells is 150 millimoles/liter (v. four mm/l outside the cell), a considerable gradient (almost 40:1) to maintain(9). Ingested protein stimulates more HCl secretion (and a stronger alkaline tide and greater kaliuria).

Other suggested mechanisms for GERD related episodes of LAF include stimulation via irritation of the vagus nerve during episodes of reflux and/or gastric distention. Some VMAFers associate their episodes with GERD(72). Curiously, many of them prefer to sleep on their right side (right lateral decubitus position). Vagal tone is increased while in this position(67). This is because the heart is slightly higher (v. the left side position) relative to the carotid baroreceptor. This pressure receptor in the neck senses more hydrostatic pressure and signals the vagus nerve to increase tone (bad for a VMAFer). However, the preference may be because this position promotes gastric emptying (our stomachs pass their contents to the right and dump them into the duodenum) and possible relief for a GERDer.

Dysinsulinism

Those with impaired glucose metabolism hyper respond with insulin (produced by the beta cells of the pancreatic islets) to a carbohydrate meal (target cells are insulin resistant). The ensuing hypoglycemia (low blood glucose) stimulates release of glucagon (produced by alpha cells of the pancreatic islets) and catecholamines with consequent hyperglycemia (high blood glucose) with a kind of yo-yo effect(72). Catecholamines but especially glucagon stimulate glycogenolysis (breakdown of glycogen, the storage form of glucose) and gluconeogenesis (release of glucose from cells that store glycogen), most notably from the liver. Gluconeogenesis involves enolase and magnesium is required as a cofactor(91). In fact five of the other eight steps in gluconeogenesis also require Mg(94). It appears that Mg is critical to the proper function of glucagon and catecholamines in this area.

There is an epidemic of overweight/obesity in the Western world, especially here in America. Syndrome X (or Metabolic Syndrome = includes high blood pressure, obesity, diabetes, high blood insulin and triglyceride levels) represents the far end of the spectrum of this disorder of carbohydrate metabolism. Useful laboratory tests include serum hemoglobin A1C, which will detect large swings in blood glucose levels over the preceding three months. Fasting blood glucose and then an OGTT (oral glucose tolerance test) are the best tests to diagnose impaired glucose tolerance and diabetes mellitus. Mg deficiency plays an important role in this process (see insulin section above).

Postprandial (after a meal) reactive hypoglycemia (PRH) is defined as low blood sugar (less than 3.3 mmol = 60 gm/dl) concurrent with symptoms (dizziness, depression, sweating, weakness, hunger, anxiety)(82,83,89). LAF has recently been added to this list(84,85,86). Although the oral glucose tolerance test (OGTT) is not abnormal (88,89), a characteristic pattern is often seen in PRH. The release of insulin is sluggish and the insulin peak delayed with respect to the peak value for blood glucose(72,98,99). These flat curves are associated with excessive vagal tone(29,72,100). Instead of insulin resistance, as seen in diabetes, there is insulin hypersensitivity in 50 to 70% of those with PRH. In addition to insulin the body secretes other hormones, such as glucagon like peptide (GLP), from GI tract endocrine cells that stimulate insulin synthesis and secretion(96). GLP-1 levels in those with PRH average 10 times those of normals(89) and it is this hormone that is felt to be responsible for this insulin hypersensitivity. However, this alone is insufficient for a diagnosis of PRH(89). Glucagon dysfunction is a necessary ingredient(97,98). Mg deficiency and its negative effect on gluconeogenesis (glucagon dysfunction) in combination with insulin hypersensitivity may well explain postprandial reactive hypoglycemia in some individuals (see glucagon section above). Furthermore, many with PRH are very lean or women with moderate lower body weight(89) with increased HDL cholesterol. These lean individuals with excessive vagal tone are precisely those that pursue endurance activities. VMAF is increased in endurance athletes (101). Studies comparing the effect of blood glucose on right and left atrial refractory periods reveal these to be shortest under hypoglycemia in the left atrium and longest under normo or hyperglycemia in the right atrium(102) (see EP discussion below). Triggering PACs in LAF typically arise in the left atrium(72,75).

Magnesium deficiency also causes release of catecholamines(12). Perhaps this is partially because Mg deficiency causes glucagon dysfunction, which stimulates catecholamine release to cover the glucose shortfall(89). Catecholamines stimulate release of fatty acids that complex with blood Mg, further aggravating the Mg shortfall(60,62,63). Insulin and catecholamines both cause intracellular migration of K and decrease serum K(12). Consequently, catecholamines (and insulin) cause a greater gradient, not only promoting steady leakage of cardiac muscle cell K to blood but also facilitating renal K excretion. Increased K within renal tubule cells stimulates more secretion of K into urine. Hypokalemia, hypoglycemia induced or otherwise, is highly arrhythmogenic.

Hyperthyroidism
Hypomagnesemia is a common problem in hyperthyroid patients. Thyroid hormones induce shifting of magnesium into the cells. Excessive levels of T3/T4 consequently cause urinary Mg wasting(13). This Mg deficiency again leads to low intracellular K (defective Na/K ATPase pump and unstable membrane), as evidenced by the fact that 14% of hyperthyroids have AF(26) and 10% have hypokalemic periodic paralysis(27).

Dehydration
Dehydration (via the renin angiotensin aldosterone system (RAAS)) stimulates release of aldosterone to reabsorb Na and with it water. In exchange, K is secreted/excreted, thereby lowering serum K. Aldosterone receptors are also present in colon and skin. In the distal colon, aldosterone enhances active Na absorption and K (and Mg) excretion/secretion (via pump and channel)(50). Exercise induced dehydration stimulates aldosterone release, causing increased loss of K and Mg in sweat(65,66). Excessive exercise also stimulates secretion of ADH, catecholamines, TSH, cortisol and aldosterone, all of which cause urinary Mg wasting(57) (see above).

Left-Handedness
Although we get the word sinister from the Latin word for left, most left-handers do not consider themselves defective. However, many left-handers have increased need of Mg. Digoxin is a steroidal glycoside often prescribed for those with LAF. It is a potent inhibitor of the Na/K pump and is contraindicated especially in those with VMAF(93,95). Curiously it also produced endogenously by the hypothalamus via the isoprenoid pathway(92). This causes an increase in intracellular Ca and a decrease in the functional availability of Mg. The resulting atrial conduction deficit, increased incidence of atrial fibrillation and stroke has been strongly associated with right hemisphere dominant (left-handed) individuals in whom the isoprenoid pathway is often up regulated (v. right-handers)(92).

*******For references please see Part I*******

Digestive Wellness – What You Need to Know

by Jackie Burgess, RDH

It is well recognized by holistic physicians that proper digestion, absorption, assimilation, and elimination of foods is the key to health. No matter how good, pure, or complete the foods and nutrients consumed are, unless they are broken down so the body can absorb and assimilate them into cells, a person will suffer from malnutrition and enjoy less than optimal health. So says Dr. Jeffrey Bland, noted lecturer and author on health and nutrition. Over 20 million Americans suffer from various digestive disorders which impair their nutrition, he reports[1].

Cardiologist, Stephen Sinatra says, “Sad but true, there are millions of people out there—many of them healthcare providers—who don’t fully appreciate the impact that digestive problems can have on the major diseases that afflict us today. Mainstream medicine, in fact, is ten years behind the times when it comes to understanding how interrelated the body’s organs and functions really are.”

Poor digestion can set you up for many diseases. Says Dr. Sinatra, “Indigestion is a little recognized symptom of impending heart problems. One of its symptoms, excessive gas, causes bowel distension which presses against organs that have a direct connection with your heart. A heavy meal may shunt so much blood over to the stomach that a person with coronary blockages can be more prone to angina following such a digestive overload[2].”

A thorough understanding of the functional aspects of digestion is best accomplished by reading any of the several referenced books because the topic is so extensive. This article discusses less than optimal digestive functioning and points out its symptoms and consequences. Its goal is to heighten awareness that many chronic conditions or diseases have origins in faulty digestion, absorption and, ultimately, elimination of toxic waste.

Scientific evidence exists to support that allergies, all types of arthritis, asthma, chronic fatigue, irritable bowel syndrome, eczema, psoriasis, and migraines, to name a few, have origins in digestive disorders[3,4].

In children, the digestive abuses frequently manifest as allergies and conditions such as colic and ear infections. As we mature, our body adapts and does the best it can with the lifestyle choices we give it.

Eventually, when poor choices are made over cumulative years, conditions develop for which mainstream medicine has little to offer in the way of cure or reversal, but rather we are placated with masking drugs—inhalers for asthma, antihistamines for allergies and steroids and antibiotics when all else fails. These remedies are only cover-ups; they are seldom a cure.

Major contributing factors to digestive ailments are:

- "Leaky gut" syndrome,
- Inadequate digestive enzymes,
- Inadequate stomach acid,
- Imbalance of intestinal flora,
- Inadequate dietary fiber intake,
- Environmental influences such as stress. In fact, along with the skin, the digestive system is the most common expression site of stress-related illnesses[5].

"Leaky Gut" Syndrome
In the process of digestion, food not broken down adequately by chewing, acid in the stomach and digestive enzymes added along the way, arrives in large molecules in the small intestine where absorption of nutrients takes place. The integrity of the intestinal wall can become damaged by incompletely digested food lying in a stagnant state where it putrefies and produces toxins which create intestinal wall inflammation. This inflammation creates tiny tears in the intestinal wall. A compromised intestinal wall allows large, partially digested molecules of fats, proteins and carbohydrates to flow through to the blood stream.

The immune system identifies these large particles as "foreign invaders" and signals for the manufacture of antibodies to the invader-antigens.

Over time, repeated "leaking" of the same food molecule through the intestinal wall causes an allergic reaction often manifesting as sneezing, flushing, coughing, hives, and headache. This is termed the "leaky gut syndrome." People commonly have these reactions to many foods but fail to make the connection.

It's interesting to observe the frequency of people in restaurants having reactions like coughing, sneezing and wiping eyes after or during a meal. The problem is exacerbated by habitually eating the same foods daily, weekly, yearly.

In children, food sensitivity can be signaled by ADD (attention deficit disorder), behavior problems and recurring ear problems[6].

The path to digestive wellness begins with thorough chewing followed by adequate stomach acid production along with enzymes required to breakdown protein, fat and starch, and abundant "good" intestinal bacteria.

Digestive Enzyme Deficiency
When your body doesn't produce enough enzymes to break down food for proper digestion, instead of nourishing, this food poisons your body. It stagnates in the bowel and becomes toxic—the origin of many illnesses and disease.

Our lifestyles of eating too many cooked, microwaved and irradiated foods kill live enzymes essential to health. Most everyone can benefit from supplemental enzymes; and, especially those over age 50, since this is about the time digestive systems begin to fail to some degree.
Supplementation helps spare the pancreas from overwork.

Some reliable brands to take at the beginning of each meal include[7]:

- Essential Enzymes by Source Naturals
- Mega-Zyme (pancreatic enzymes) and Pro-Gest-Aid (mixed enzymes) by Enzymatic Therapies
- Super Enzymes by NOW
- Cotazym or Zypan (prescription medications).

There is some indication that pancreatic enzymes (especially amylase) work better if the content of the capsule is sprinkled on the food prior to eating rather than swallowing the capsule whole.

Adequate Stomach Acid

One common cause of poor digestion is an inadequate level of stomach acid. Some symptoms of this condition are:

- Burping,
- Fullness for an extended time after meals,
- Bloating,
- Poor appetite,
- Stomach upsets easily,
- History of constipation,
- Food allergies,
- Weak, brittle fingernails,
- Rosacea,
- Hair loss in women.

An adequate level of stomach acid (gastric acid or hydrochloric acid [HCl]) is essential. The digestive enzyme, pepsin, is activated by HCl. Pepsin breaks down proteins and thereby releases vitamins, minerals and other nutrients into a "digestive soup" which eventually will be absorbed into the blood stream.

Gastric acid (HCl) is a barrier against infection. Bacteria, viruses and fungi inhaled or ingested are normally destroyed in the stomach. It is the first line of defense against food poisoning. Low stomach acid allows bad bacteria to flourish and interfere with nutrient absorption[8].

With all the ads we see on TV promoting antacids to "neutralize excess stomach acid," it may seem hard to believe that too little acid may be as big a problem as too much. But, symptoms of hypo-acidity often mimic hyper-acidity. Often, the very problem we try to correct, gas and bloating after meals is actually the result of too little HCl and is compounded by what we do—take antacids[9].

Disease conditions caused by low stomach acid include asthma, chronic hepatitis, diabetes, eczema, osteoporosis, thyroid disorders, gallbladder disease, vitiligo, various rheumatic conditions including rheumatoid, lupus, Sjögren's and weak adrenals[10].

The negative effects of low stomach acid production can be ameliorated by taking betaine hydrochloride capsules with each meal.

Interesting dental note: Hypochlorhydria (low stomach acid) may result in reduced absorption of calcium, magnesium, copper, folic acid and other nutrients related to osteoporosis prevention. Stomach acid production was measured in 79 people aged 16 to 53 years. Those with evidence of alveolar bone loss produced less than half as much HCl as those without alveolar bone loss[11].

Imbalance of Intestinal Flora

There are 400 types of bacteria in the digestive system numbering 100 trillion and weighing about four pounds. They all thrive together in symbiotic or antagonistic relationships and manufacture substances that raise and lower our risk of disease, cancer, immune competence, nutritional status and rate of aging.

Some cause acute or chronic illness and others offer protective and nutritive properties. The latter, the friendly bacteria, are known as intestinal flora or "probiotics" meaning "healthful to life." It is these bacteria we want to nurture because they, in turn help keep us well[12]. Over-consumption of colas, coffee and alcohol disturbs the acid-base relationship in the bowel and this can also lead to an overgrowth of bad bacteria. Bad bacteria discharge nasty toxins, many of which are carcinogenic. A toxic bowel can initiate a chain of reactions resulting in digestive problems which ultimately lead to immune dysfunction, allergies, skin rashes, osteoporosis, high cholesterol, chronic fatigue, vitamin deficiencies, bad breath and cancer of the colon[13].

Good bacteria help with the absorption of nutrients and are involved in the manufacture of several vitamins. The balance is disrupted by processed foods, excess sugar and carbohydrates, and flour products along with a diet heavy in red meat and saturated fats. Too much animal protein putrefies in the bowel. It actually breaks down into carcinogens inducing the worst cancer-producing chemicals around—phenolic compounds[14].

Antibiotics wipe out all intestinal bacteria—good and bad—and allow an overgrowth of the yeast, Candida. Steroids (like Prednisone), birth control pills and chlorinated water seriously impact the friendly bacteria in a negative manner[15].

Probiotic Supplements
Supplementation with friendly bacteria, called probiotics, helps insure the proper balance in the intestinal flora. Probiotics produce natural chemicals that kill harmful bacteria and prevent many illnesses and fatal diseases. A popular phrase, "death begins in the colon" stems from these facts about bowel physiology. The bowel must have more good bacteria than bad and because of lifestyles and aging, everyone can benefit from daily probiotic supplements[16].

Some reliable brands are:

- DDS-Plus by UAS laboratories. (Acidophilus, Bifidus, FOS – Non-dairy. Refrigerate) and DDS-Junior
- Kyo-Dophilus, Flora Balance, and Healthy Trinity by Natren.

Take them upon arising and between meals so they aren't subjected to excessive acidity from meal activity.

It is wise, also, to take probiotics whenever taking antibiotics is unavoidable. Obviously, take at different times from the antibiotic. In cases of diarrhea, it is also helpful to take additional doses of probiotics.

Friendly bacteria label names include: for infants and toddlers, bifido bacteria infantis; for children and adults—lactobacillus acidophilus, bifido bacterium bifidus longum, lactobacillus bulgaricus in a base of FOS (fructo-oligosaccharides) a carbohydrate to support bacterial proliferation. FOS is a prebiotic that fertilizes probiotics. Food prebiotics are in barley, wheat, rye, tomato, garlic, onion, bananas and whey.

Just think of gardening. Adding pre-and probiotics to your diet is just fertilizing the good bacteria so they grow healthy and crowd out the bad....just as in fertilizing the lawn to crowd out weeds.

Inadequate Dietary Fiber
The longer between bowel movements, the longer toxins and bile acids accumulate and irritate the lining of the colon causing health problems of the colon such as constipation, appendicitis, diarrhea, diverticular disease, Crohn's disease, colitis, polyps, colon cancer, irritable bowel syndrome, parasites and hemorrhoids[17].

People on good diets with plenty of water have one to two bowel movements a day; and the transit time (from when first swallowed to exit) should be from 18 to 36 hours. The book, "*Digestive Wellness"*, by Elizabeth Lipski, describes how to check transit time and offers remedies for improvement[3].

The recommended daily intake of dietary fiber is between 25 and 30 grams. Pearled barley, beans, oat bran, prunes, tomatoes, and raspberries are good sources of dietary fiber. However, it can be difficult to reach the recommended daily intake through diet alone so a fiber supplement may be necessary.

An economical fiber, such as plain whole psyllium husk sold in bulk quantities in health food stores for about $8 for 12 ounces, mixes easily into water, soup or juice. Follow the directions and add to the diet very gradually to become accustomed to the effects. Remember, soluble fiber such as found in oat bran and psyllium binds up cholesterol and sweeps it out of the body.

Conclusion

The digestive process tends to become impaired with age. Poor digestion is associated with many common health problems and many afibbers have reported a connection between digestive problems and the initiation of an afib episode. Healthy digestion can be ensured by proper dietary choices and by judicious supplementation to correct deficiencies in digestive enzymes, stomach acid production, dietary fiber intake, and intestinal flora balance.

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