

THE AFIB REPORT
Your Premier Information Resource for Lone Atrial Fibrillation
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VIRTUAL LAF CONFERENCE

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SUBJECT: HYPOGLYCEMIA

Fellow fibbers,

The Conference Room has been a bit lifeless recently. Has the hormone connection been declared unofficially dead? As a pathologist, I'm not normally accustomed to bringing life to the situation, however,.....

The vast majority of previous posts addressing the probable hormone connection to LAF have centered on the adrenal gland, especially aldosterone. Given the wide spectrum of conditions associated with LAF, the "everybody's different" mentality and the seeming difficulty in moving toward a unifying theory for LAF, I think it's time to broaden our horizons. Make no bones about it, this is another post on one of the many faces of Mg.

In Hans' LAF survey 25% of all respondents reported hypoglycemia and another 24% had symptoms of hyoglycemia (p63 of his book). Not many people have mentioned glucagon, a hormone produced by the pancreas. Few people actually understand its role in maintaining normoglycemia (normal blood glucose levels). Insulin and glucagon (and to a lesser extent catecholamines) counterregulate each other in maintaining this glucose balance. Glucagon causes glycogenolysis and gluconeogenesis. This is fancy talk for breaking down glycogen, the storage form of glucose (mostly in the liver), and producing glucose to be secreted by the cell into the blood. Magnesium has been strongly associated with chronic fatigue syndrome (CFS) and some have suggested that at least part of the reason for this is the fact that Mg is required for 6 of the 9 enzymatic steps in glycolysis. Glycolysis is the metabolic pathway the body uses to generate energy from glucose. Hence, those with CFS due to Mg deficiency have less energy. However, the interesting thing about enzymes is that they are required to facilitate the reaction in both directions. Glycolysis is the opposite of gluconeogenesis. Therefore, in order to be effective in its job of gluconeogenesis glucagon requires Mg. This is a very enticing explanation for some forms hypoglycemia, perhaps the type experienced by many LAFers.

PC, MD v54

Fellow fibbers,

Guess you didn't like my glucagon post. Perhaps I can "sweeten" the pot.

Postprandial reactive hypoglycemia (PRH) = symptoms concurrently with low blood sugar (less than 3.3 mmol = 60 gm/dl).

The oral glucose tolerance test (OGTT) is not diagnostic but 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. See pp 63-64 of Hans' book in section on hypoglycemia where he states, "It is also possible, but purely speculative on my part, that a blunted glucose response could be associated with LAF".

Accordingly, I would like to second that suggestion via the following evidence lifted from the medical literature:

- 1) Insulin hypersensitivity is present in more than 50% of those with PRH.
- 2) High carbohydrate-low fat diet increases insulin sensitivity, and this pattern is frequently found in PRH patients.
- 3) Glucagon like peptide (GLP-1) is also significantly increased in most. GLP-1 (also known as incretin) controls blood glucose after a meal via stimulation of insulin secretion.
- 4) Insulin enhances epinephrine, norepinephrine and cortisol secretion in response to hypoglycemia.
- 5) Insulin sensitivity alone can induce hypoglycemia, since high values like those found in PRH are found in young, lean people who never report suffering PRH.
- 6) An increase in insulin sensitivity associated with a deficiency in glucagon secretion is the widely accepted explanation for hypoglycemia in the late postprandial phase.
- 7) Glucose production secondary to glucagon requires Mg (see above post).
- 8) Glucagon receptor activity is mediated by adenylate cyclase, which is Mg dependent.
- 9) This failure of glucagon induced glucose production in response to insulin induced hypoglycemia stimulates epinephrine secretion.
- 10) Insulin and catecholamines both stimulate cellular uptake of K.
- 11) Hypokalemia is highly arrhythmogenic.
- 12) LAF is a well known symptom of hypoglycemia.

Other pertinent particulars:

- 1) Very lean people or in women with moderate lower body overweight are prone to hypoglycemia.
- 2) Insulin sensitivity is twofold higher in follicular than in luteal phase. These women frequently describe hypoglycemic symptoms late in the morning.
- 3) Slimming increases insulin sensitivity.
- 4) PRH individuals have high HDL cholesterol.
- 5) Symptomatic hypoglycemia typically occurs after a sugar drink or alcohol while in the fasting state.

Conclusion: Many LAFers whose episodes are triggered by hypoglycemia have PRH, because they have insulin hypersensitivity due to increased GLP-1. They also have the required defective glucagon secretion and sensitivity due to Mg deficiency. This results in LAF because the insulin and reflexive catecholamine secretion result in lower blood K levels.

How many times have you wondered why that fat person walking down the street is AF free while you with a BMI (body mass index) well under 25 and high good cholesterol suffer from LAF?

For a more detailed discussion and recommendations for treatment
Diabetes & Metabolism (Paris)

2000, 26, 337-351

POSTPRANDIAL REACTIVE HYPOGLYCEMIA

J.F. BRUN, C. FEDOU, J. MERCIER

<http://www.alfediam.org/media/pdf/RevueBrunD&M5-2000.pdf>

PC, MD v54

PC---Since no one is jumping in here I'll throw in my two cents worth from a layman's point of view based on my personal experience with hypoglycemia. I've had hypoglycemia most of my life and was severe in earlier years. The cause was from living on sugar and even though I have a significantly different diet since then the insulin sensitivity still exists after many, many years of better diet.

I have believed for some time, but have never stated so, that the hypoglycemia is part of the equation. I am also tall and slender, 6-4, 190, with lots of good cholesterol. If I eat a very low carbohydrate diet my afib doesn't give me much trouble. If I so much as have a nice big piece of chocolate cake or pie after supper the afib is almost automatic.

Something strange that I have not got a handle on yet [still experimenting] is the more Mg or WW that I take the worse the afib problem. Of late I have been experimenting with K [lots of it] and no salt based on discussions from the last conference] with, what appears to be very good results. The lots of K appears, based on what you stated, ties in with the hypoglycemia thing.----Very interesting.

Ben

PC - I like both your topics and ask that you give me a bit of time to formulate a response. I have extensive experience with depleted adrenal function, hypoglycemia and insulin resistance. All of which have been corrected through nutritional methods.

Give me a day or two to logically present my observations rather than just tossing them on the page at random....

I definitely feel there is a connection and it is an important topic to review for afibbers.

I'll be back.

Jackie

Fellow fibbers,

An interesting aside on GLP-1 is that it is also an appetite suppressant. This may partly explain increased levels in very lean individuals. Research into its usage in weight loss programs is ongoing. It also inhibits gastric emptying, no doubt compounding appetite suppression by causing early satiety through gastric distention. This would probably be a bad thing for those with GERD (gastroesophageal reflux disease). All that sloshing around with the way forward compromised leads to a backup in the plumbing.

Anyone lean individuals out there with LAF episodes associated with possible hypoglycemia and/or GERD?

PC, MD v54

I'm afraid that I've got nothing particularly learned to proffer, but I would comment that I often feel thick-headed when I stand up during the hour or two after I've eaten my early evening meal... particularly if it had any carbs in it. I guess that fact might well put me in the camp of mild hypoglycemia??

Mike F.

PC

It is very interesting that GLP-1 is an appetite suppressant. All my life I have had to force myself to eat. I still do to some extent. But now I know if I do not eat I will get sick and induce AF.

Now, whilst the slimming world is looking for ways to increase GLP-1 is it possible that we should be looking for ways to decrease it. I know you mentioned Mg for the glucagon dysfunction, but I am one of the few who cannot tolerate it in supplemental form. I'm going to search further on this. Do you have any good articles you could reference?

Fran (luckily without GERD)

Mike

This used to happen to me too. Thick headedness was the bane of my life. Do you get it on waking in the morning too? Since cutting out grains, potatoes, dairy etc it does not happen now. However, I made myself a sandwich or two last night!! I was tired and emotional and couldn't be bothered making a paleo snack. I thought once would be alright since it was such a long time since my last one (the pancakes). Thick head all last night, thick head on waking. Today I have been throwing teaspoons in the bin instead of T bags, dropping things that have been handed to me, extremely dizzy on standing, but no ectopics.

Fran

Fran,

You're the queen of the Internet as far as finding references for topics discussed on this BB. I don't know where you find some of the references you've posted.

Are you looking for something on Mg and absorption or GLP-1? I certainly don't have any other references explaining or hypothesizing other reasons for glucagon dysfunction.

This Mg and insulin thing is very interesting. In obese diabetics (or those with impaired glucose tolerance) Mg deficiency causes insulin resistance or insulin hyposensitivity (OGTT shows insulin spiking upon presentation of simple carbohydrates). In the lean individuals with PRH (flat OGTT and relatively low levels of insulin) Mg deficiency causes insulin hypersensitivity. Body habitus determines manifestation of the Mg deficiency. And I'm sure eating habits determine body habitus. Why is there no clinical glucagon dysfunction (PRH) in the former group. This appears to be yet another indication that Mg deficiency has many faces.

Talk about an interesting trial. I would certainly like to know the postprandial GLP-1 levels in LAFers.

The typical endurance athlete is quite lean. I would certainly be interested in knowing the postprandial GLP-1 levels in that subgroup of VMAFers. Which came first the elevated GLP-1 (chicken) or the lean habitus (egg)? Is the endurance sports thing just a convenient "bystander"? There are reports that increased GLP-1 is secondary to increased vagal tone.

PC, MD v54

PC

Thanks for that. In my case at the moment I am looking for GLP-1 references. I feel I have followed through with Mg and am secure that my Mg is at a good level. I can stretch all I like now, whatever the time of the month and not induce any cramps or spasms. So I think that GLP-1 for me is the culprit.

That trial you talked about would be very interesting. Can one go to the Dr and ask for GPL-1 to be measured. If so I am going for it.

I haven't read the article properly you posted on your second post about PRH, but on skimming it I saw mention of adrenergic symptoms too. And what really got to me was the wide ranging symptoms that include all the ailments in our family. Including rheumatoid symptoms. Thanks so much for this. I have new leads to follow.

Fran

Fran,

Below are a few websites with info on GLP-1. It's nothing you can't get directly from a google search.

<http://www.glucagon.com/reviews.htm>

<http://www.obesity-101.com/101-3.htm>

<http://www.glucagon.com/Glucagon-like%20peptides%202001%20Review.%20pdf.pdf>

http://www.phoenixpeptide.com/allobesity/Catalog%20Files/_Glucagon%20Section/GLP.html

Also, if you're going to go for a GLP-1 level, I think it ought to be postprandial. They have a nice hyperglucidic breakfast in that article that will enable you to compare your level with those from PRH patients, post gastrectomy patients and controls. However, it sure didn't look like what I know you would normally have for breakie.

PC, MD v54

PC

Thanks for the links to GLP-1. Normally I would be on it like a ball, but I don't have much time at the moment and I was being lazy. And you are right about the breakfast test. But I did it a while back when I discovered reactive hypoglycemia and it showed without a shadow of a doubt that I had it. This is what initiated my further diet changes (despite getting rid of AF) to where I am today on the paleo diet. The connection is there. But finding the exact spot, or culprit, is what is

defying me. Thanks

I am taking the liberty of copying two paragraphs from your POSTPRANDIAL REACTIVE HYPOGLYCEMIA link. It may be old news to you PC, but the significance of it has really hit home for me, and may for others.

J.F. BRUN, C. FEDOU, J. MERCIER

<http://www.alfediam.org/media/pdf/RevueBrunD&M5-2000.pdf>

P J Lefevre proposes the term of "Adrenergic hormone postprandial syndrome" to describe autonomic symptoms (anxiety, palpitations, sweating, irritability, tremor..) that are experimentally observed after insulin infusion, at plasma glucose levels of about 3.7mmol/l. It is likely that in some individuals, after a meal, such autonomic counterregulation may occur. This counterregulatory response induces symptoms but also prevents biochemical hypoglycemia being achieved. In such cases, since low blood glucose levels do not occur, the term "postprandial" or "reactive" hypoglycemia should thus be avoided.

This adrenergic hormone postprandial syndrome is likely to be of clinical relevance, since Rokas and coworkers published a case report of a patient with refractory atrioventricular nodal reentry tachycardia in whom it was possible to document that reactive hypoglycemia was the trigger for aggravation of the arrhythmia. Over a period of 6 years, a series of electrophysiological studies revealed that, when the patient was in a hypoglycemic state, initiation of tachycardia was easy and most importantly that tachycardia termination by extra stimulus pacing always failed. Furthermore, Atrial Fibrillation was inducible or spontaneously occurred when the blood glucose level was reduced by IV insulin administration.

Also...., hyperinsulinemia [thought to be caused by exaggerated response of GPL-1] has been reported to enhance epinephrine, norepinephrine and cortisol secretion in response to hypoglycemia, while it does not modify glucagen and GH responses. Thus excess insulin may be a factor involved in in postprandial adrenergic syndrome whose link with PRH is discussed above.

----- spelling mistakes and bad grammar mine!!

I may be totally wrong here, so jump in and put me straight if I am. But I have read this to mean that in some cases AF may be a reaction by the body to prevent clinical hypoglycemia from occurring. I myself only ever realised I had a problem with reactive hypoglycemia when I managed to stop AF. So from my point of view they are on to something here.

However, I also know that the heart drugs and the anticonvulsants I took will also mask these symptoms. So the plot still thickens. Still have to go research GLP-1 to find an answer.

And in response to GLP-1 facilitating gastric distention with poorly digested contents and your theory of a relationship with GERD without the hyperacidity. The very first time I tried WW I got the immediate stomach discomfort in the form of an ice cold stomach radiating round the solar plexus (I remember thinking at the time it was like a reverse heart burn). Can you recommend any good pancreatic enzymes. I have never thought of myself with tummy problems, rather the opposite. But it may be worth me trying these. Do normal Dr's prescribe these?

Fran (still with low blood pressure)

Fran,

Hans book mentions two pancreatic enzyme preparations, Cotazym and ?. I'm at work and don't have access to his book here.

Regarding adrenergic hormone postprandial syndrome, I think you're right on track. As stated in my earlier post, insulin and catecholamines both stimulate transcellular migration of K into cells. This is what I think causes the PACs/LAF. It matters little whether hypoglycemia is actually achieved, it's the hypokalemia, transient or otherwise, that is critical.

The hyperlink for the 6 year case history of AF inducible by insulin is:

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=1374880&dopt=Abstract

PC, MD v54

Hi PC,

Just looked in here today & have just read your posts. I am lean, and do believe some(maybe all?) of my episodes are due to hypoglycemia. I skimmed over the article and wonder why maybe some of the drugs to treat PRH haven't been used in conjunction with cardiac arrhythmias, or maybe if it has been reported somewhere in the literature that treatment of PRH with drugs has inadvertently corrected arrhythmias? Also, would it be correct to assume if you had a healthy appetite that you probably wouldn't have too much GLP-1? Wish I could add something here, but I'm like Mike here.

Jim

Jim,

Your posts always contribute something, even if you think they don't.

Yes, your surmise about healthy appetite and GLP-1 is correct, I believe. They are inversely correlated. The article specifically stated that diabetes was very rare in PRH patients. I would venture to say that this was probably early onset diabetes and definitely not the Syndrome X kind of diabetes caused by obesity. Such individuals must have a low GLP-1, which is being investigated for a possible role in weight reduction programs.

Regarding the therapeutic recommendations for treating PRH and inadvertent correction of an arrhythmia, I would venture to say that we are in an early phase of discovery here (personal opinion and nothing more). Heck, up until the late 80's we were still doing OGTTs to diagnose this condition, which was called reactive hypoglycemia, then idiopathic postprandial syndrome and then PRH. This article has added postprandial reactive syndrome (PRS) to describe the postprandial state of adrenergic symptoms unaccompanied by hypoglycemia. I don't think the hypoglycemia research is looking at LAF. I don't think the LAF research (what little there is) is looking at hypoglycemia. Neither group has made the connection. At this point it's "patient heal thyself", because the healthcare industry is not interested in LAF. They are certainly not interested in the nutritional approach. That's why this BB is so popular.

We'll just let them do the research and we'll make the correlations.

And was that like Mike F. or like Mike J.?

PC, MD v54

Brilliant PC

This may be a a stupid question. But the it occurred to me - which came first, the chicken or the egg. Is all reactive hypoglycemia, or postprandial reactive hypoglycemia caused by diet alone (high in carbs and low in fat). Or is it possible that there could be a problem with GLP-1 which then induces the PRH.

Sorry if this has been covered above but I did not see it

Fran, very lean, high good cholesterol - but just got leaner and not suffering with PRH at present.

PS what is body mass under 25 mean?

Fran

Fran,

As always, thank you so much for your input and opinion.

Regarding your first question about the chicken and the egg:
to quote from the above referenced article, "It is not likely that increased insulin sensitivity alone can induce hypoglycemia, since high values like those found in PRH are found in young, lean people who never report suffering PRH." The GLP-1 can certainly explain the insulin hypersensitivity. However, the recipe for PRH also requires glucagon dysfunction. Mg deficiency conveniently provides for this.

Again, according to the article, "High carbohydrate-low fat diet increases insulin sensitivity, and this pattern is frequently found in PRH patients." This probably develops over many years and might explain at least part of the connection between endurance sports and LAF. I know this was my diet when I was running all those marathons, skipping all those meals and not properly hydrating.

BMI = body mass index = weight in kg/(height in meters)squared
<25 is good and the recommended target
25 - 30 is defined as overweight
30 - 40 is defined as obese
>40 is defined as massively obese

I hope those with LAF and hypoglycemia don't neglect reading the dietary suggestions for improvement listed in the article.

PC, MD v54

Fellow Fibbers,

GLP-1 is not only stimulates insulin secretion, suppresses appetite and inhibits gastric emptying but also suppresses gastric acid secretion. This combination would seem to facilitate gastric distention with poorly digested contents. This would seem to be vagotonic. It may in some way be

related to GERD without the hyperacidity. Furthermore, alkaline fluids (WW, Noah's, unique water) would further compromise this digestive process by soaking up the diminished HCl. It may be this and not CO2 production that is more instrumental in alkaline WW causing gastric discomfort. In view of this perhaps a trial of pancreatic enzymes might be in order. Any thoughts or experiences to relate?

It appears that GLP-1 is not generally commercially available. I've talked to our sendout reference lab and they are researching this for me, since they don't do it (and we are one of their biggest customers).

PC, MD v54

Hi everyone

Whenever i go in the conference room i need to do a bit of reading to understand it all but i think i'm grasping some of it

I can definitely relate to hypo symptoms i wasn't really aware of it until my second pregnancy. From twenty weeks i wasn't feeling right and they picked up lots of sugar in my urine. They sent me for the five blood tests over 2 1/2 hours and diagnosed impaired glucose tolerance .I know this can be common in pregnancy and it went ok afterwards but it made me very aware of the symptoms - a cloudy head when my blood sugar was over 10+ on the reading (about 1 1/2 after eating) and palpitations when it dropped under 4 which was within about 3-4 hours after eating

i was told by the diabetic nurse to start eating well after my pregnancy as it could show itself later in life.

After having my son i joined slimming world and i follow the red day eating plan . It's very like the Atkins diet but a bit lower in fat and it allows controlled portions of complex carbs. Its easy to follow and its now my way of eating all the time but if i do let things slip i get the symptoms back just like Fran has mentioned, not as bad as when i was pregnant but they are there.

Just a note for Fran, what would be the best snacks to help keep blood sugar levels steady between meals? i tend to flag a bit between breakfast and lunch i like fruit but could some make things worse?

toni 29 alafer x

Toni

My mainstay snacks are almonds, pumpkin seeds, sunflower seeds, olives, fruit and dried fruit. I used to need protein with every snack, but find now the nuts and seeds hold me together. Sometimes if I have left over roast I will also have a slice of meat. I also make myself some cookies from time to time. I make them with ground almonds and coconut, grated apples (seeds and sliced almonds too for texture), olive oil, honey and egg. This is because I eat no flour anymore. They do the job of a comfort food.

I do find the need to add extra fat in my diet though as I get really hungry quickly without it. I always pour some olive oil over salads and veggies. I've even started eating the fat from my chops!! Cholesterol does not worry me now as it is the high carb diet that contributes to that. I'm going to ask for my cholesterol to be done again, but keep putting it off as they want me fasting till

they see me at about 11 in the morning from the night before. That is a sure fire way for me to get symptoms back.

Fran

Another 'hypo diet' suggestion that I use is home made peanut butter--grind canned peanuts in a coffee grinder [the small electric type with a blender like blade] [Walmart ~\$12] to a powder and add olive oil. It tastes as good as any 'store bought' peanut butter but without all the chemicals. ""Don't use 'dry roasted' peanuts as they are FULL of chemicals"". I've tried also pecans and almonds and they taste great.

Ben