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

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SUBJECT: Ativan (Lorazepam) & LAF

Time for a new conference subject!

Several afibbers have reported that taking an Ativan (lorazepam) before going out for a drink with the boys, before a big meal or before going to bed seems to calm ectopic beats and may possibly prevent a full blown afib episode. I believe the dose used by most is 0.5 mg in the form of a sublingual tablet. At least that is what I have been using. I have found this dose of Ativan quite useful in calming erratic heartbeats occurring just as I lie down to go to sleep, but have also used it to defuse stressful situations during the day.

Ativan and other minor tranquilizers are relatively harmless (certainly when compared to powerful antiarrhythmics), but could result in a psychological dependency so are not altogether benevolent. They can also cause drowsiness and perhaps interfere with motor skills, but I have found this latter effect quite minimal with the 0.5 mg dose.

There is not a whole lot in the medical literature regarding lorazepam and atrial fibrillation (like zilch actually); however, I do have a few statements in my book that are relevant:

- Pages 22-23. Minor tranquilizers and beta-blockers are sometimes quite effective in reducing premature atrial complexes (PACs) (1)
- Page 99. The tranquilizer lorazepam (Ativan) has been found to decrease parasympathetic (vagal) activity significantly while increasing heart rate by about 8% (6 beats per minute)(2). This change in autonomic system balance may be enough to prevent vagal episodes that begin after going to bed….

I have the adrenergic/mixed kind of LAF myself so it would appear that the possible benefits of Ativan may not be limited to vagal afibbers and may perhaps, be primarily due to its ability to increase heart rate.

Anyway, I thought it might be worthwhile to give Ativan a bit of a trial. For those willing to participate I would suggest the following:

- Get a prescription from your doctor for the 0.5mg sublingual lorazepam tablets. Make sure to check with him that the lorazepam will not interfere with any other medications you may be taking.
- Try one just before going to bed, just before dinner or just before a stressful event
- Keep track of the effects following. It generally starts working 20 minutes to 1 hour after ingestion.
- Keep track of any side effects and note how long the beneficial effect lasted.
- Make special note of whether it stopped irregular heart beats and whether its use may actually have extended the time between afib episodes.
- Keep track of any changes in pulse rate or blood pressure if you have the equipment to do so.

Hans

(1) Harrison's Principles of Internal Medicine, 12th Edition, 1991, McGraw-Hill, NY, pp. 909-14
(2) Vogel, L.R. et al. Lorazepam reduces cardiac vagal modulation in normal subjects. Journal of Clinical Psychopharmacology, Vol 16, December 1996, pp. 449-53.

Hans, I'm very interested trying ativan for stress relief and lessening the episodes of AF. I have been in persistent AF since 2/03 after having LAF for 10 years but the episodes were every 4-6 weeks lasting about 12 hours. My EP has run me thru the gamut of drugs. I am currently taking 3000 mg of procainamide daily. It's the only drug that has brought relief. I still am in AF about 60 to 75% of the time but the episodes are very mild so I can function normally. I'am wanting to get off antiarrhythmics, but the episodes (without drugs) are so intense it's not an opposition right now. I've lost 35 lbs since February and eat a healthy low carb diet. But in my case I feel stress is a major trigger of my AF. I lost my job in January and then my son was sent to Iraq and has been there 5 months now. Any suggestions would be appreciated.

Tom T.

Tom,

I am sorry to hear about your almost continuous AF and I am sure the tremendous stress you are under has a lot to do with it. Ativan would probably be quite effective in relieving anxiety, but I don't think it is a good idea to take it on a continuous basis. Have you thought of taking up yoga, meditation, Qi Gong or some other relaxation technique; that might actually help you quite a bit?

Hans

Hans,
I would like to participate in the Ativan trial.

I already have a filled prescription for Ativan 0.5 mg. tablets - small, round, white with an "m" imprinted on the tablet - which I have used once or twice as a sleeping pill during well established afib episodes. The directions on the bottle do not indicate how to take it, so I swallowed a tablet the usual way with water.

Are there two different forms? Why do you specify sublingual form? If it is important for this study to take it sublingual, can I use my current prescription sub lingual?

Carol

Carol,

The reason why I suggested that we standardize on the sublingual 0.5 mg Ativan (a green pill) is that it is absorbed into the system so much quicker than the normal oral version (the white pill). Getting the quickest possible absorption could be important if the pill is used in an attempt to ward off an afib episode. The sublingual tablet dissolves in about 20 seconds and should be left in the mouth for about two minutes before any other liquid or food is taken by mouth.

Hans

I now understand.

I guess my interest was academic after all, because I continue to be afib free! I have had a couple of recent spells of tachycardia about 4 am. after good sleep without any afib. But my heart/ vagus nerve now seems to be settling down once again into a consistent NSR pattern. Incidentally, I seem to drift off to sleep faster now that I am taking 600 mg. of magnesium and potassium.

Carol

In doing some reading on the prescribing insert. if Ativan, I found more info on injections, than tabs. There is only a slight difference between the two (2-chlorophenyl, rather than 0-chlorophenyl).

Here is what the clinical pharmacology stated for injections:

Lorazepam (lor) interacts with GABA-benzodiazapine (ben) receptor complex, which is widespread in the brain. The interaction is presumed to be responsible for lor.'s mechanism of action. Lor. exhibits relatively high specific affinity for its recognition site, but does not displace GABA. Attachment to the specific binding site enhances the affinity of GABA for its receptor site on the same receptor complex. The pharmacodynamic consequences of ben. agonist actions include antianxiety effects, sedation, and reduction of seizure activity. The intensity of action is directly related to the degree of ben. receptor occupancy.

http://www.wyeth.com/products/wpp_products/ativan_pi.asp

I found this to be of interest in the book, "The Healing Nutrients Within".

"Two of the best examples of the blending of medical technology and nutritional science are gabapentin (Neurontin) and tiagabine (Gabitril), which are almost identical to the chemical structure of GABA. They represent one of the largest breakthroughs in medicine to assist individuals to remain calm without having to resort to using a narcotic."

As we all know, magnesium has worked very well for some people, and the reason for this, is its helpful nature to assist the amino acids with their pathways to do their jobs. Mg. coupled with vit. B6, as PC so eloquently put it, helps glutamate break down to GABA, but what else we should know, is that lysine, through its metabolite pipecolic acid, seems to amplify GABA action in the brain. Maybe the Mg. coupled with lysine, is what's working for Carol. I, for one, was amazed with the scar that almost disappeared when taking lysine. (The scar was of chicken pox herpes origin). I have not been taking any supps. of late, until my next appt. with Dr. Gersten.

Just found it to be of interest, that Ativan was having positive effects for some of you, and wondered why. I find some consolation in this fact, as it makes me feel more strongly, that this is a possible fix, once we figure out what is lacking. What is still so baffling, however, is that there seems to be so many differences with all of us, yet we have the same affliction, which keeps bringing me back to the orchestrator of the neurotransmitters, SEROTONIN, and the ruler of the circadian rhythm, MELOTONIN. They seem to be the only common link that makes any sense.

I can't wait until I can start incorporating the uses of aminos, esp. tryptophan.

Richard

Richard,

Very interesting observation regarding lorazepam, GABA and lysine. I wonder if the fibrotic tissue in the heart of afibbers is similar in nature to your scar tissue that disappeared upon taking a lysine supplement. If so, lysine could indeed be a very important supplement for afibbers.

Hans

Hans and All,

Here's a bit about lysine, and one must know that it is the precursor of carnitine.

Lysine is degraded into various important metabolites during its metabolism, and not surprisingly, it is metabolized through many different pathways.

Lysine is degraded principally to acetyl CoA, a form of coenzyme A, a vital catalyst in the body and a critical nutrient in carbohydrate metabolism.

During its metabolism, lysine also assists in transanimation by forming the linkage, or peptide bond, between transaminase enzymes such as SGPT and SGOT, and pyridoxal phosphate, the coenzyme needed for this activity to occur. Lysine is able to do this because it is constructed of two amino groups: one connects to the transaminase enzyme while the other joins with pyridoxal phosphate. Enzymes performing these reactions have been found present in tissues of the liver, kidney, heart, adrenal gland, thymus gland, brain and skin (in order of decreasing activity).

When some lysine is broken down into citrulline, which is needed in the body for normal protein metabolism, minor amounts of lysine also enter into the homocitrulline, homoarginine, and pipecolic acid pathways of metabolism (pipecolic acid, a neurotransmitter, is found to be highly concentrated in the brain when lysine is given intravenously).

Lysine and arginine share a common transport system that, because of their various chemical properties, can make them antagonistic. An excess of arginine can lead to a depletion of lysine, and the reverse happens, as well.

Lysine's successful conversion to these metabolites along these different metabolic pathways primarily requires B6, B2, and niacin (B3). Vit. C and iron help increase absorbtion and utilization of lysine by the body. Metabolism of lysine is particularly sensitive to the presence of viral infections, stress, and aging.

Lysine is an essential amino acid and this became evident when researchers at Kansas State Univ. found that a diet deficient in lysine results in depressed growth when fed to experimental animals. This lysine deficient diet when fed to three consecutive generations of animals resulted in persistent differences in the animal's growth. The offspring of these three generations continued to show the effects of lysine deficiency for several more generations and the increased requirements for the amino acid.

Lotan and colleagues, working at the Univ. of Houston, found that lysine deficiency suppressed the immune system in the same proportion that overall body growth was suppressed. Resupplementing with lysine resulted in increased growth of thymus and improved immune system parameters.

Deficiency symptoms:

Signs and symptoms of lysine deficiency include fatigue, decreased concentration, irritability, hair loss, poor appetite, weight loss, anemia, enzyme disorders, and abnormalities in gastric functioning, including absorption of calcium.

So there's a small lesson on lysine for today. Would it be less expensive to supplement with lysine and its cofactors, compared to the cost of L-carnitine, which is derived from lysine, along with attaining extra benefits of lysine? Or should one take both, at least for therapeutic reasons in the beginning? I like the fact that lysine enhances GABA.

Richard

Richard,
Maybe you will some day find an answer to why I have a terrible problem with jet lag, which is tied in with the circadian rhythm and melatonin.

It takes me a good 10 practically sleepless days before my sleep cycle begins to readjust when traveling to England and France. I have given up traveling over there - and I have a one year old grandchild in France, whom I have never

seen. I have been deterred by a combination of severe afib and jet lag. My afib went wild from no sleep the last time I went to France and my jet lag just kept lagging.

I get frightening nightmares from even the smallest amounts of melatonin.

Carol

Carol--

Regarding jet lag: there is a method called the Argonne National Laboratory Jet Lag Diet which is very effective in reducing or eliminating jet lag when traveling.

I have tried the method when traveling to Europe from the west coast of the U.S.

The method modifies the circadian rhythm by using simple dietary techniques. It is a bit complex but reading the instructions a couple of times should make it clear how it works. One starts modifying one's diet two or three days before one's trip.

You can find the diet by searching for it in Google.

Michael

Hans,

I have used different anti-anxiety meds over the years to ward off impending afib attacks. In the beginning it did seem to help somewhat, but in the end the length of time between afib attacks seemed to be the same whether I took a pill or not. I realize we are all different, but I would suspect most, if not all people who are vagal afibbers, that try this will find no relief in the end. This may not be the case for adrenergic afibbers though because stress (I believe) may play more of a roll in afib.

Jim W.
