INTERNATIONAL HEALTH NEWS

Your Gateway to Better Health!

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Editorial

It is estimated that about 50 million Americans now take an aspirin every day or every second day in order to avoid a heart attack. Researchers at Oxford University have just released a study questioning the value of this practice. They reckon that 1100 people need to take aspirin for one year in order to prevent 2 heart attacks. On the downside, 27 of the 100 people would end up in hospital with gastrointestinal bleeding and 3 of them would die from the bleeding. Among non-aspirin takers 16 out of 1100 would develop gastrointestinal bleeding in the course of a year and 2 of them would die. Not much of a bargain is it? Particularly not when there are so many totally safe alternative approaches to reducing your risk of a heart attack. Regular exercise, eating fish, drinking tea, eating nuts or supplementing with folic acid, vitamin E, beta-carotene or lycopene have all been proven to decrease the risk of a heart attack by at least 50% and to my knowledge no one has ever died from taking these preventive measures.

In other news, Dutch and Indonesian researchers have discovered a worldwide deficiency of vitamin A. So it's back to the cod liver oil that our mothers used to think was so indispensable – it seems like it really is! The safety of cell phones is still being debated. The best approach is to use them as little as possible; this is especially important for teenagers and preteens. Blood clots and circulatory problems are becoming more common on long distance flights. The best approach is to stay away from alcohol and drink lots of water.

Yours in health,
Hans Larsen

February Highlights

LETTERS TO THE EDITOR

I have just read your article on Parkinson's disease and found it most interesting. I wonder if you would be so kind as to comment on glutathione therapy for Parkinson's and especially on a Dr. David Perlmutter of Naples, Florida who offers this treatment.

LP, USA

Editor: *You can find the latest information on alternative treatments for Parkinson's including glutathione supplementation at www.thorne.com/altmedrev/.fulltext/5/6/502.html*

I don't know anything about Dr. Perlmutter, but you can find his website at www.brainrecovery.com. It sounds reasonable that glutathione would help prevent and perhaps even slow the progression of Parkinson's disease.

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Are there any side effects from taking folic acid?

TA, Canada

Editor: *Folic acid is a very safe vitamin. It is water-soluble so any excess is excreted in the urine. As much as 40 mg/day has been used in the treatment of depression and 15 mg/day has been recommended for other conditions. Very high doses may, in extremely rare cases, cause itching of the skin or hives (urticaria). Folic acid therapy may decrease serum levels of phenytoin in patients being treated with the drug Dilantin. The recommended daily intake of folic acid is 400-800 micrograms (0.4-0.8 mg); these low dosages are considered entirely safe and free of side effects. Folic acid should be accompanied by vitamins B12 and B6 for best effect.*

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I read that vitamin C may cause artery thickening at 500 mg/day. Is this true? And is artery thickening desirable (thick, healthy, strong artery walls) or undesirable (hardening, clogging, high blood pressure, stroke, heart attack, etc.)?

PS, USA

Editor: *I would like to refer you to the Linus Pauling Institute at the University of Oregon for answers to your questions. You can find their comments at osu.orst.edu/dept/ncs/newsarch/2000/Mar00/artery.htm.*

I have personally taken 500 mg of vitamin C 3 times daily for at least the last 25 years and believe this amount to be safe and beneficial.

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I am wondering what the bad things are about fish oils.

JS, USA

Editor: *I have done a lot of research on fish oils and have never come across any mention of adverse effects. I do believe that they and, of course, fatty fish on their own are truly good for us. This statement comes with a big caveat though. Fish oils are not beneficial if they are rancid or full of mercury, PCBs or other contaminants. So you need to make sure that you get a reputable brand.*

ABSTRACTS

New world requirements for vitamin A

NIJMEGEN, THE NETHERLANDS. Vitamin A (retinol) is a fat-soluble vitamin required to ensure proper function of the immune system and to counteract the development of night blindness and weak eyesight; it may also play an important role in cancer prevention. The current Recommended Daily Allowance (RDA) in North America is 5000 IU or, more correctly, 1000 RE equivalents for men and 4000 IU or 800 RE for women. Children need between 400 and 700 RE daily depending on age. Any intake above the daily requirement is stored in the liver. This fact has led to cautions about taking too much vitamin A as it can be toxic in large quantities. Vitamin A toxicity may occur in adults who take more than 10,000 RE daily for several years. Pregnant women should keep their daily intake below 1000 RE/day.

Vitamin A is obtained from the diet in two forms either as retinol (from animal products) or in the form of carotenes (from fruits and vegetables) which the body converts to retinol. In 1967 the World Health Organization reported that it took six micrograms of beta-carotene to produce one microgram (1 RE) of pure vitamin A. This conversion factor has been used ever since to determine the average daily vitamin A intake throughout the world. Using the WHO factor it was estimated that the average daily vitamin A intake varied from about 600 RE in South America and Asia to about 1000 RE in Europe and North America. In other words, it's adequate in Europe and North America, but deficient in Asia and South America.

Dutch and Indonesian researchers now report that the 1967 WHO conversion factor is seriously wrong. Using up-to-date analysis techniques and new information about bioavailability and bioconversion of vitamin A and carotenes they conclude that it takes not six micrograms of beta-carotene from fruits and vegetables to yield one microgram of vitamin A, but rather 21

micrograms. This finding puts a completely different complexion on things. Essentially, the entire world is likely to be vitamin A deficient. The revised estimated daily intake of vitamin A is now only 780 RE in Europe, 581 RE in North America, 372 RE in South America, and a mere 258 RE in Asia where blindness among children is becoming endemic.

The researchers urge further work to determine how to tackle this massive problem. Editor's Note: This project obviously could take years. In the meantime it would seem wise to supplement with cod or halibut liver oil which are both excellent sources of vitamins A and D.

West, Clive E. Meeting requirements for vitamin A. **Nutrition Reviews***, Vol. 58, November 2000, pp. 341-45*

Folic acid and heart disease

HYATTSVILLE, MARYLAND. High homocysteine levels are associated with an increased risk of heart disease. High homocysteine levels have also been linked to a relative folic acid deficiency. Researchers at the Centers for Disease Control and Prevention now report that low blood levels of folic acid are associated with a substantially increased risk of dying from cardiovascular disease. Their study involved 689 adults aged between 30 and 75 years who were free of heart disease at the start of the study in 1976-1980. After 12 to 16 years of follow-up 122 of the participants without diabetes had died - 49 of them from heart disease. Among the participants with diabetes, 52 in all, 25 died - 12 of them from heart disease.

In the non-diabetic group there was a clear association between blood levels of folate and death from heart disease. The participants with folate levels below 10 nmol/L had a 2.64 times higher age and sex adjusted risk of dying from cardiovascular disease than did the participants with levels above 16.8 nmol/L. Even when adjusting for other risk factors (education level, race, cigarette smoking, alcohol consumption, cholesterol levels, blood pressure, and body mass index) the death rate among the participants with low folate status was still 2.28 times higher than among the people with higher levels. The observations made in the non-diabetic group tended to parallel those in the diabetes group, but because of the small sample size in the diabetes group the observed trends were not statistically significant.

The researchers conclude that at least a third of the participants had folate levels at baseline (1976-1980) so low that they would be in the high-risk category for dying from cardiovascular disease. They urge further work to determine if recent efforts to fortify the US food supply with folic acid are sufficient to decrease the proportion of the population at risk for heart disease because of insufficient folate levels.

Loria, Catherine M., et al. Serum folate and cardiovascular disease mortality among US men and women. **Archives of Internal Medicine**, *Vol. 160, November 27, 2000, pp. 3258-62*

Safety of cell phones revisited

COVENTRY, UNITED KINGDOM. Public concern over the safety of cell phones (mobile telephones) continues to grow. Dr. G.J. Hyland of the University of Warwick and the International Institute of Biophysics in Germany has just released an excellent review of the current knowledge on the subject. Current safety guidelines essentially look at cell phones as small microwave ovens and as long as they don't heat up your scull and adjacent brain tissue by more than one degree Celsius they are deemed to be safe. Dr. Hyland points out that the phone emits low intensity, pulsed radiation that can have a variety of non-thermal effects. For example, cell phones emit frequencies in the 2 Hz and 8.34 Hz bands that correspond exactly to the frequencies of electrical oscillations found in the human brain (delta and alpha brain waves).

Animal experiments have discovered many serious effects of cell phone radiation including errors in cell division, induction of epilepsy, depression of melatonin levels, increase in DNA breaks, and promotion of lymphomas. Reports of headaches and sleep disturbances in people using cell phones have also been published and some fairly recent research concluded that exposure to cell phone frequencies increases blood pressure. Of equal concern is the fact that

cell phones produce strong electromagnetic fields (EMFs). As a matter of fact, the EMF from a cell phone placed right next to your ear is 160 times stronger than the maximum allowable EMF from a computer video monitor. The evidence of risk from base stations (transmission towers) is somewhat sparser although there is strong anecdotal evidence that certain animals thrive much better away from the towers than near them.

Dr. Hyland points out that Soviet microwave radiation of western embassies during the "Cold War" was quite successful in making the personnel sick. He is convinced that the non-thermal effects of cell phone radiation may have serious health repercussions and urges further research on the matter. [47 references]

G.J. Hyland. Physics and biology of mobile telephony. **The Lancet**, *Vol. 356, November 25, 2000, pp. 1833-36*

Dendy, Philip P. Mobile phones and the illusory pursuit of safety. **The Lancet**, *Vol. 356, November 25, 2000, pp. 1782-83 (commentary)*

Aspirin – treat with care!

OXFORD, UNITED KINGDOM. It is estimated that over 50 million Americans now take a daily aspirin in an attempt to ward off a stroke or heart attack. There is evidence that aspirin is somewhat effective in preventing a second stroke. It is estimated that one second stroke can be avoided for every 100 stroke patients treated with aspirin for a year. The evidence of benefit is somewhat murkier when it comes to prevention of a first heart attack. Here 555 people must be treated with aspirin for a year in order to claim the prevention of one heart attack.

Unfortunately, aspirin is not innocuous. It can cause serious bleeding in the gastrointestinal tract and can aggravate existing ulcers. The estimated death rate from gastrointestinal bleeding is 12 per cent. Researchers at Oxford University have just released the results of a very large study aimed at establishing the magnitude of aspirin-related bleeding incidents. They carefully studied the results of 24 major randomized clinical trials involving almost 66,000 participants. They conclude that when treated for a year 2.47 per cent of aspirin takers develop gastrointestinal bleeding as compared to 1.42 per cent among placebo takers. Put in terms of the 50 million Americans now taking aspirin this means that the excess incidence of gastrointestinal bleeding attributable to aspirin would be 525,000 and the excess mortality would be 63,000 every year.

The researchers also investigated whether lower dosages of aspirin would be safer. They found that they were not. The incidence of gastrointestinal bleeding among low-dose aspirin takers was 2.30 per cent compared with 1.45 per cent for placebo takers. Somewhat surprisingly, the study also found that enterically-coated or otherwise modified formulations were no safer than standard aspirin. The increase in gastrointestinal bleeding among users of modified formulations was 93 per cent as compared to 68 per cent for all aspirin users and 59 per cent for low-dose users.

The researchers conclude that patients and their physicians need to consider the trade-off between the benefits and harms of long term aspirin use.

Dr. Martin Tramer of the Geneva University Hospitals in Switzerland wholeheartedly agrees with this conclusion and adds, "It may be more appropriate for some people to eat an apple rather than an aspirin a day."

Derry, Sheena and Loke, Yoon Kong. Risk of gastrointestinal haemorrhage with long term use of aspirin: meta-analysis. **British Medical Journal**, *Vol. 321, November 11, 2000, pp. 1183-87*

Tramer, Martin R. Aspirin, like all other drugs, is a poison. **British Medical Journal**, *Vol. 321, November 11, 2000, pp. 1170-71 (editorial)*

Breast cancer mortality and mammography

TORONTO, CANADA. Several clinical trials have observed a reduction in mortality from breast cancer in women over 50 years of age who received regular mammograms. It is not known, however, whether this benefit is greater than that obtained by an annual physical examination alone. Researchers at the University of Toronto now report the results of a study designed to answer this question.

The clinical trial involved 39,405 women aged between 50 and 59 years at time of entry into the study between 1980 and 1985. The women were

randomized to receive either an annual mammogram (two-view) and physical examination of the breasts or just physical examination alone. All participants were taught and encouraged to practice self-examination as well.

By December 31, 1993 622 invasive and 71 *in situ* breast carcinomas had been discovered in the mammography plus physical examination group and 610 invasive and 16 *in situ* cases had been observed in the physical examination group only. Although the cancers tended to be discovered earlier in the mammography group there was, after 13 years of follow-up, no difference in breast cancer mortality between the two groups (107 deaths in the mammography group and 105 in the physical examination group only)

The biopsy rates were considerably higher in the mammography group. In this group 24.3 per cent of the participants underwent biopsy after the first screen as compared to 8.7 per cent in the physical examination group. The researchers also noted a significant increase in deaths from pancreatic cancer in the mammography group (42 deaths) as compared to the physical examination group (18 deaths). Although this difference is statistically significant it could, according to the researchers, be due to chance.

The researchers conclude that mammography screening does not result in a decrease in the absolute rate of advanced breast cancer and does not reduce mortality when compared to physical examination only. They suggest that physicians and their patients (women aged 50-59 years) consider the option of an annual physical examination carried out by a health professional trained to recognize the signs of early breast cancer plus regular self-examination as an alternative to annual mammograms. [43 references]

Miller, Anthony B., et al. Canadian National Breast Cancer Screening Study-2: 13-year results of a randomized trial in women aged 50-59 years. **Journal of the National Cancer Institute**, *Vol. 92, September 20, 2000, pp. 1490-99*

All yogurts are not the same

PARIS, FRANCE. Researchers at the Hotel-Dieu Hospital have just released the results of an interesting study that clearly proves that yogurt containing live bacteria is superior to pasteurized yogurt. The clinical trial involved 24 healthy men (aged 20 to 60 years), 12 of whom were lactose intolerant. The men were randomized to consume 500 grams/day of either fresh or pasteurized yogurt for two periods of 15 days each, separated by a 15-day washout interval. The researchers found no detectable changes in fasting plasma glucose, insulin, fatty acid, triglyceride or cholesterol concentration. They did find that consumption of fresh yogurt (non-pasteurized) ameliorated lactose malabsorption in the lactose-intolerant men and that this beneficial effect became more pronounced the longer the men consumed the yogurt. On the other hand, ingestion of pasteurized yogurt and the absence of live bacterial cultures tended to further worsen the lactose maldigestion. They also noted a slight but significant increase in calcium intake after 15 days of fresh yogurt consumption in both the normal and lactose-intolerant men. Blood plasma levels of propionate increased significantly after fresh yogurt consumption. Higher propionate levels have been associated with improved glucose tolerance and a reduction in cholesterol levels in some studies.

The researchers conclude that long term consumption of fresh, unpasteurized yogurt can ameliorate lactose intolerance and may improve glucose and lipid metabolism in both normal and lactose-intolerant individuals.

Rizkalla, Salwa W., et al. Chronic consumption of fresh but not heated yogurt improves breath-hydrogen status and short-chain fatty acid profiles: a controlled study in healthy men with or without lactose maldigestion. **American Journal of Clinical Nutrition,** *Vol. 72, December 2000, pp. 1474-79*

Soy protein lowers heart disease risk

DALLAS, TEXAS. Epidemiological studies have shown that populations which consume relatively large quantities of soy protein experience significantly lower mortalities from heart disease than do populations consuming little or no soy products. The American Heart Association has now come out in favour of increasing soy consumption as a means of decreasing the risk of

cardiovascular disease. A recent statement made by the AHA Nutrition Committee points out that numerous clinical trials have found that substituting soy protein for animal protein significantly lowers total cholesterol, low-density (LDL) cholesterol and triglycerides without affecting the level of beneficial high-density (HDL) cholesterol. Soy protein contains all of the essential amino acids in sufficient quantities to support human life – in other words, it is a complete protein. Soy protein also contains trypsin inhibitors, phytic acid, fiber, isoflavones, and several other components known to reduce cholesterol levels. It is found in many fermented and non-fermented soy foods including tofu, tempeh, miso, soybeans, soy nuts, soymilk, soy yogurt, and soy cheese.

The AHA Committee points out that soy products are safe and do not lower cholesterol levels in people with low or normal levels. They recommend the inclusion of 25 grams or more of soy protein, with its associated phytochemicals intact, in the daily diet as a means of lowering cholesterol levels and promoting heart health. This recommendation follows the FDA's (Food and Drug Administration) recent ruling allowing soy protein products to carry the health claim "25 grams/day of soy protein, as part of a diet low in saturated fat and cholesterol, may reduce the risk of heart disease."

Erdman, John W., Jr. Soy protein and cardiovascular disease: a statement for healthcare professionals from the Nutrition Committee of the AHA. **Circulation**, *Vol. 102, November 14, 2000, pp. 2555-59*

Codeine implicated in acute pancreatitis

NICE, FRANCE. Acute pancreatitis is a serious and painful medical condition. There have been several reports of associations between certain drugs and pancreatitis. Now French researchers report that codeine, a popular painkiller derived from opium, can cause acute pancreatitis particularly in patients who have had their gall bladder removed (cholecystectomy).

The medical doctors at the Nice Hospital noted four cases where patients admitted with acute pancreatitis had been taking codeine-containing products one to three hours before having their attack. The first patient, a 65-year-old man, had taken 60 mg of codeine in combination with one gram of acetaminophen 90 minutes before his attack. A 53-year-old woman experienced her attack 90 minutes after taking 40 mg of codeine for a migraine, and a 57-year-old woman had an attack two hours after taking 40 mg of codeine. A 26-year-old woman had an attack two days after taking 40 mg of codeine for an upper respiratory tract infection. All the patients had had their gall bladder removed at some time in the past. The researchers conclude that codeine and codeine-containing products can induce acute pancreatitis especially in people who have had their gall bladder removed.

Hastier, Patrick, et al. A new source of drug-induced acute pancreatitis: codeine. **American Journal of Gastroenterology**, *Vol. 95, November 2000, pp. 3295-98*

NEWSBRIEFS

Tylenol consumption linked to asthma. Researchers at Kings College in London, England have discovered a correlation between the consumption of Tylenol (acetaminophen, paracetamol) and the incidence of allergic diseases. They compared acetaminophen sales in 36 countries with the incidence of asthma, rhinitis, wheeze, and other allergic diseases and concluded that countries with higher sales of acetaminophen (Australia, Canada, New Zealand, Ireland, the USA, and the UK) had a greater prevalence of these diseases. The researchers believe that acetaminophen reduces glutathione levels and thereby impairs antioxidant defenses and promotes allergic inflammation. They caution that further studies are needed to confirm the connection.

British Medical Journal, November 11, 2000, p. 1178

Lycopene: a powerful cancer fighter? A study at the Harvard Medical School found that regular consumption of tomato products could reduce the risk of prostate cancer by up to a third. Other researchers have found that diets containing high levels of lycopene (a carotenoid found in

tomatoes) can reduce the risk of heart attacks and breast, pancreatic and colon cancers. Now researchers at the Hebrew University of Jerusalem report that lycopene on its own will kill human oral cancer cells. They plan to test the effectiveness of lycopene in people next. The best sources of lycopene are concentrated tomato products that also contain some fat such as ketchup and pizza toppings.
New Scientist, December 23/30, p. 4

Solvent-based paints damage nervous system. Researchers at the University of Aberdeen have discovered that painters who have been heavily exposed to industrial solvents and paints have significantly higher levels of personality disorders than do unexposed people. They suspect that commonly used solvents such as xylene and toluene damage the nervous system.
New Scientist, January 27, 2001, p. 21

Stay off the booze on long flights. Blood clots and circulatory problems are becoming increasingly common among passengers on long distance flights. The medical clinic at Tokyo International Airport treats between 100 and 150 passengers for these conditions every year – most of them economy class passengers. Sitting still for long periods of time increases the risk of forming blood clots in the legs. If these clots move into the heart or lungs the results can be fatal. Japanese researchers have just completed a clinical trial to determine if drinking plenty of water during the flight would help alleviate the problem. They found that water drinkers maintained their blood pressure (it dropped in controls), had more oxygen in their brain, and greater blood flow in their arteries. Says Toshiro Makino, the director of the airport clinic "The biggest problem is dehydration, and that is made worse by drinking alcohol. My advice is to keep off the alcohol and drink lots of water."
New Scientist, January 13, 2001, p. 7

A little dirt may be good for you. The "hygiene hypothesis" postulates that the emphasis on cleanliness prevalent in modern life leads to more allergies and autoimmune diseases. This is especially true in children who are protected from contact with dirt and its pathogens. Dr. Barbara Fazekas of the Institute of Cancer Medicine and Cell Biology in Sydney, Australia now suggests that this may be due to the fact that the dendritic cells which turn on the T cells (the body's key immune system defenders) become too sensitive when they are not exposed to outside pathogens. So "to keep themselves busy" they may inadvertently stimulate T cells to attack the body's own proteins and trigger autoimmune disease.
New Scientist, January 13, 2001, p. 12

RESEARCH REPORT

GH Enhancers: Are They Worth the Risk?

Growth hormone (GH) enhancers are getting a great deal of attention these days especially among bodybuilders. An 8-week course of GH enhancer is reputed to produce 15 or more pounds of solid muscle when combined with regular weight training.

Human growth hormone is necessary for growth and a deficiency produces short people. GH does not actually stimulate growth directly, but causes the release of insulin-like growth factors, particularly insulin-like growth factor 1 or IGF-1. It is IGF-1 that is responsible for growth and it stimulates the synthesis of lean muscle mass in particular. Human IGF-1 levels vary with age; they are particularly high during puberty and by the age of 60 years they are only about half the average value (200 micrograms/liter) of a younger adult.

Experiments to increase IGF-1 levels in older men through injections of recombinant (synthetic) GH produced astounding results. An 8.8% increase in lean body mass, a 14.4% decrease in fatty tissue, a 1.6% increase in vertebral bone density, and a 7.1% increase in skin thickness were reported by American medical researchers in 1990. Their trial lasted a year and although the 21 participants all remained healthy except for one who developed prostate cancer, the researchers warned that side effects such as edema, hypertension, diabetes, and enlargement of the heart could occur with prolonged use of synthetic GH. Other

researchers found that GH injections in young people produced larger muscles and kidneys. More recently GH injections have become popular among athletes as a super-efficient way to increase muscle mass and strength.

Growth hormone is naturally secreted by cells in the pituitary gland and acts on the liver to produce IGF-1. IGF-1 levels are normally quite steady, but increase during periods of excessive stress, through exercise, and by consuming a diet rich in certain amino acids especially arginine, ornithine, glycine, and lysine. These amino acids act directly on the pituitary gland to stimulate the production of GH and its downstream fellow hormone, IGF-1. So why not just eat a lot of these amino acids if you want to grow bigger muscles? Unfortunately, or perhaps fortunately as we shall see later, stomach acid is very tough on amino acids and only 10% or less of them actually survive long enough to get into the blood stream. This is where GH enhancers play a role. These products use a patented process to protect the amino acids in the stomach and as a result 90% or more of them are absorbed into the blood stream. It is claimed that the resulting flooding of the pituitary gland with the raw materials it needs to produce growth hormone can result in IGF-1 level increases of 200% or more. Surprise, surprise! The body has a built-in mechanism to prevent IGF-1 levels from going too high. Somatostatin is released by the hypothalamus and its major role is to keep IGF-1 levels under control. Another challenge for supplement purveyors? Not really since GH enhancers also contain special peptides, which suppress the natural release of somatostatin.

Now, why would the body go out of its way to prevent high IGF-1 levels when they result in rippling muscles, sculpted bodies, and virtual 10 to 20 year age reversal in older men? Why indeed? The answer is simple, excessive IGF-1 levels can make you very, very sick and yes, they can actually kill you! High IGF-1 levels in children who are not yet fully grown cause gigantism and excessive levels in adults are associated with acromegaly. Acromegaly is not a fun thing with such manifestations as fatigue, coarse facial features, headaches, decreased vision, congestive heart failure, kidney stones, joint pains, and of particular interest to young men, impotence and a lack of sexual desire. It is said that acromegalics look more like each other than like their own family members. As a matter of fact, some pictures of bodybuilders on GH enhancers look suspiciously like the classic depictions of acromegalics. Acromegalics also have higher incidence of cancer especially colon cancer and pituitary tumors.

OK you may say, I can live with this as long as I have the largest muscles on the block. But can you also live with prostate, lung, and colon cancer? Researchers at the National Institutes of Health reported a connection between cancer risk and high IGF-1 levels in 1995. In 1998 researchers at the Harvard School of Public Health reported that a high IGF-1 level is the single most important risk factor for prostate cancer and that high IGF-1 levels were present many years before the cancer was actually diagnosed. Other researchers have found that high IGF-1 levels combined with high testosterone levels are a potent risk. High IGF-1 levels have also been implicated as strong risk factors in breast and colon cancers and now lung cancer is about to be officially added to this list. Recent research has shown that artificially increasing IGF-1 levels in mice accelerates the growth of cancerous tumors.

Dr. Samuel Epstein, MD, a professor at the University of Illinois School of Public Health says, "Taking supplements to increase your IGF-1 levels is reckless, extreme, and bordering on the criminal". Dr. Derek LeRoith of the National Institutes of Health agrees and says that there is now enough evidence that taking GH supplements when you are not deficient will increase the risk of cancer and acromegaly. Says Dr. LeRoith "If you ask me if I would take them, the answer is a definite no". Dr. Michael Pollak, a member of the Harvard team who reported the prostate cancer connection also condemns the use of GH enhancers by normal, healthy individuals. Dr. Pollak points out that growth hormone supplementation has a definite place in medicine in cases where people are deficient and need to increase their IGF-1 levels from sub-normal to normal. However, people who have normal levels would run a significantly increased risk of acromegaly and prostate cancer if they were to take GH enhancers on a sustained basis. Dr. Pollak is also concerned about giving IGF-1 to older people with normal levels for their age. He says the benefits are uncertain and the risks unknown.

A distinguished group of researchers at the University of Bristol in the UK recently voiced their concern about the increasing use of IGF-1 and growth hormone enhancers by bodybuilders and elderly people trying

to recapture their youth. Says Dr. George Davey Smith “People using growth hormone and IGF-1 enhancers are unlikely to be aware of their potentially harmful effects”.

LITERATURE REFERENCES AVAILABLE UPON REQUEST

THE AFIB REPORT

*Welcome to the second issue of **The Afib Report**. Our aim is to keep you abreast of new discoveries concerning atrial fibrillation but, even more important, to ultimately help find a solution to the problem. In order to maximize your benefits from **The Afib Report** I would highly recommend that you read my earlier report entitled “Lone Atrial Fibrillation: Causes and Management” (www.yourhealthbase.com/atrial_fibrillation.html). Although this report covers many of the basics of LAF some elaboration is needed in order to gain a fuller understanding of the problem and be able to interpret the significance of new findings. So the next few issues will be a combination of basic background information and the latest news. If you are already conversant with cardiology and electrophysiology you can skip these sections.*

Cardiology 101

The heart, apart from what other mythical and emotional characteristics we may ascribe to it, is basically a living pump. It is one of the hardest working organs in the body; it contracts and expands about 100,000 times every day. It supplies a blood vessel network 96,000 kilometers long and pumps in excess of 10,000 liters of blood around the body every single day. The heart has four chambers, the right *atrium*, the left atrium, and the right and left *ventricles*. The atria are situated above the ventricles with the right atrium being connected to the right ventricle through the *tricuspid* valve and the left atrium being connected to the left ventricle through the *mitral valve*.

Returning blood enters the right atrium from the superior and inferior *vena cava*. It is propelled onward by contraction of the muscular tissue of the atrium and then enters the right ventricle which pumps it through the lungs and back to the left atrium through the *pulmonary veins*. The passage through the lung capillaries eliminates carbon dioxide and other waste products and re-oxygenates the blood. From the left atrium the “rejuvenated” blood flows through the mitral valve into the left ventricle which contracts with enough force to pump the blood through the *aorta* into the smaller arteries and capillaries, where the actual nutrient and oxygen exchange with individual body cells takes place, and then back to the heart through the veins. Immediately after exiting from the heart the aorta branches off into the right and left *coronary arteries* which supply the heart itself with fresh blood and the nutrients it requires.

Electrophysiology 101

The fibers and individual cells of the heart muscle are unique in that they are able to contract and relax spontaneously. Thus the heart will beat, albeit at a very slow rate, even if it receives no external stimuli. Normally though, the operation of the heart is under the control of the autonomic nervous system.

The autonomic nervous system (ANS) controls the body’s internal organs including the heart and digestive system and is responsible for regulating blood pressure. It has its origin in the hypothalamus region of the brain from where it divides into two branches – the sympathetic (*adrenergic*) branch and the parasympathetic (*vagal*) branch. The neurotransmitter used in the adrenergic branch is *norepinephrine* (noradrenaline); the parasympathetic system uses *acetylcholine* to transmit its messages.

The adrenal gland is an outgrowth on the adrenergic branch and its medulla (the inner part of the gland) produces two neurotransmitters, norepinephrine and *epinephrine* (adrenaline) collectively known as

catecholamines. Norepinephrine is normally synthesized (from the amino acid tyrosine) right at the nerve endings as needed, but when the body is under excessive stress the adrenal medulla kicks in and produces large amounts of both epinephrine and norepinephrine as part of the "fight or flight" reaction and this, as we shall see later, can spell big trouble for afibbers.

The autonomic nervous system is responsible for maintaining the body's inner balance (homeostasis). It does this by continuously adjusting the secretion of the two neurotransmitters, norepinephrine (from the sympathetic nerve endings) and acetylcholine (from the parasympathetic nerve endings). Norepinephrine speeds up muscle contractions and heart rate while acetylcholine slows them down. Constant maintenance of a finely tuned balance is necessary to keep the body functioning at its optimum.

Maintaining a blood pressure sufficient to ensure an adequate blood supply throughout the body but low enough to avoid bursting small capillaries in the brain is perhaps one of the most important tasks of the autonomic nervous system. The cardiac control center of the ANS constantly receives input from *baroreceptors*. These specialized muscle fibers are located in the walls of the heart and the major arteries and they "measure" the blood pressure by stretching and relaxing as the blood flows past them. A lower than desired pressure will cause the ANS to activate the sympathetic nervous system and thus make the heart beat faster while too high a pressure will activate the parasympathetic system.

The atria are suffused with nerve endings from the sympathetic system which also has a direct connection to the *sinoatrial* (or sinus) *node* located at the junction of the superior vena cava and the right atrium. Although nerve endings from the parasympathetic system can also be found throughout the tissue most of the parasympathetic activation takes place at the sinoatrial (SA) node.

Impulses from the SA node spread across the atrium and cause it to contract and relax at a rate of about 70-75 contractions (beats) per minute. When the impulses reach the *atrioventricular* (AV) *node* located near the tricuspid valve the cells of the *bundle of His* are activated. This is followed by activation of the *Purkinje fibers* resulting in contraction of the ventricles.

Sounds complicated? It is, but unfortunately it is absolutely essential to have a clear understanding of the interaction between the heart and the autonomic nervous system if we are to comprehend and eventually vanquish arrhythmias. To sum up, the heart's operation is controlled by the "cardiac control center" of the autonomic nervous system. This center receives input from baroreceptors regarding blood pressure and then activates either the sympathetic or the parasympathetic branch in order to bring the blood pressure into the desired range. An activation of the sympathetic system will speed up the heart and increase blood pressure while activating the parasympathetic system (increasing vagal tone) acts as a brake by slowing the heart and decreasing blood pressure.

The autonomic nervous system, of course, controls several other involuntary body functions and responds to many other stimuli than just blood pressure. However, in so far as arrhythmias and atrial fibrillation are concerned, the heart rate connection is clearly the most important.

The ANS is only capable of maintaining homeostasis within certain limits. Exposure to stressful stimuli such as low blood sugar, extreme temperatures or a visit from the tax inspector can throw it off balance and as a result impair the smooth functioning of the internal organs including the heart. If the heart tissue and SA node are sensitive to autonomic nervous system disturbances it is quite possible that an atrial fibrillation attack or other arrhythmia will result.

Atrial Fibrillation 101

Atrial fibrillation is caused by a dysfunction of the heart tissue or nodes, by a dysfunction of the autonomic nervous system or by a combination of both. As we saw earlier, individual heart cells are capable of "beating" on their own outside the control of the autonomic system. Sometimes agglomerations of very active cells form and create a focus for so called *ectopic beats* (beats originating outside the SA node). The junction between the left atrium and the pulmonary vein is a particularly popular spot for these "rogue" cell

agglomerations and some arrhythmias can be successfully treated by removing them with radio frequency ablation. If the ectopic beats become very frequent they may run together and create atrial fibrillation.

Atrial fibrillation basically involves a chaotic movement of electrical impulses across the atria and leads to a loss of synchrony between the atria and the ventricles. Once an attack has begun the atria may quiver or fibrillate at a rate as high as 300 to 600 times per minute. This causes a very inefficient filling and emptying of the atria; the chaos is transferred to the ventricles causing them to lose their regular rhythm and begin to contract fast and in a totally irregular manner. This is what gives rise to the fast and irregular pulse rate felt during an AF attack (90-160 beats/minute).

Atrial fibrillation in itself is not a disease, but rather a symptom of some other disorder of the body. Atherosclerosis, angina, valvular (rheumatic) heart disease, hypoglycemia, hyperthyroidism, anemia, pheochromocytoma, strenuous exercise, binge drinking, consumption of tyramine-containing foods, and exposure to mental or physical stress can all trigger atrial fibrillation. All these conditions have one thing in common – when active they are associated with an excessive release of norepinephrine and, in some cases, epinephrine as well.

Lone (primary) atrial fibrillation (LAF), by definition, is atrial fibrillation without underlying heart disease. So it stands to reason that this arrhythmia is primarily related to a dysfunction of the autonomic nervous system. The dysfunction can be an overactive sympathetic system or an underactive parasympathetic system or perhaps an overactive parasympathetic system followed by a too vigorous correction by the sympathetic system. These are finer points that may be covered in a future issue of The Afib Report. For now suffice it to say that atrial fibrillation ultimately involves an excessive release of norepinephrine from the autonomic nervous system.

Control of Norepinephrine

So what does this mean in terms of preventing lone atrial fibrillation? Clearly the key is to control or inhibit an excessive norepinephrine release. The simplest way to do this is to assist the autonomic nervous system to stay in balance by avoiding trigger factors. Staying away from alcohol, caffeine and tyramine-containing foods, and avoiding excessive physical and emotional stress are a good start. Another very important preventive measure is to avoid large dips in blood sugar levels. There are at least two documented cases of atrial fibrillation associated with *hypoglycemia* (low blood sugar) and probably many more unrecorded ones(1,2).

Hypoglycemia manifests itself as an excessive drop in blood sugar levels 3 to 6 hours after eating. A hypoglycemic episode is treated as a major emergency by the autonomic nervous system; it proceeds to dump vast quantities of epinephrine into the blood stream in order to prompt the liver to release glucose for use by the starving brain. The chaos created by this sequence of events will more than likely result in an AF attack. Hypoglycemic episodes can be avoided by eating small meals throughout the day (including before bedtime) and eliminating sugar and sugar-containing products as well as white flour-based products from the diet. It is also important to base the diet on low glycemic index foods.

Norepinephrine Inhibition and Paroxetine

The idea of inhibiting norepinephrine secretion by pharmaceutical drugs is an intriguing one. Recent work done by Dr. Jack Gorman, MD at Columbia University concludes that the antidepressant paroxetine (Paxil) may normalize heart rate variability and, in turn, help prevent panic attacks(3). Panic attacks, in many respects, are similar to LAF attacks. I actually tried paroxetine a couple of years ago and found that 20 mg/day did indeed significantly reduce the frequency of my attacks.

Prior to starting on paroxetine I experienced a LAF attack every 7 to 14 days and each one lasted between 12 and 17 hours. I had an attack 10 days after starting the paroxetine, but then went 55 days without one.

The interval before the next one was 37 days, but this attack lasted 20 hours. Then it was 76 days without an attack, but when it occurred it lasted 108 hours. The next one came 40 days later and lasted 58 hours. However, it took the form of severe *bradycardia* with heart rates as low as 39 beats/minute. I later came across an article by Erfurth et al [ECG changes after paroxetine: 3 case reports. Nervenarzt 1998 Jul; 69(7):629-31] that reported 2 cases of severe bradycardia in connection with paroxetine treatment. So to make a long story short, I found paroxetine very helpful in the beginning, but had to discontinue it after the bradycardia episode which I found very scary. It may be that paroxetine in smaller doses (5-10 mg/day) may be helpful and have fewer side effects. If anybody tries it please let me know how it works out.

News

Researchers at the Mayo Clinic report that sildenafil (Viagra) can increase sympathetic nerve activity by as much as 141% and norepinephrine release by more than 30%. Definitely not a good choice for afibbers(4).

Medical doctors at the St. James University Hospital in the UK have found that drinking large quantities (500 ml) of water significantly increases sympathetic activity. Athletes may want to consider this when they rehydrate after exercise(5).

That's all for now. I hope you found this issue of The Afib Report useful. In future issues we plan on covering the benefits of supplementation as well as the role of a magnesium deficiency and amalgam dental fillings in the promotion of lone atrial fibrillation. We will also take a detailed look at antiarrhythmic drugs, RF ablation surgery, and the maze operation. Please let me have your feedback.

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