Cases from the Periphery

Though in general good health, I have had two life-threatening medical conditions which were exacerbated by self-medication. I present these cases with a plea for caution in areas where orthomolecular medicine has not yet made specific recommendations.

Enlarged prostate runs in my family. I started recording serious difficulties in July 1991 when I was 62 years old. I made diary entries of my sleep interruption, such as:
5 Sept 1991 12:50 - 2:30 4:30 6.30 (but free flowing)
10 September 1991 11:30 1:50 4:00 - 6.30 (with coaxing)

I developed a system of ranking urination experiences from 10 "perfect flow" (never experienced) to l "small amounts only after trying and failing for sometime." There were many 1-1-1-5 nights, and once almost a zero crisis. I discovered rules like "Pee when you don't want to!" and "Cohabitate, cohabitate, then you can pee." But I couldn't cohabitate all the time. This was the era of all the Proscar advertisements, but I knew about saw palmetto and pygeum. I did see a urologist in September 1993 when I had a slight bleeding problem. He said my prostate was as big as an orange and if we didn't do anything now I'd soon be back. I told him saw palmetto and pygeum were my method. He said some of his Croatian patients used pumpkin seeds. I tried those too, and I was living with it OK, I thought. In fact, by the time of my retirement at 65 in 1994 1 seemed to have conquered the problem of urgency. There was still some difficulty getting much out, but that horrible urgency had gone. Diary entry: "mirabile 12:20 - 7:45. Here's to saw palmetto!"

Then came the real crisis, 26 September 1996: much bleeding, so enormous I had to throw myself on the mercy of the medical profession. The urologist couldn't even get a catheter in. "You've been peeing on will-power alone," he said. "The time for talk is over." I was in the operating room within a few days as an emergency patient. What had happened was that my bladder had been expanding and at some point lost a lot of its elasticity. That was the reason for the lack of urgency. I was now in dire straits, for the bladder was about to rise further, up against the kidneys, and that would be a quick end. But for the hemorrhaging I would have stubbornly killed myself taking two European strength saw palmetto tablets a day that were simply not doing the job I thought they were. The quantity of saw palmetto needed to control BPH is not known. Orthomolecular medicine has not faced that question. So I was saved by the surgeon's knife.

The second instance was to do with a thrombic embolism episode on 17 November 1998 which caused pneumonia but, after a week's recuperation in hospital, was not otherwise disabling. Of course I was put on warfarin for a year. During that time I was puzzled (if nobody else seemed to be) by what might have caused the blood clot. Though I had had no painful leg symptoms the doctors were content to think it was a transatlantic flight two months previous. I insisted on a DNA test. The hematologist said, "If you've lived this long, you don't have a genetic mutation." But it turned out that I did have one, one that doesn't have too much effect normally on the coagulation cascade. Factor V Leiden is still very much of a mystery. Statistics supposedly indicate that there is a slight percentage greater than average likelihood of a blood clot during a lifetime. (It was my mother who had it, I'm sure, and she lived healthily to 93.) The chemistry is known but there is no practical guidance for avoiding the effects of Factor V Leiden specifically.

I think I know what happened. At the time I had been taking a lot of alfalfa tablets and wheat germ as well as the usual copious green-leaf salads at our table. That was a great deal of vitamin K, which is a major factor in coagulation. I asked the hematologist point blank if overabundance

of vitamin K could have caused clotting. "No" he said "The body gets rid of excess K." He didn't say "normally"-but that's what he meant. There have not, according to my research, been any experiments to see if there could be an excess of K in abnormal circumstances, with Factor V Leiden, say.

I was taken off warfarin at my request by a new hematologist who read the odds in a different way; she considered that there was more risk of fatality from bleeding with warfarin than from blood clots without it. Naturally, am careful about vitamin K now. Why was I taking a lot of alfalfa in the first place? I don't know. I'd probably read an article in a health magazine.

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Selenium, EPA and Schizophrenia

Perhaps as the result of an allergy to latex,[1] schizophrenics frequently display high levels of adrenochrome in their urine.[2] This is created by the oxidation of adrenalin to adrenochrome. Schizophrenics also are typically very deficient in selenium, because this trace element is required to produce glutathione peroxidase, which acts as a natural defence against adrenochrome. It has been shown that this deficiency in selenium and hence in the selenoenzyme glutathione peroxidise appears linked to brain essential fatty acid abnormalities which may account for the atrophy and increased ventricle-brain ratios identified in chronic schizophrenics by Buckman and co-workers.[3] Confirmation of a brain selenium-fatty acid link recently has been discovered by Celik and colleagues.[4] These researchers have shown that, in lambs, dietary differences in vitamin E and selenium have a major impact on the fatty acid composition of the brain. Specifically, diets enriched in selenium and/or vitamin E increase eicosapentaenoic, total unsaturated fats and highly unsaturated fatty acids in the brain. Conversely, they result in a reduction of brain myristic, pentadecanoic, palmitic, linoleic and total saturated fatty acids. It seems likely, therefore, that in schizophrenia, as adrenochrome excess depletes the body of selenium and other antioxidants, significant changes occur in the fatty acid composition of brain tissues. The importance of this has been established by Horrobin[5] who has shown, in five trials, that eicosa-pentaenoic acid supplementation can produce substantial improvement in schizophrenia, without the usual side effects of sedation, movement disorders, weight gain or dysphoria. It appears, therefore, that an optimum treatment protocol for many schizophrenics would include niacin, selenium, vitamin E and eicosa-pentaenoic acid.

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Alzheimer's Disease and Aluminum Soil Concentration

In the Journal of Orthomolecular Medicine in 2000 Foster' wrote a compre-

hensive and compelling article that provides evidence that excessive aluminum may be a causal factor in Alzheimer's disease. The risk of persons in Ontario developing Alzheimer's is 2.5 times greater in communities that have more than 100 mcg per litre aluminum in the drinking water. Foster also pointed out that miners exposed to aluminum dust have cognitive deficits and other neurological problems. However, there have apparently not been any published studies that have related Alzheimer's incidence or prevalence to the amount of aluminum in the soil.

The Alzheimer's disease rate for the U.S. states were obtained from the United States Department of Health and Human Services2 2 . The aluminum soil concentration was reported by Hansford, Shacklett, Hamilton, Boerngen and Bowles 3 . Their document had circles for each location in which soil aluminum concentration was determined. There were five types of circles that differed a's a function of concentration range. We assigned a 5 to the highest range, a 4 to the next highest range, a 3 to the third highest range, a 2 to the fourth highest range, and a I to the lowest concentration range. The mean was determined for each state except for Alaska, and Hawaii. These two states did not have soil aluminum concentration provided.

The product-moment correlation coefficient was .33 (p = .0 12) for all ages/all races death rate, .27 (p = .03 5) for all ages/ White death rate, .3 1 (p = .020) for > 65/ all races death rate, .28 (p = .03 1) for >65/ White death rate, .25 (p = .047) for all ages/ all races age-adjusted death rate, .19 (p 109) for all ages/White age-adjusted death rate, .27 (p = .03 3) for >65/all races-age adjusted death rate, and .23 (p = .064) for >65/ White age adjusted death rate. There were four death rate variables, all with "Black" persons, for which a correlation was not computed. For reasons not understood this information was not given for most of the states.

Although the above correlations do not permit inferences upon the mechanisms involved in the relationships, they do add to the already strong argument of Foster that aluminum is implicated in Alzheimer's Disease.

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