used in these studies, and to Professor Geoffrey P. Mason, University of Victoria, for doing the statistical analysis.

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To the Editor. Because of the apparent “epidemic” that we are all seeing of coronary heart disease, and also because of the simultaneous interest of the American public in vitamin E, early in 1972, I decided to review the available medical literature on the subject. I also included some personal correspondence with some of the most famous proponents of vitamin E therapy for a multitude of medical conditions. The conclusions of my reviews and correspondence were ambiguous. Because the only conclusion that I could draw from my studies was that vitamin E was harmless, even though its benefits were questionable, I decided to place a number of my patients on vitamin E therapy. These patients included some with arteriosclerotic heart disease, but mostly men in their mid-thirties and early forties, who were in excellent health at present and in the past. I included myself and my partner in the study. Vitamin E, in the form of n-alpha tocopherol, was the formulation used, and the starting dose was 800 IU daily. The study was only an informal one and not controlled by any means.

After about one week on the medication I began to feel an amazing weakness and fatigue as if I were suffering from a severe influenza-like syndrome—the symptoms stopped after withdrawal of vitamin E. Still thinking that I had likely suffered a viral illness, I resumed the vitamin E, and his symptoms promptly disappeared on the following day. By this time, virtually all the patients and colleagues whom I had instilled on the therapy were calling me and relating the same thing, and had to stop their vitamin E. Some, like myself, were able to tolerate the vitamin at 400 IU daily, with only minimal fatigue.

Now, in the course of my day-to-day practice of general and internal medicine, I have come to recognize many people who have become obsessed with the megavitamin concept for “good health,” and these people take literally handfuls of vitamins daily—usually the vitamin B complex, vitamin C and vitamin E. Many of these young people have been coming into my office complaining of very severe fatigue. Very comprehensive work-ups are done along the traditional lines: physical examination, complete blood count, urinalysis, stool examination, chest x-ray study, etc. These tests are inevitably negative, and the overpowering fatigue responds promptly to the withdrawal of vitamin E.

I wish to report this observation, though it is uncontrollable, because I cannot find it anywhere in the literature, and because my partner and I see it over and over—and in the vitamin-conscious age in which we live, we shall see it more and more. We included it in our discussion of vitamin E in our recent book. However, we have no conclusive evidence to support the use of vitamin E for any marked cross-reactivity between T. gondii and isospora is provided by Doby and Beaucournu, who summarize earlier records (including those relating to isospora species of animals), with two exceptions. Moreover, it is apparent from a number of recent papers that prior infections of isospora in cats do not influence susceptibility to T. gondii or vice versa.

Isosporan oocysts indistinguishable from those of T. hominis are shed in man after ingestion in uncooked meat of tissue cysts of Sarcocystis suihominis from cattle and S. miescheriana from swine. As incidental to collaborative experimental work (Draper, Garnham, Hutchison, Killick-Kendrick, Markus and Sim) on sarcocysts in animals, to be published elsewhere, human serum specimens were tested in Draper’s laboratory, with use of S. suihominis antigen prepared by liberating cystozoites from macroscopic cysts extracted from infected bovine diaphragm. Our preliminary findings in the indirect fluorescent-antibody test suggest an absence of cross-reaction between S. suihominis (in the light of present knowledge, a stage in the life cycle of T. hominis) and T. gondii; persons reacting with what is apparently a positive titer (found up to a dilution of 1:1024) may prove to be (or have been) T. hominis carriers. Further experimentation—e.g., inactivation of serum of adults—is indicated to assess the value of these results. Doby and Beaucournu, using T. gondii antigen and serum of known carriers of T. hominis in the indirect fluorescent-antibody-test, also demonstrated that the two protozoa are immunologically distinct.

As far as the tissue stage of sarcocystis is concerned, the specificity of various serologic tests for T. gondii has been confirmed by earlier workers who used sera from man and monkeys infected with sarcocystis species and serum of sheep with Steniera infections. Wallace, in studying the life cycle of an animal parasite that was either an unusual strain of T. gondii or a species of sarcocystis, found that this organism and T. gondii share a few antigens but that there is little or no cross-immunity.

It must be concluded that there are as yet no grounds for questioning the reliability of routine serologic tests for human toxoplasmosis.

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