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Chronic fatigue syndrome

Drs. Gerald H. Ross, William J. Rea and Alfred R. Johnson (*Can Med Assoc J* 1989; 141: 11-12) find it regrettable that I should object to the term "chronic fatigue syndrome" (*Can Med Assoc J* 1989; 140: 1016), particularly as I appear to be at odds with the US Centers for Disease Control (CDC). What I find regrettable is that the originators of this term[1] (some of whom were working at the CDC) had thought the illness to be due to the Epstein-Barr virus (EBV); hence, the term "EBV syndrome", which has persisted, with unfortunate consequences due to overdiagnosis of infection with this hapless virus.

That such overdiagnosis is apt to recur with regard to the same illness under another name ("chronic fatigue syndrome") is evidenced by Ross and colleagues' implying that psychologic illnesses are merely those for which organic causes have not yet been discovered. Ross and colleagues are obviously unaware that a psychiatrist's first duty is to determine whether an illness is primarily organic (which includes environmental causes) or psychologic and to establish a positive psychiatric diagnosis. To come to a psychiatric diagnosis merely by exclusion of organic conditions is psychiatric sacrilege.

Having ascertained that a "substantial" percentage of cases of "chronic fatigue" arise from the environment, Ross and colleagues now appear to be intent on doing the same for panic disorder. Personally, I have not encountered a case of chronic fatigue or panic disorder due *primarily* to environmental factors, although I have witnessed the worsening of primary psychiatric disorders by organic factors (including environmental) and vice versa. This does not imply, however, that an environmental cause does not exist, but that it is rare in the general population. (As a former industrial physician I am aware that certain inhalants can do more than cause chronic fatigue or panic attacks.) Nor do I dispute that there is a constitutional predisposition to panic disorder in addition to a learned response (if in doubt, panic).

I hope that an iatrogenic component to panic disorder, as in the ordering of a useless serology test for "EBV syndrome", will not aid and abet the ever-increasing tendency to self-diagnosis.

Meanwhile, the general public will welcome the remarks of Ross and colleagues, because they make both physician and patient feel more comfortable (a psychiatric diagnosis makes everyone feel uncomfortable) in today's "age of entitlement".

Sigmund Freud, however, must be turning in his grave.

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[Correspondence on this issue is closed until new information about this perplexing syndrome is available. — Ed.]

An analysis of birth weight by gestational age in Canada

Tye E. Arbuckle and Dr. Gregory J. Sherman, having reviewed Canadian national data for birth weight and gestational age for the years 1972 and 1986, report that the median weight of babies born at term has increased by about 180 g over the 14-year period but that the weight for gestational age among babies born before term has not changed (*Can Med Assoc J* 1989; 140: 157-160, 165). This discrepancy would require that fetuses destined to be born at term grew more rapidly in utero in 1986 than in 1972 but that fetuses destined to be born before term grew at the same rate. We think this is unlikely and suggest an alternative explanation.

We hypothesize that earlier Canadian national data, like data from national surveys, suffer from inaccurate ascertainment of gestational age; specifically, erroneous attribution of preterm gestational age to some full-size babies who were, in fact, born at term.[1] We hypothesize further that this bias, which skews upward the distribution of birth weight among "preterm" babies, was more prevalent in the 1972 data than in the 1986 data. This hypothesis would suggest that the apparent absence of a secular increase in birth weight among babies born before term is an artefact caused by an overestimate of birth weight for gestational age among those born before term in 1972 but not those born in 1986.

To test this hypothesis we examined the intercentile distance (10th to 50th versus 50th to 90th percentiles) for birth weight in the data reported by Arbuckle and Sherman for 1972 and 1986. There is indeed a skewing upward of the birth-weight distribution throughout the gestational ages below 36 weeks, and it is much less in 1986 than in 1972. This finding is consistent with

the hypothesis that some "preterm" babies born in 1972 were in fact born at term and that this systematic error was reduced in the 1986 data. We infer, therefore, that there has been a secular increase in birth weight among both preterm and term babies and that the reported increase among term babies may actually be an underestimate of the true secular trend.

Further support, or lack thereof, for this hypothesis could be provided by examining the sex ratio of heavy "preterm" babies born in 1972 and 1986. Milner and Richards,[1] in their analysis of national data for England and Wales, ingeniously showed that the sex ratio of a subpopulation of heavier, supposedly preterm babies was characteristic of that of babies born at term. It would be interesting to know if the same were true in 1972 but not 1986 for the Canadian babies.

Our Hamilton population-based study of size at birth[2] employed special measures to ensure the accuracy of gestational age. In that study a skewing upward of birth weight distribution among preterm babies was not evident; moreover, the 10th to 90th intercentile ranges for birth weight among preterm babies were substantially less than those for the 1972 Canadian national data.

We agree with Arbuckle and Sherman that a survey of birth weight by gestational age should be repeated at intervals to account for secular trends. The increasingly accurate assessment of gestational age will provide increased precision and accuracy in determining any true secular trend in size for gestational age.

Arbuckle and Sherman do not discuss the mechanism of the secular increase in birth weight. We found that the major determinants of birth weight in our population (after controlling for gestational age and sex) were the positive effects of increasing maternal prepregnant weight and weight gain during pregnancy and the negative, dose-related effect of maternal cigarette smoking during pregnancy.[3] In 1972, women in Canada were commonly under pressure from their physicians to limit weight gain during pregnancy. Since then there has been a relaxation of criteria for weight gain and increasing awareness of the hazards to the fetus of maternal cigarette smoking. It would be important to quantitate the national trends of such major determinants of fetal growth. Modest increases in maternal prepregnant weight and in weight gain during pregnancy, coupled with a decrease in the amount of cigarette smoking during pregnancy, could readily account for the magnitude of the increase in birth weight reported by Arbuckle and Sherman.

In addition, one must consider the possible effect of antenatal screening for fetal growth retardation. Could this result in the early delivery of enough growth-retarded fetuses to contribute to a secular trend favouring increased birth weight among babies born at term and the opposite effect among those born before term? Evidence consistent with the latter effect obtained from hospital-based data has been reported from England,[4] but we are unaware of similar data based on a national survey.

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Making the use of isotretinoin safer

The teratogenicity of isotretinoin (Accutane) is causing alarm. This drug has become widely used in the treatment of severe acne vulgaris. Reports indicate that from 35%[1] to 66%[2] of women use no contraceptive measures while taking isotretinoin in spite of warnings from physicians, pharmacists and the drug company in the package literature.

One solution is not to prescribe this highly effective but teratogenic drug to women. We might, however, learn from our colleagues in Bogota that regularly treat patients who have actinic prurigo (a severe familial photodermatitis) with thalidomide,[3] a well-known teratogen. The thalidomide is dispensed to women only after they have received an injection of medroxyprogesterone acetate, which makes them sterile for 2 months. A 2-month supply of thalidomide is then dispensed. The patient returns for reassessment in 2 months.

This appears to be a safe method of administering a teratogenic drug and may be desirable when one is prescribing isotretinoin for fertile women.

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