


Competing interests Jonathan E. Prousky is a consultant for Swin Herbal Remedies, Ltd., a company that sells nutritional supplements.

DOI:10.1503/cmaj.1041424

[One of the authors responds:]

We agree with Joel Ray and David Cole regarding the relative practical value of serum total homocysteine and methylmalonic acid in elderly patients with suspected cobalamin deficiency. Although testing for serum holo trancobalamin is not routinely available in many countries, we believe that it may be appropriate in future as a routine clinical test for cobalamin deficiency. However, to date, a consensus on the definition for cobalamin deficiency, especially among elderly patients, has not been achieved.¹ Thus, in our experience, serum total homocysteine is currently a helpful, inexpensive indicator of true (tissue) cobalamin deficiency, as suggested in our article.²

We agree with Peter Wetterberg’s comments on oral cobalamin. However, the usefulness of oral cobalamin therapy has only recently been documented, starting in 1995, with studies that meet the criteria of evidence-based medicine.¹⁴ We read with great interest Jonathan Prousky’s comments, although we have no experience with the therapies he describes. Nevertheless, we believe that this information indirectly supports the concept of food-cobalamin malabsorption.³

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References

Competing interests: None declared.

DOI:10.1503/cmaj.1041701

Short ACTH stimulation test for adrenal reserves of cortisol, not adrenal function

T he classical description of Addison’s disease in a 15-year-old girl¹ is a timely reminder of this well-known but uncommon disorder. In their description of the investigative work-up and discussion, Chantelle Barnard and associates¹ imply that the short adrenocorticotropic hormone (ACTH) stimulation test is diagnostic of primary adrenal insufficiency. This is a common misapprehension.

In the test, an intravenous (or intramuscular) injection of 250 μg of synthetic ACTH (tetracosactrin) results in release of preformed cortisol from adrenal stores, which is measured in the serum 30 (and/or 60) minutes later and compared with the baseline concentration. An abnormal response (a serum cortisol peak below 550 nmol/L or an increment of less than 200 nmol/L from baseline or both) identifies adrenal insufficiency but cannot distinguish Addison’s disease (primary adrenal failure) from secondary hypoadrenalism. In pituitary disease (ACTH deficiency), for instance, the result of the test may be abnormal because of reduced stores of cortisol, even though the adrenal glands themselves have normal biosynthetic and secretory func-

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tion. In this situation, a prolonged ACTH stimulation test using 1 mg depot tetracosactrin, with serial measurements of serum cortisol concentrations over 24 hours, would allow sufficient time for the otherwise healthy adrenal glands to mount an adequate cortisol response, whereas the test result would be abnormal in Addison’s disease (particularly in preclinical disease, in which the result of the shorter test may be normal).

As the authors correctly point out, the hyperpigmentation seen in Addison’s disease reflects increased ACTH and melanocyte-stimulating hormone due to dysinhibition of the hypothalamic–pituitary axis, which is in turn a result of low circulating cortisol concentrations. This is a relatively specific sign, and there is therefore little doubt that the hypoadrenalism in the patient described was due to primary adrenal failure. However, an elevated plasma ACTH concentration at baseline reliably distinguishes between primary and secondary causes and would have provided incontrovertible evidence of Addison’s disease; besides being far simpler than the prolonged ACTH stimulation test.

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References


[The authors respond:]

We agree with the interpretation of the short ACTH stimulation test described by Deeba Syeda and associates. In the case that we described, a long ACTH stimulation test was unnecessary because of the clinical picture, especially the hyperpigmentation and the markedly increased ACTH level (285 [normally less than 18] pmol/L).

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Reference


Russia and social “reform”

Paul Webster, in his description of the Russian government’s plans “to ‘monetize’ its social commitments” through reform of a variety of Soviet-era entitlements, fails to cover some important details.

It is true, as stated by a health care analyst from Moscow, that “Russia’s system of privileges was never designed to support the poor.” But the estimate that “the poorest 10% of the population receive 4% of existing benefits, while the richest 10% receive 20%” is nothing more than speculation. The government used such estimates to prove that the system of natural entitlements must be changed. Shortly after the new law was enacted, when the government started to check how many poor people were eligible for “monetization” of their entitlements, it found that the numbers had been underestimated by up to 30%. Now nobody knows how many people are entitled to monthly subsistence. Of course, this detail is not relevant to the government; the president set the amount without any supporting research.

Economist Mikhail Zurabov, who chairs the Health and Social Development Ministry, is leading the transformation of the most attractive health care institutions to an intermediary propriety state, which would allow them to be privatized in the short term. Former health minister Yuri Shevchenko, during his years in cabinet, created the huge “national hospital,” using federal money to equip it; he then slipped from his ministerial position to that of director of the hospital.

At the other end of the health care spectrum, the new legislation prevents municipal health care services from having access to any monies from the federal or regional budgets; as a result, the wages of staff as well as health care expenditures must come from the limited resources of the municipal bodies. Finally, in the budget for 2005 military spending will increase by up to 30%, but there will be no increase for health care.

The title of Webster’s article is absolutely correct: “Reforms mean [that] Russians lose free health care.” In January people all across Russia took to the streets to protest against the “monetization” of their entitlements. Members of parliament have called on the government to resign, but in fact, by approving the proposed regulations, they share responsibility for these reforms.

The problem of natural entitlements has become the starting point for a huge crisis.

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Reference