Letters to the Editor

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Ticlopidine, Trials, and “Torture”

To the Editor:

The title of the editorial by van Gijn and Algra,1 “Ticlopidine, Trials, and Torture,” may leave the impression that real torture is a phenomenon of the distant, cruel past, and that the word “torture” can now be used to describe any misconduct towards persons or, as in this case, scientific data.

Torture, however, is a sad and unacceptable reality of today in many countries. In the United Nations’ convention against torture the following definition is given: “The term torture means any act by which severe pain or suffering, whether physical or mental, is intentionally inflicted on a person for such purposes as obtaining from him or a third person information or a confession, punishing him for an act he or a third person has committed or is suspected of having committed, or intimidating or coercing him or a third person, or for any reason based on discrimination of any kind, when such pain or suffering is inflicted by or at the instigation of or with the consent or acquiescence of a public official or other person acting in an official capacity.”

According to the latest Amnesty International report, government-sanctioned torture took place in 74 countries during 1993.

The word “torture” should not be used as a negatively loaded metaphor in the discussion of researchers’ handling of scientific data.

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Reference

Response

We fully understand Dr Marcussen’s point of view. Doctors and others who, like him, are actively committed to the eradication of torture in its cruel, literal sense must find it flippant if others extend the figurative sense of that word to statistical ill-treatment of scientific observations.1 We can only plead that we left no room for misunderstanding and that medical parlance allows the metaphorical use of other malicious-sounding terms (eg, “steal phenomenon,” “misery perfusion,” “cell death,” “aspirin wars”). We feel of mishandling of scientific data.

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Utrecht, The Netherlands

Reference

Alteration in Serum Pituitary Hormone Levels in Postmenopausal Women With Stroke

To the Editor:

The documentation by Pepper et al1 of hypogonadotropic hypogonadism (HGH) in elderly females with stroke is in accord with the findings in an 85-year-old woman whom I followed up for 20 months (Table).

In August 1991 an 85-year-old woman presented with primary hypothyroidism 5 months after a stroke that had left her with residual left hemiparesis. She was otherwise well enough to attend the day hospital for physical therapy. Serum thyroxine (T4) was 1.01 μg/dL, thyroid-stimulating hormone (TSH) was 29.4 μU/L, luteinizing hormone (LH) was 5.8 mIU/mL, follicle-stimulating hormone (FSH) was 22.1 mIU/mL, and serum estradiol was <24.5 pg/mL. The short synacthen test indicated a peak serum cortisol of 36.8 μg/dL, and there was no evidence of pituitary tumor or cerebral infarction on computerised axial tomography. The chest X-ray was also normal. As a result of T4 replacement therapy, the patient became euthyroid in February 1992, with a T4 of 6.68 μg/dL and a TSH of 2.3 μU/L, but the biochemical stigmata of HGH persisted, with an LH of 8.3 mIU/mL and an FSH of 21.7 mIU/mL. In July 1992, when her serum T4 was 8.03 μg/dL and TSH was 3.7 μU/mL, the LH-releasing hormone (LHRH) test was performed using a 100-μg intravenous bolus of LHRH. Peak responses were obtained at 60 minutes, with an LH of 27.82 mIU/mL and an FSH of 30.62 mIU/mL. Biochemical review in April 1993, after 14 months of euthyroidism, yielded the following results: T4, 6.46 μg/dL; TSH, 2.99 μU/L; LH, 8.82 mIU/mL; FSH, 26.26 mIU/mL; and estradiol, 19.07 pg/mL.

The original diagnosis in this patient was primary hypothyroidism with coexisting ischemic hypopituitarism. The feature that raised the index of suspicion for the latter diagnosis was the fact that the serum TSH, although higher than the upper limit of 11 μU/L generally associated with central hypothyroidism,2 nevertheless did not reach the high levels usually associated with the degree of severity of primary hypothyroidism seen in this patient. In view of the patient’s recent stroke, ischemic damage to thyrotrhops and gonadotrophs was considered to be the most likely mechanism for coexisting hypopituitarism. The alternative explanation is that offered by Pepper et al1 who suggest that cerebrovascular disease may interrupt neurotransmitter pathways involved in the secretion of gonadotrophins.

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References
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