



## Human chorionic gonadotropin in weight reduction

Dear Sir:

Space has been given in medical publications to the use of human chorionic gonadotropin (HCG) in obesity. The scepticism of most academics is matched only by its commercial success and like most practitioners who believe in its value, I realize that trials to date do not substantiate our clinical impressions.

However, as the twin realities of a high perinatal mortality rate and recurrent periods of insufficient food were always with man and his precursors the following physiological adaptations during pregnancy would seem logical.

- 1) Increased fat storage capacity during pregnancy involving temporary suppression of the homeostatic mechanisms (adipostat) that normally keep body mass constant despite often wide discrepancies between caloric intake and energy expenditure (1, 2).
- 2) Increased intake of high calorie foods when available readily stored as fat. (Pregnant females often develop cravings for sweet foods and Frank (3) reported increased appetite in some patients having HCG but not the placebo).
- 3) More efficient absorption of food and decreased faecal energy excretion. (Constipation (? prolonged intestinal transit time) is commonly observed in pregnancy and in patients receiving HCG).
- 4) Decreased energy expenditure by metabolic efficiencies and decreased physical activity. (Euphoria, lethargy, and improved sleeping pattern reported in pregnancy and in patients receiving HCG).
- 5) Decreased stress response particularly toleration of semistarvation with equanimity thus conserving energy. (HCG has been reported in the literature as being useful in a wide spectrum of psy-

chological stress states from nocturnal enuresis to schizophrenia. (Postpartum depression may be precipitated by the HCG withdrawal at childbirth). It was an observation in prisoner-of-war camps that pregnant females appeared to tolerate semistarvation better than other inmates.

- 6) When caloric intake fell below a critical level (lower than for a nonpregnant individual) causing an energy deficit then the least essential tissues for survival, namely excess subcutaneous fat readily mobilized as an energy source for the foetus (4). (Patients having injections of HCG who don't keep to their diet and who lose little or no weight appear to undergo a significant decrease in their girth).

These hypothesized adaptations, mediated by HCG, for which there is clinical and experimental evidence not only explain the success of Simeons (5) and of those (6) who followed his empirical rules scrupulously in weight reduction, some observations of pregnancy, but also the failure of some double blind trials to confirm Simeons' results (3, 7). These researchers assumed a simple anorectic effect, ignored the possibility of generalized energy conservation by HCG, failed to observe the critical immediate post-treatment procedures, generally ignored Simeons' empirical rules, and neglected any long term follow-up.

Evidence for the suppression of the adipostat by HCG comes from clinical observations of commonly occurring substantial weight gains during pregnancy, the significant weight gain in children given injections of HCG (8) and the clinical impression of Simeons and others that weight loss occurs physiologically and psychologically with



ease under the influence of HCG when the caloric intake is below a critical level (5, 6, 9, 10).

This is probably the only physiological situation where the adipostat is suppressed temporarily and the new body mass level established under this umbrella has a remarkable stability as evidenced by the difficulty in removing weight gained during pregnancy and the clinical impression of relative permanency of weight losses using HCG with proper immediate aftercare compared to weight losses made against a functioning adipostat by simple calorie restriction with or without chemical anorexiant (5, 9).

The only known relatively long term double blind trial, which again did not adhere at all to Simeons' rules and concluded negatively to the value of HCG in degree of weight loss, which has never been claimed anyway, suggested confirmation of this relative long term stability using HCG (7). There is now some experimental evidence for a metabolic basis for some of these clinical observations (4, 11).

Stein et al. published a paper entitled "Ineffectiveness of human chorionic gonadotrophin in weight reduction: a double-blind study" in the *American Journal of Clinical Nutrition* 29: 940, 1976.

The authors were attempting to evaluate the relative merits of HCG in weight reduction but made no attempt to neutralize other factors that are known to contribute greatly to the success of weight reduction programs. They reported there was considerable peer pressure, a "group spirit," and patients all attended the surgery at the same time. Studies suggest that well motivated commercial weight watchers' groups have twice the weight loss obtained by hospital outpatients on ostensibly the same diet (12). I am sure a double blind trial of patients evaluating the effectiveness of a  $\beta$  blocker compared to a placebo (particularly while on other treatment) for angina which developed a group spirit in the subjects and required the participants to appear together at the clinic half dressed at the same time for an objective evaluation of their subjective symptoms would yield similarly inconclusive results. The importance of the leveling effect in a

conforming group situation is well documented.

It has never been claimed that HCG increased the weight loss of patients who strictly adhere to a certain caloric intake, (they would probably actually lose less weight because of energy conserving effects) but that they can adhere to it easily with a feeling of well being without needing the "milieu" of "reinforcement" by "peer pressure" and strong "group spirit" as described in this trial.

The trial also concluded that there was no statistically significant difference in the feeling of hunger. Again this is misleading as obese patients are notoriously poor at distinguishing food deprivation from other psychological feelings and needs (13). It would have been more accurate to report on the incidence of precisely described symptoms of food deprivation. Yet even in this trial which incorporated a strong group spirit there were twice as many "no hunger" responses recorded in patients having HCG than the placebo. It would be more helpful to compare the physiological hunger symptoms in patients from both groups who lost a lot of weight and those who lost little weight. "Hunger" is too loose a term, particularly for obese patients.

It was also concluded that there was no difference in hip and waist measurement changes in both groups. However the average weight loss was only about 15 pounds. It would be much more enlightening to observe these comparative measurement changes in patients from both groups who lost very little weight or more than 30 pounds. It has been my impression that patients who continuously break the diet and whose weights do not decrease by much that they lose inches off their waist-line while receiving injections of HCG.

The other most important facet which was ignored by this trial is whether there was any long-term difference in the maintenance of the new weight levels between the two groups. This is probably the most important issue of all.

However, as this trial ignored one of Simeons' (5) most crucial rules, namely the importance of overeating during the first 3 days of injections, there will probably be no



statistically significant difference in relapse rates. Asher and Harper (9) observed this rule and all other rules which may again explain the difference in results in other parameters. Related to this rule is the requirement that patients must not diet prior to beginning treatment and if they have they must overeat at least for 1 week before injections.

It has been assumed by many that these requirements were just "psychological" to prepare the patients for the rigid diet. Yet these rules are consistent with elementary cybernetics and adipostat theory. The adipostat being a negative feed back system is activated in this case by food restriction and reacts to resist decreasing body weight by physiological and psychological means and when adequate food is available it attempts to return the body to its previous mass level. Previous dieting would activate the adipostat and make suppression by HCG difficult or impossible. And again the adipostat was neutralized by 3 days of HCG before dieting began whereas if the dieting had begun immediately the adipostat would be activated before neutralisation by HCG had occurred. Looking in detail at the results of Craig et al. (7) this interpretation appears substantiated.

Although they don't specify whether patients were asked to overeat during the first 3 days, weight losses were so poor the diet was obviously not adhered to strictly so that the adipostat would not have been vigorously activated making some sort of neutralization by HCG possible.

However, as we have the weights 6 months before commencing the injections we can possibly deduce the patients who dieted before the course. If we regard a weight loss of a pound or more as evidence of dieting before the course we are left with seven patients in the HCG group, out of a total of 11 and seven patients in the placebo group out of a total of nine. In the 6 months after injections only one out of seven of the HCG group gained weight and the other six went on to lose an average of a further 12.5 pounds. However, in the placebo group four

out of the seven regained weight in the 6 months after the injections and three of those four were heavier than when they started the injections.

*Patrick Bradley*

Sydney  
Australia

## References

1. EDHOLM, O. G., J. G. FLETCHER, E. M. WIDDOWSON AND R. A. McCAMCE. The energy expenditure and food intake of individual men.
2. SIMMS, E. A. H., R. F. GOLDMAN, C. M. GLUCK, E. S. HORTON, P. C. KELLEHER AND D. W. ROWE. Experimental obesity in man. *Trans. Assoc. Am. Physicians* 81: 153, 1968.
3. FRANK, B. W. The use of chorionic gonadotrophin hormone in the treatment of obesity. *Am. J. Clin. Nutr.* 14: 133, 1964.
4. MELICHAR, V., M. RAZOVA, H. DYKOVA AND K. VIZEK. Effect of human chorionic gonadotrophin on blood free fatty acids, glucose, and on the release of free fatty acids from subcutaneous adipose in various groups of newborns and adults. *Biol. Neonate* 27: 80, 1975.
5. SIMEONS, A. T. W. Chorionic gonadotrophin in geriatrics. *Geriat. Soc.* 4: 36, 1956.
6. LEBON, P. Treatment of overweight patients with chorionic gonadotrophin: follow-up study. *Am. Geriat. Soc.* 14: 116, 1966.
7. CRAIG, L. S., R. E. RAY, S. H. WAXLER AND H. MADIGAN. Chorionic gonadotrophin in the treatment of obese women. *Am. J. Clin. Nutr.* 12: 230, 1963.
8. DE JONGE, G. A. Human chorionic gonadotrophin in the treatment of nocturnal enuresis. *Develop. Med. Child Neurol.* 15: 497, 1973.
9. GUSMAN, H. Chorionic gonadotrophin in obesity. *Am. J. Clin. Nutr.* 22: 686, 1969.
10. ASHER, W. L., AND H. W. HARPER. Effect of human chorionic gonadotrophin on weight loss, hunger, and feeling of well-being. *Am. J. Clin. Nutr.* 26: 211, 1973.
11. SURMACZYNSKA, B. Z., M. NITZAN, B. E. METZGER AND N. FREINKEL. The effect of oral glucose on plasma concentrations of human placental lactogen and chorionic gonadotrophin during late pregnancy in normal subjects and gestational diabetics. *Israel J. Med. Sci.* 10: 1481, 1974.
12. HOWARD, A. N. Dietary treatment of obesity. In: *Obesity, Medical and Scientific Aspects*, edited by I. M. Baird and A. N. Howard. London: Livingstone, 1969, p. 107.
13. BRUCH, H. Hunger awareness and individuation. In: *Eating Disorders, Obesity, Anorexia Nervosa and the Person Within*. London: Routledge and Kegan Paul, 1974 pp. 44-65.