South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

Cape Town, 9 February 1963 Volume 37 No. 6 Deel 37 Kaapstad, 9 Februarie 1963

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SHEEP PROLACTIN — A HUMAN GROWTH HORMONE?

The species specificity of growth hormone preparations has limited their use in man to preparations from primate sources. Very little human growth hormone (HGH) has been available, and so far that which is available is still being used to study the physiological actions of this hormone in man. There is no doubt that HGH has produced a significant and satisfactory growth response in hypopituitary dwarfed children, and experiments are still being conducted to ascertain its role, if any, in other varieties of dwarfism. Further therapeutic uses of HGH might include the promotion of anabolism or prevention of severe catabolic effects of illness or injury in many clinical circumstances. So far the paucity of HGH available has precluded an adequate trial of the hormone in these instances.

A very interesting report from McGarry and Beck[1] has recently appeared concerning the metabolic effect in man of a highly purified preparation of sheep (ovine) prolactin. The difficulty in separating prolactin from growth hormone obtained from human pituitaries, and the demonstration in animals of an overlapping biological activity of these two human hormones, promoted the idea behind this study. It had been previously reported that prolactin was able to stimulate growth in young normal rats and in hypophysectomized ducks and pigeons, and that it had an anti-insulin effect in hypophysectomized dogs and occasionally in hypophysectomized monkeys. A report of ovine prolactin producing nitrogen retention in 4 hypophysectomized women had already appeared, and the ovine prolactin preparation used by McGarry and Beck[1] had already been shown to produce an increase in body weight of adult female rats and an increase in the non-esterified fatty acids in the blood of fasting dogs.

The patients on whom prolactin was tried were 3 pituitary dwarfs who had shown a metabolic response to HGH in earlier studies. The ovine prolactin contained approximately 25 IU per mg. and was given intramuscularly in doses of 25 mg. twice daily. The 3 patients were kept on balance study in a metabolic ward.

Although the period of time over which the study ran was limited to a few days, there was a distinct increase in the urinary calcium output in all patients, nitrogen retention in 1 patient, and a fall of blood urea nitrogen in all 3. No consistent changes were found in the urinary phosphorus, sodium or potassium. Carbohydrate tolerance tests were performed in 2 cases, in one of which a decrease in tolerance was found during the administration of prolactin. In none of the patients was there any increase in the fasting free fatty acids of the plasma.

The metabolic response of human subjects to HGH is known to be characterized by a rise in fasting plasma free fatty acids, a fall in serum non-protein nitrogen and blood urea nitrogen, an increase in urinary calcium excretion, a decrease in urinary nitrogen excretion, and, in high doses in pituitary dwarfs, a decreased carbohydrate tolerance. The studies of McGarry and Beck[1] suggest that ovine prolactin in the dose used can mimic all these effects with the exception of the influence on the plasma free fatty acids. The considerable hypercalciuria produced by prolactin was very interesting; in view of the short time of the balance experiments it was impossible to tell whether it was accompanied by a significantly decreased faecal calcium and whether it had produced any change in the actual calcium balance. It would appear that the hypercalciuria induced by growth hormone does not produce a negative calcium balance because of a concomitant compensatory reduction in faecal calcium output.

These results seem rather surprising in spite of the reported similarities between ovine prolactin and human growth hormone with regard to their patterns on starch-gel electrophoresis. Although human prolactin and HGH are chemically and immunologically similar, ovine prolactin is both chemically and immunologically different from both ovine growth hormone and human growth hormone. It would be important and interesting to see whether this preparation of prolactin can take the place of human growth hormone in the treatment of patients requiring it.

1. McGarry, E. E. and Beck, J. C. (1962): Lancet, **2**, 915.

MAATREËLS TEEN DIE GEBRUIK VAN VERSLAWENDE MIDDELS

In die mediese pers, dwarsdeur die wêreld, word daar van tyd tot tyd gewaarsku teen die gevaar van die gebruik van verslawende middels. Dat hierdie herhaalde waarskuwing nodig is, ly geen twyfel nie. Daar is onteenseglik 'n wyd-verspreide neiging by jeugdiges en ouer mense, om al meer en meer gebruik te maak van gewoontevormende en verslawende middels.

Ons het self al by 'n vorige geleentheid gewys op die sterk toename, in Amerika byvoorbeeld, van die gebruik van sulke middels soos heroïen; en ons het aangetoon dat die ‚Federal Bureau of Narcotics of America' bereken dat die swartmarkhandel in verslawende middels in Amerika jaarliks ongeveer $275,000,000 aan inkomste oplewer.[1] Ons het ook verwys na die waarskuwing, van die Interdepartementele Komitee oor Verslawende Middels in Engeland, dat patente medisynes wat verslawende middels bevat, te maklik direk bekombaar was in Engeland.[2]

In hierdie artikel is ons hoofdoel om sommige maniere te beklemtoon waarop beveiliging teen die gebruik van dié soort middels versterk kan word. In die eerste plaas moet die opleiding van studente, wat hierdie saak betref, baie meer uitgesproke wees. Studente is veels te dikwels nie genoeg bewus van die diepgaande implikasies van verslawing nie. Nie alleen moet studente gedurig en sonder onderbreking teen dié gevare gewaarsku word nie, maar

hulle moet so leer dink dat hulle altyd ingestel kan wees op die moontlike verslawende uitwerking van die middels wat hulle later in hulle praktyke sal gebruik. Dit geld sowel middels wat bewese verslawende middels is, as ander middels wat potensieel verslawend kan wees.

Geneeshere self kan ook nooit te veel bedag wees op die gevare op hierdie gebied nie — wat hulle pasiënte betref en wat selftoediening betref. Te dikwels gebeur dit dat 'n besige geneesheer homself nie die tyd gun om 'n lastige en pynlike kwaal behoorlik te laat ondersoek en behandel nie, en hy neem dan maar 'n inspuiting om hom in staat te stel om voort te gaan met sy werk. As hy alreeds in die dae van sy opleiding so gekondisioneer was teen hierdie soort handeling dat dit as onmoontlik vir hom sou voorkom, sou dit makliker wees om nie in die versoeking te val nie.

Wat toediening van middels aan pasiënte betref wat met chroniese toestande gepla is, kan 'n dokter ook nooit versigtig genoeg wees om nie iets te doen wat 'n kwaaie verloop van sake tot gevolg gaan hê nie. Daar is genoeg soorte middels wat 'n pynstillende en kalmerende uitwerking het, om die dokter in staat te stel om nie die fisiologiese en psigologiese behoeftes aan verslawende middels by sy pasiënte te wek nie.

Die probleem van verslawing is natuurlik geensins enkelvoudig van aard nie, en 'n hele reeks omstandighede speel almal 'n rol by die voorkoms daarvan. Maar, omdat verslawing so 'n definitiewe fisiologiese komponent het deur die opwekking van 'n behoefte en die voorkoms van ernstige onthoudingsimptome by staking van gebruik van die middels, moet daar allerweë probeer word om die eerste stap — dit is die eerste gewoontevormende en verslawende toedienings — nie te laat plaasvind nie.

Een van die belangrikste benaderings tot dié probleem is dus voorkomende behandeling. En ons moet almal toesien dat daar 'n volgehoue opvoedingsprogram wat hierdie sake betref, daarop nagehou word, veral wat betref al die persone wat dié soort middels hanteer. Hierdie persone sluit mediese studente, geneeshere, verpleegsters, aptekers, maatskaplike werkers en ook winkeliers in. Ons moet die probleem bekamp deur nie toe te laat dat hy ontstaan nie.

1. Howe, H. S. (1955): N.Y. St. J. Med., **55**, 341.
2. Aantekening (1960): Brit. Med. J., **1**, 260.