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EDITORIAL : VAN DIE REDAKSIE

DIABETES IN SOUTH AFRICA

In any community diabetes exists in two distinct groups: known, clinically apparent diabetes under treatment, and unknown, often asymptomatic, hyperglycaemia of such a degree as to allow the clinical diagnosis of diabetes. The frequency of each of these varieties of diabetes among our different racial groups is clearer following 2 publications from the University of Cape Town Medical School.[1,2] These workers have attempted to assess the amount of diabetes present in whole communities representative of the country in general by the most sensitive methods known at the present time. Several previous studies have been made in Natal and the Transvaal,[3-10] but these have employed relatively insensitive methods or were made on unrepresentative groups of people,[2] and have not included White people. The Cape Town results indicate that the more or less urbanized Bantu of that region have just about the same amount of diabetes as the White people, both known diabetes (0·8% of the population over the age of 10 years) and diabetes discovered on survey (2%). This surprising finding does not mean that diabetes is equally common among the rural Bantu, since it is most likely that urbanization increases the incidence very considerably,[10] but it does mean that the Bantu has at least the same potential for developing hyperglycaemia given the necessary environment.

For several years the high prevalence of diabetes among Natal and Transvaal Indians has been discussed, and shown to be true among limited groups.[3-8] In Cape Town the known diabetes prevalence was found to be 4% over 10 years of age and the total diabetes rate was 8·4%, which comes to the massive figure of 16% on age correction. A similar but more extensive survey in Natal[2] (made in conjunction with the University of Natal Medical School) indicated the rather lower figure of 6% actual diabetes over the age of 10 years. The greater liability of the Indian to develop diabetes is thus confirmed. Another finding of interest was the occurrence of diabetes in 1% of young Indians under 20 years and its absence in other races in this age-group.

The reason for the prevalence of diabetes among our Indians is not clear, though on the whole they are better-off financially, eat more refined carbohydrates, weigh more and are more consanguineous than members of the same race who remain in India. They are certainly not heavy by White standards.

On the question of weight it needs emphasizing that among the White community examined in Cape Town, 23% of the women over 15 years of age and 16% of the men were judged to be obese.[1] Jewish people were more overweight than non-Jews.

Comparisons of the results in our White population with those of Europe and America are difficult because of difference in methods and interpretation. However, it appears that when similar methods are used diabetes is found considerably more frequently in these overseas populations than in Cape Town.[1] Nevertheless, it is estimated that there may be 100,000 White inhabitants of South Africa who have diabetes but do not know it!

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DIE VET KIND

Waarom word 'n maer kind beskou as sieklik en 'n vet kind as 'n toonbeeld van blakende gesondheid? So baie ouers is nog geneig om te dink dat as hul jong seuns of dogters maar soos poffertjies op mollig-ronde beentjies kan rondbaljaar het hulle hul dure plig as ouers grootliks vervul en kan geen mens na hulle 'n vinger wys nie en sê: 'Daardie kind is verwaarloos'. As die arme dogter miskien alte veel uitstoel dan word die saak tersyde geskuif met die handige verduideliking dat dit ongelukkig haar kliere is wat òf te veel werk, òf nie werk nie, al gelang na die mode wat op die betrokke oomblik in die kwasi-geneeskundige droomwêreld van die leek aanvaarbaar is. Helaas; meestal is dit inderdaad oorwerkte kliere wat die skuld moet dra, naamlik die speekselkliere.

Daar is vele opsigte waarin die ouers hul kinders kan faal en hulle nie die geestelik en fisies gesonde lewe verseker waarop die kinders met reg kan aanspraak maak nie. Maar om iemand van kindsbeen af te leer om oormatig te eet is seker een van die grootste sondes, want vir die res van sy of haar lewe sal die indiwidu moet ly onder sulke verkeerde eetgewoontes. Dit is uiters moeilik om die norme van mens se ouers te negeer, veral as sulke norme juis skynbaar die lewe soveel aangenamer maak. Iedere huisarts is bewus daarvan dat 'n vet kind 'n opdraende stryd moet voer om weer die oortollige gewig in latere jare te verloor, en gewoonlik help die ouers nie juis om die stryd makliker te maak nie. Daar is soveel handige skuiwergate wat gebruik kan word om die geveg teen vetsug op die lang baan te skuif, afgesien van die reedsgenoemde verskoning van verkeerd werkende kliere. Kindervet met die daarmee gepaardgaande kuiltjies is dan kwansuis so dierbaar en hoe dikwels hoor ons nie die selftevrede verklarings dat hierdie of daardie kind tog so 'n *gesonde* eetlus het. 'Ons het nog nooit 'n dag se pro-

bleme met sy etery gehad nie; hy geniet alles wat mens hom voorsit.'

Liewe moeders, dink tog daaraan dat die 'gesonde' eetlus miskien niks anders is as aangeleerde vraatsugtigheid nie. Maar nee, ons moet maar liefs nie sê dat 'n kind 'n vraat is nie; dit sal gevoelens darem té seer maak. Dit is beter om die arme seun of dogter maar aan die toekomstige diabetis of koronêre vaatsiekte oor te laat as om op sensitiewe tone te trap.

Eintlik is dit eienaardig dat vetsug by kinders so geredelik aanvaar word, want daar is seker min dinge wat meer onaantreklik is as 'n jong seun wat met nou-sluitende kortbroek op dik, sianotiese bene probeer om agter 'n bal aan te hardloop. Die lang, lenige kêrel wat met helder verstand en inherente handigheid sy plek in die skoolsport vol staan en wat met grenslose energie die een projek na die ander met geesdrif aanpak is skynbaar vir ons gemeenskap minder aanvaarbaar. Stop hom liewer 'n homp brood in die hand en sorg dat sy ratsheid omskep word in 'n lompe waggelgang—dan is ouma en al die bure tevrede. Ons het onlangs die pragtige beskrywing by 'n Kaapse Kleurling gehoor: 'Hy het sy groei dood ge-eet'.

Dit is die plig van iedere geneesheer om voortdurend te waarsku teen hierdie gevaarlike sosiale aanvaarding van vet kinders. Ons moet die taak op ons skouers neem om die eetgewoontes van kinders op 'n gesonder basis te plaas en ons is ongelukkig geneig om meer aandag te gee aan wan- en ondervoeding as aan vetsug. Mens wil nie die tragiese ondervoeding wat nog onder baie van ons land se kinders te vinde is as onbelangrik bestempel nie; dit is 'n saak wat ons intensiewe aandag vereis, maar ons moet ons nie op daardie sosiale euwel blind staar en die teenoorgestelde toestand ignoreer nie. Die vetsug is juis so gevaarlik omdat dit dikwels 'n familie aangeleentheid is wat nog verdere oorvoeding uitlok. Die oorgewig kind is gewoonlik te lief vir soet goed en vir koeldranke en omdat hierdie grootskaalse koolhidraat inname die eetlus demp, neig die ma om nog meer en meer op groter borde kos aan te dring.

'n Versigtige anamnese en dikwels selfs 'n bietjie speurwerk by die skool en onder die kind se vriende sal telkens aan die lig bring dat die snuisterye winkel in die kind 'n waardevolle klant gevind het. Om dan die verkeerde eetgewoontes te korrigeer met 'n ma wat eenkant staan en karring dat die dokter sy bes doen om die pragtige gesonde dogter soos 'n uitgeteerde spook te laat lyk, is geen maklike taak nie.

TRIMETHOPRIM

Sulphonamides alone are often active against certain common pathogenic organisms. However, their usefulness has lessened because many organisms have become resistant and because more effective drugs, antibiotics and others, have been introduced. The discovery of a potent antimicrobial pyrimidine compound known as trimethoprim,[1] which acts with sulphonamides in a synergistic and bactericidal manner, is regarded by many workers as 'a major discovery and to have far-reaching therapeutic possibilities'.[2]

Trimethoprim competitively inhibits the enzyme dihydrofolate reductase which is necessary to convert dihydrofolic to tetrahydrofolic acid, a stage in the synthesis of purines and ultimately of DNA. Trimethoprim has a much greater action on the enzyme in bacteria than in mammalian cells. It does have a small effect on human folate metabolism,[3] but there is little indication of interference with folate metabolism when the drug is given to animals or man.

Sulphonamides also interfere with folate metabolism, but at an earlier stage than does trimethoprim; they compete with para-aminobenzoic acid in the synthesis of dihydropteroate, the precursor of folic acid. Thus trimethoprim and sulphonamides act by sequential blockade of two enzymes intervening in the biosynthesis of folinic acid in the microorganism; alone they are bacteriostatic but in combination they are bactericidal.

On the basis of laboratory and clinical studies,[2] a combination of trimethoprim with sulphamethoxazole has been made available in a ratio of 1:5 (for example in the preparations Bactrim and Septin). Sulphamethoxazole was chosen as the most suitable sulphonamide because its duration of action in man is similar to that of trimethoprim. When given by mouth their rates of appearance in and disappearance from the blood are similar.

Organisms that are usually susceptible to the combination are streptococci, staphylococci (including penicillin-resistant strains), coliforms, salmonellae, shigellae, neisseriae, haemophilus, klebsiella, and proteus.

The results of clinical studies have been encouraging. Good results have been obtained in the treatment of gonococcal urethritis,[4] also in elderly patients with urinary infections, acute exacerbations of chronic bronchitis, and infections such as staphylococcal pneumonia and Gram-negative septicaemias. In one study patients with typhoid fever who were given 640 mg. of trimethoprim with 3·2 G of sulphamethoxazole daily for 14 days recovered, and their fever subsided more rapidly than in patients given 2 G of chloramphenicol daily for 14 days.[5] Further controlled studies are needed to confirm the results so far reported; trials should be undertaken in the treatment of infections resistant to antibiotics but sensitive to sulphonamides. The combination is expected to have a wider range of action and to be more effective than sulphonamides alone in the treatment of infections caused by sulphonamide-sensitive bacteria.

Unwanted effects have been reported but their importance remains to be evaluated. Nausea, vomiting and rashes occur rarely. The possibility of haematological effects should be considered in patients predisposed to folate deficiency in malabsorption syndromes, malnutrition, or during therapy with phenytoin sodium. Caution is required in dehydrated patients. The drug combination should not be given to patients sensitive to sulphonamides.

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