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OVERWEIGHT AND DIABETES

Obesity is a very important factor in the development of diabetes. Joslin,[1] quoting from insurance statistics, has said that people who are 25 per cent overweight are 8 times more likely to develop diabetes than those who maintain their correct weight. It is generally found that about 80 per cent of all new adult diabetics are more than five per cent over their standard weight when first seen. Many of them would have lost weight as a result of their glycosuria shortly before reporting to a doctor, so that their maximum weight will have been even higher than is recorded.

The frequency of obesity varies enormously in different countries. In many tropical countries it is a social asset, being an indication of success in life—largeness in personal size symbolizing largeness in economic and civic importance. Furthermore, in women it may be a sexual attribute—'my husband likes me fat'. Tulloch[2] has analysed the prevalence of diabetes in comparison to that of obesity in several tropical and sub-tropical countries, and concludes that where obesity is uncommon, diabetes is also uncommon, and vice versa. A pertinent illustration is that of the Zulu royal family in which diabetes is found in conjunction with extreme obesity, in contrast to the rarity of diabetes and of obesity among rural Zulu commoners.[3] Where age- and sex-grouped diabetics and non-diabetics from the same populations have been studied, adult diabetics have been found to be far more commonly obese than non-diabetics.

In almost every series obesity is considerably commoner in women than in men. Of 869 diabetics attending a clinic in Cape Town, 43 per cent of women were clearly overweight, but only 16 per cent of men. In most reports overweight appears to increase with increasing parity. Nevertheless, the high incidence of obesity in diabetic women is not entirely dependent upon parity, since even nulliparous diabetics are more overweight than male diabetics.

Simple reduction of weight by dieting is sufficient to afford good control of the diabetes in a large number of obese patients, and in many of these even the glucose tolerance will then revert to normal. However, the great majority of obese subjects never develop diabetes and retain normal glucose tolerance even when their carbohydrate-regulating mechanism is subject to the added stress of cortisone. Beaudoin and his colleagues[4] believe that discrepant reports from different authors with regard to carbohydrate tolerance in obesity may be explained in part by the type or phase of obesity in the individual patients concerned. They examined two groups of obese women, one group representing 'active' obesity (i.e. weight gain was actually progressing or the obesity was of recent onset), and the other group representing 'static' obesity (i.e. obesity of several years' duration, usually since childhood, and not having increased for a long time). 'Actively' obese subjects had a far greater tolerance for carbohydrate. In fact, during periods of rapid weight gain a tendency to *hypoglycaemia* has sometimes been found.

The actual mechanisms of the relationship between diabetes and obesity are uncertain. Although obesity frequently appears to 'bring on' diabetes, there is a widespread belief that some factor in the basic metabolic derangement of diabetes predisposes to obesity, at least in the adult-onset cases.

Murray and Wang,[5] in Glasgow, found that 33·7 per cent of their 'old' clinic patients and 41·3 per cent of new patients were more than 20 per cent overweight. They observed that a very high proportion (67·7 per cent) of obese diabetics had non-diabetic obese relatives, but of course this does not help us to say which comes first, the diabetic trait or the obesity trait, or which causes which.

The several modern theories connecting obesity and diabetes all postulate an increased production of insulin at some stage:

1. Inheritance. It has been suggested that the tendency to obesity and to diabetes are inherited together as connected genetic factors. We are not aware of any direct evidence in favour of this hypothesis, although it is extremely attractive. The inherited mechanism linking obesity and prediabetes might be a tendency to supernormal insulin production.
2. Extra bulk in latent diabetics. The frequency of a history of diabetes in other members of the family of the obese diabetic, and the comparatively small proportion of all obese subjects who actually become overtly diabetic, suggest that obesity acts as an additional factor in an already insufficient carbohydrate-regulating mechanism. In other words, the patient may have been previously prediabetic, and the additional body mass was then sufficient to overcome the ability of the pancreatic islet tissue to keep pace with the metabolic demands made upon it. This idea is supported by the work of Mayer and his colleagues,[6] who showed that the pancreatic islets in hereditary, obese, diabetic mice were hypertrophied, as though trying to keep up with their bodies' needs.
3. Overwork exhaustion. The overeating associated with obesity may produce an overstimulation of pancreatic beta cells followed by damage from overwork. Experimental evidence in favour of this theory includes the production of diabetes in certain animals by grossly overloading them with carbohydrate for a long period. However, it does not accord with the clinical improvement in carbohydrate tolerance so frequently seen after relatively small weight loss.
4. Fat-active insulin. The abnormal metabolic sequences that occur in diabetes do not favour the accum-

ulation of fat in the body, but rather tend to deplete the fat stores. It may, however, be recalled that in prediabetes and in recent-onset diabetes of both juvenile and maturity types, fasting serum has been found to contain raised levels of insulin-like activity, effective on rat adipose tissue but not effective on rat diaphragm. Whatever the exact cause of this may be, the ability of such insulin to act preferentially on adipose tissue could result in excessive accumulation and retention of fat, at the same time as a 'diabetic' effect was occurring in other tissues such as muscle. The relative resistance of obese diabetics to ketosis fits in with this hypothesis.

5. Glucose-utilization defect in obesity. Forsham and his colleagues[7] have found that the serum insulin of obese, non-diabetic subjects rises to higher levels after a glucose load than that of non-obese controls, at the same levels of blood sugar. They suggest therefore that obese people may have a primary defect in peripheral utilization of glucose resulting in a compensatory overstimulation of pancreatic beta cells after administration of glucose. Normal glucose tolerance persists as long as the extra insulin is sufficient to compensate for this defect; if the insulin production cannot keep pace with the metabolic demands, diabetes results.

We may not fully understand the connection between obesity and diabetes, but we do know the moral: Don't get fat anyway, but if there is diabetes in your family, doubly don't!

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2. Tulloch, J. A. (1962): *Diabetes Mellitus in the Tropics*. Edinburgh: Livingstone.
3. Campbell, G. D. (1960): S. Afr. Med. J., **34**, 332.
4. Beaudoin, R., Van Itallie, J. B., Mayer, J. (1953): J. Clin. Nutr., **1**, 91.
5. Murray, I. and Wang, I. (1956): Diabetes, **5**, 49.
6. Mayer, J., Bates, M. W. and Dickie, M. M. (1951): Science, **113**, 746.
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NUWE TYDSKRIFTE VAN DIE MEDIESE VERENIGING

Gedurende die loop van 1963 het ons 'n belangrike nuwe veld op die gebied van die mediese joernalistiek in ons land betree deur die publikasie van die *Suid-Afrikaanse Tydskrif vir Obstetrie en Ginekologie* en die *Suid-Afrikaanse Tydskrif vir Radiologie* as byvoegsels tot die *Suid-Afrikaanse Tydskrif vir Geneeskunde*. Hierdie publikasie is met groot entoesiasme ontvang, nie net in ons eie land nie maar ook in die buiteland. Die agtergrond van hierdie ontwikkeling is kortliks soos volg:

Alhoewel daar algemene ooreenstemming is oor die feit dat die algemene praktisyn, om baie redes waarmee ons bekend is, nog 'n onbepaalde tyd lank die basis van ons mediese praktyk sal en moet bly, ontwikkel die medisyne al meer in die rigting van gespesialiseerde groepsbelange. Dit is 'n noodwendige ontwikkeling in die tyd waarin ons leef.

Die vraag het ontstaan of dit moontlik en/of verstandig sal wees om gespesialiseerde tydskrifte in Suid-Afrika te publiseer. Omdat die meeste spesialiste in elk geval op hul eie gespesialiseerde oorsese vakblaaie inteken, en omdat die aantal lede in ons land in elke groep so (relatief) klein is, lyk dit onverstandig om vir elke groep 'n aparte gespesialiseerde blad te publiseer.

'n Moontlike oplossing van hierdie probleem is gevind deur die weeklikse *Tydskrif* van die Vereniging, wat onder alle lede van die Mediese Vereniging sirkuleer, as basis te gebruik vir die publikasie van gespesialiseerde byvoegsels. Op aandrang van die verskillende groepe wat betrokke is, het ons nou 'n stap verder gegaan en besluit om hierdie byvoegsels van 1964 af as volwaardige tydskrifte onder hulle eie name te publiseer. In die betrokke weke waarin die byvoegsels dus sou verskyn het, sal die nuwe tydskrifte nou onder hulle eie name verskyn. Hulle sal nogtans op die basis van die sirkulasie van die weeklikse tydskrif versprei word na alle lede van die Mediese Vereniging en nie net na die lede van die betrokke groepe nie. Elke lid sal dus nog twee-en-vyftig weeklikse publikasies kry, soos in die verlede.

Op hierdie manier sal dit dus wel vir ons moontlik wees om in Suid-Afrika gespesialiseerde tydskrifte te publiseer. Lede van die Vereniging sal hierdeur die voordeel hê dat hulle, net deur die Vereniging en sy tydskrifte te ondersteun, spesiale uitgawes in hulle eie vakgebiede kry en ook dat spesiale uitgawes op ander vakgebiede vir almal beskikbaar gestel sal word. Op hierdie manier hoop ons om 'n waardige bydrae te maak tot die doelstelling om eenheid binne verskeidenheid in die mediese vakgebiede te bewerkstellig.

Reëlings is al reeds getref om die *Suid-Afrikaanse Tydskrif vir Obstetrie en Ginekologie* en die *Suid-Afrikaanse Tydskrif vir Radiologie* as aparte tydskrifte gedurende die loop van die jaar te laat verskyn. Ook sal die *Suid-Afrikaanse Tydskrif vir Laboratorium- en Kliniekwerk*, wat voorheen as 'n kwartaalblad op sy eie gepubliseer is en slegs 'n klein leserskring gehad het, ook nou op die nuwe basis gedruk en versprei word na alle lede van die Vereniging.

Bowe en behalwe hierdie genoemde spesiale tydskrifte hoop ons om nog gedurende die jaar 'n aantal spesiale uitgawes van die *Suid-Afrikaanse Tydskrif vir Geneeskunde* te publiseer, onder andere die Verhandelinge van die Voedingsvereniging van Suidelike Afrika, die Verhandelinge van die Oftalmologiese Vereniging, 'n spesiale uitgawe oor genetiese onderwerpe, die Stellenbosse uitgawe, en moontlik ook 'n spesiale tydskrif oor die plastiese chirurgie.

Die samewerking en ondersteuning van alle lede van die Vereniging en van ons adverteerders om van hierdie onderneming 'n sukses te maak, sal ten seerste waardeer word.