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CORONARY ARTERY DISEASE AND DIABETES

Among diabetics coronary artery disease is almost as common in women as in men. One usual advantage in being born female is the relative immunity from myocardial ischaemia that it affords, especially before the menopause. This advantage is virtually abolished by diabetes. Why this should be is uncertain—female diabetics have as much and as good oestrogen as non-diabetics, for instance. It is an odds-on chance that a woman in her thirties who develops symptomatic coronary artery disease, who is not a chronic hypertensive, nor hypothyroid, does not suffer from familial hypercholesterolaemia or from loss of her ovaries, will have diabetes which is evident either clinically or by the glucose-tolerance test.

A Coloured girl aged 32 was a recent example. She was 10-para, with five live babies, all under eight pounds at birth, two stillbirths, and three miscarriages. Her mother 'died of diabetes'.

Three days before admission she complained of dyspnoea, palpitation, pain in lower chest, nausea and vomiting.

On admission to hospital she was very drowsy, dehydrated, with acidotic breathing. BP 115/80 mm.Hg, pulse rate 150; no infection found. Urine contained 4+ sugar and ketones. ECG: classical antero-lateral infarction, with deep Q waves and very high ST segment take-off over chest leads. Blood sugar 1,050 mg. per 100 ml.; bicarbonate, 6·7 mEq. per 1.; potassium, 4·7 mg. per 100 ml.

She recovered from diabetic ketosis after 300 units of insulin and appropriate fluids. She also recovered from the effects of the myocardial infarction. Serum cholesterol levels were 228 and 232 mg. per 100 ml. She was discharged taking 40 units of lente insulin daily, on a 1,550 calorie diet, weighing 110 lb.

As an outpatient she remained on diet but stopped insulin after hypoglycaemic episodes. She kept well and the urine remained sugar-free. During her eleventh pregnancy glycosuria returned in the third month; fasting blood-sugar was 152 mg. per 100 ml.; weight 119 lb. Her urine became sugar-free on tolbutamide, and a fasting blood-sugar level was 87 mg. per 100 ml. Tolbutamide later became unnecessary and, in the eighth month, her blood-sugar levels were well within the normal range, with diet only. Labour was induced at 37 weeks; the baby's arrival was uneventful and six months later she still required no insulin.

Evidently myocardial infarction, occurring in a young woman with pre- or latent diabetes, had precipitated diabetic coma. This was followed by a substantial remission, rendering continuation of insulin therapy unnecessary, even during subsequent pregnancy.

In a series of 210 diabetics from the Groote Schuur Hospital diabetes clinic, selected at random, all but 32 were over 40 years of age.[1] Clear-cut clinical or electrocardiographic evidence of coronary disease was found in 21·4 per cent of the total; in 26 per cent of the men and 20 per cent of the women. The occurrence rate among diabetic men aged 40 - 59 was more than double that found in a group of non-diabetic males of a similar age. There was no correlation between poor control of diabetes and prevalence of coronary disease.

In an analysis of almost 50,000 autopsy records from 1910 to 1948 Bell[2] found that four per cent of all deaths from coronary disease were associated with diabetes in men and 14 per cent in women. Wahlberg[3] found that the remarkably high proportion of 46 per cent of patients with 'atherosclerotic disease' had diabetic intravenous glucose-tolerance tests. The test was abnormal in 10 per cent of a control group—itself a notably high incidence for a white Scandinavian population. Sowton[4] noted abnormal glucose tolerance in 73 per cent of 30 patients tested soon after an episode of myocardial infarction. Nye[5] examined 109 hypertensive subjects and found that 35 per cent of those with evidence of coronary artery disease had abnormal oral glucose tolerance, compared to 16 per cent abnormal among patients without coronary disease.

The connection between diabetes and myocardial infarction has recently been cemented in a remarkable manner by Vallance-Owen and Ashton.[6] They found that 19 out of 28 unselected infarct patients had an increased antagonism to insulin associated with their plasma-albumin. This is the same abnormality as is found in genetic diabetes. The authors suggest that 'many patients with cardiac infarction are constituted as essential diabetics, although they only relatively rarely show carbohydrate intolerance'.

This syn-albumin antagonist of Vallance-Owen is believed by him to be the basic abnormality in diabetes, inherited as a dominant characteristic and present already in the prediabetic phase. In this phase, as in many infarct patients, an increased insulin production prevents overt diabetes from developing. If, however, we are to consider myocardial infarction as a purely diabetic phenomenon, then the mechanism by which the syn-albumin antagonist leads to coronary atheroma and thrombosis is quite unclear—indeed it is equally unclear how it may lead to the small blood vessel disease which characterizes diabetes.

Against the apparent extremely intimate connection between diabetes and coronary artery disease must be set the lack of coronary artery disease found in Bantu diabetics, although the more certainly diabetic vascular lesion of retinopathy is not uncommon. Thus Seftel[7] could not find evidence of coronary artery disease in a single Bantu diabetic over the age of 40 out of a group of 112 attending a Johannesburg clinic; in the same group 44 per cent of those whose diabetes had lasted beyond 7 years had retinopathy. Whatever may eventually be found, it is plain that the inherited diabetic abnormality alone is not enough to produce coronary artery disease—another factor is also necessary.

1. Markman, P., Allen, E. A. and Jackson, W. P. U. (1959): S. Afr. Med. J., **33**, 682.
2. Bell, E. P. (1952): Arch. Path., **53**, 1952.
3. Wahlberg, F. (1962): Acta med. scand., **171**, 1.
4. Sowton, E. (1962): Brit. Med. J., **1**, 84.
5. Nye, E. R. (1964): *Ibid.*, **2**, 727.
6. Vallance-Owen, J. and Ashton, W. L. (1963): Lancet, **1**, 1226.
7. Seftel, H. C. (1964): Paper presented at the 21st Annual Conference of the Students Medical Council of the University of the Witwatersrand, April.

LEONARDO DA VINCI

Leonardo da Vinci word eintlik as 'n skilder onthou—die skepper van sulke meesterstukke soos ‚Mona Lisa' en ‚Die Laaste Avondmaal'. Dog, min mense het nog die veelsydigheid van hierdie genie geëwenaar. Hy het ook as uitvinder, beeldhouer, plantkundige, geoloog, aardrykskundige, musikant, kaarttekenaar, argitek, ingineur, matematikus, anatomis en fisioloog vaardige en oorspronklike werk gelewer. Terwyl sy kunswerke al vir meer as 500 jaar bewonder word, is sy geskrifte eers die afgelope 50 jaar behoorlik ontleed.

Sy lewensuitkyk word saamgevat in sy eie woorde: ‚Goeie mense is weetgierig'. Met grenslose energie het hy hom van die een taak tot die ander gewend en verklaar: ‚Geen werk kan my uitput nie'. Die kritiek is dikwels teen hom gerig dat sy kop so vol ondernemingsgees was dat hy selde 'n taak behoorlik afgehandel het—hier word bv. verwys na die verbrokkeling wat in ‚Die Laaste Avondmaal' begin intree het, kort na die voltooiing daarvan met 'n rewolusionêre nuwe skildertegniek op pleister.

Nogtans kan van sy anatomiese studies en geskrifte alleen gesê word dat dit 'n lewenstaak vir 'n gewone mens sou gewees het. Die manuskrip en tekeninge beslaan 2,000 paginas. Dit is deur MacCurdy[1] in 1,000 bladsye saamgevat en sal dus hopelik behoue bly. Tragies genoeg was die vrug van sy arbeid op hierdie gebied verlore vir sy onmiddellike opvolgers toe dit sedert die dae van Karel I tot aan die begin van hierdie eeu in Windsor Castle opgesluit gebly het.

Aanvanklik was dit net Leonardo se bedoeling om die anatomie te bestudeer sodat dit hom met sy kuns kon help. Mettertyd het dit egter 'n studie op sigself geword—'n drang om sy weetgierigheid te bevredig. Dit was sy voorneme om 'n boek oor die anatomie te publiseer en volgens sy eie verklaring het hy met hierdie doel meer as dertig lyke gedissekteer. Hy het egter nooit die kans gekry om sy tekeninge te rangskik en sy geskrifte te redigeer nie.

Veral in sy vroeëre studies kom heelwat ongerymdhede voor as hulle getoets word aan die lig van hedendaagse kennis. Die foute was egter nie soseer die gevolg van swak waarneming nie as dat hulle gekondisioneer en aangevul is deur sommige wanverkondigings uit die werke van Galen en Avicenna. So het hy bv. die uterus as 'n dubbele buis geteken, die onderste as die vervoerder van urine en saadselle, terwyl die boonste die gees van die dier na die embrio gedra het—soos deur Avicenna verkondig. Gewoonlik het hy sy eie foute na behoorlike waarneming later gekorrigeer. Oor die algemeen kan gesê word dat hy die menslike anatomie goed begryp het en dat sommige van sy tekeninge die werk van moderne kunstenaars oortref.

Sy tekeninge van die bene van die arm, by die gewrigte saamgevoeg en in verskillende fisiologiese posisies, is onoortrefbaar. In die skedel het hy die maksillêre sinus ingeteken, hoewel dit eers 150 jaar later deur Highmore herontdek is. Volgens Leonard Murphy[2] gee sy weergawe van die nier 'n getroue weergawe van die nier en bloedvate soos van die buitekant gesien. Daar is egter geen bewys dat hy sy nota: ‚kloof dit deur die middel en toon aan hoe die urinegange gesluit word en hoe dit urine distilleer', opgevolg het nie. Hy was ook voornemens om die verhouding van die nier tot die ‚lende en vals ribbes' aan te dui.

Maar miskien is Da Vinci se meesterlikste uitbeelding dié van die swanger uterus. William Hunter het in 1774 daarvan gesê, toe hy op die punt gestaan het om sy *Atlas of the Gravid Uterus* te publiseer, dat Da Vinci sy tyd 300 jaar vooruit was. Selfs in sy sorgvuldige beskrywing van die plasenta en hoe dit losgaan, was hy sy tydgenote voor.

Nadat hy die gewenste hoedanighede van die kunstenaar-anatomis beskryf het, sê Leonardo: ‚Of hierdie eienskappe in my aanwesig was of nie, daarvan sal die 120 boeke wat ek saamgestel het, moet rekenskap gee. In my werk is ek nie deur gierigheid of agtelosigheid gepla nie, maar net deur my eie tekortkominge . . .'

1. MacCurdy, E. (1954): *The Notebooks of Leonardo da Vinci.* Londen: Reprint Society.
2. Murphy, L. (1964): Med. J. Aust., **2**, 556.