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MIND AND THE MENSTRUAL CYCLE

Every student of gynaecology soon knows that strong emotion may cause the abrupt interruption of the menstrual cycle physiologically (as in women students and nurses away from home) or pathologically, as in anorexia nervosa. The nature of the underlying mechanisms is as elusive as the paradox that in some women a powerful emotion has the opposite effect and induces menorrhagia or dysfunctional bleeding. Psychological aspects of obstetrics and gynaecology had been of particular interest to the late Professor James Louw, hence the choice by his successor of 'Mind, matter and menses' as the title of the memorial symposium organized by the Association of Medical Students (AMSSA) in Cape Town on 5 July. Professor Davey himself participated as chairman, together with a member of his staff, a psychiatrist and a general practitioner.

It is in fact surprising how little advance there has been since the work of Tilt in 1851; who, emulated by Stieve a century later,[1] published anatomical material derived from executed women prisoners who had begun to menstruate just before their executions. The bleeding appeared to emanate from atrophic endometrium, and ripe ovarian follicles were not to be found, so that Stieve macabrely designated the phenomenon as *Schreckblutungen* (fright bleeding). Heiman[2] reviewed reports of 82 women with functional uterine bleeding and found that in three-quarters a radical worsening in a close personal relationship preceded the onset. This is however a liberal interpretation of no less than 28 different emotional precipitants cited in the small series. Heiman's conclusion that functional uterine bleeding is the 'separation bleeding of the mourning womb' seems not especially helpful.

More interest appears to have centred on the menstrual syndrome. Katharina Dalton's[3-6] several epidemiological contributions have indicated the serious significance that menstrual morbidity carries, with its increased risk of traffic accidents,[3] crime, attempted suicide and admissions for acute breakdown in those so predisposed.[4] Industrial absenteeism rates go up[5] and even public-school girls are more likely to be punished for minor misdemeanors.[6] Since women spend one-quarter of their reproductive lives in the premenstruum and in menstruating this syndrome merits close attention.

The premenstrual syndrome is an ill-defined syndrome with subjective complaints of irritability, depression, anxiety, lethargy, tender breasts, headache, backache, and a stuffy, bloated feeling. Hippocrates had already observed it and ascribed it to 'agitated blood attempting to escape at the uterus'. In the nineteen-thirties Sweeney[7] and others noticed that some women tended to gain weight at this time of the cycle, usually about 3 lb., but rarely, as much as 11 lb. Bickers later was so impressed by this finding and occasional frank oedema that he suggested water toxicity as the cause.[8] Various mechanisms were then adduced to explain the water retention, among them alteration in the oestradiol/pregnandiol fraction, oversecretion of ADH or aldosterone, autonomic imbalance, vitamin B deficiency, and menstrual toxins.

In 1962 Russell and Bruce, psychiatrists at the Maudsley Hospital, published careful metabolic studies of the problem.[9] Their patients were mostly severe neurotics who complained of premenstrual exacerbation of their symptoms. One group of 24 were allowed relative freedom of diet and activity and were weighed daily for 3 months. Surprisingly, 9 of them actually lost weight premenstrually and the group's over-all negligible premenstrual gain of 0·04 lb. was grossly overshadowed by the fluctuations of 3 lb. that occurred during their weekends at home. The second group of 10 patients were more closely studied on diets of known content, with analysis of 24-hour urines. But here again the small premenstrual weight gain with sodium retention was surpassed by weight gain with fluid retention at midcycle. In only 3 of the 10 did the subjective symptoms worsen premenstrually, nor did any complain at midcycle.

These findings complement the work of Thorn in the USA on normal women. It is clear that while many women may gain weight premenstrually, no-one has shown a correlation between weight gain and the subjective complaint of tension. Sometimes it has in fact been noted that those who gain weight most (and may even show pitting oedema) feel no irritability or depression; while some who lose weight may complain of much distress. Furthermore, attempts to treat the syndrome with diuretics such as chlorothiazide are only successful in less than half of cases.

A random sample of 500 women were questioned by Kessel and Coppen[10] and scored on the Maudsley Personality Inventory for 'neuroticism' (emotional vulnerability). Dysmenorrhoea was common (45 per cent) but not related to neuroticism (it in fact declined with age and with parity). About 1 in 3 women complained of premenstrual irritability (1 in 9 experienced this severely) and this did correlate with neuroticism, as well as with the marital state.

One of the suggestions arising from their study was that the bloated feeling might be due, not to absolute water retention, but to a redistribution of water and electrolytes between intra- and extracellular compartments of the body, as occurs in severe depressions.[11] Their next step was therefore to examine an abnormal population of neurotics, severe depressives, and schizophrenics using their earlier findings as control data.

Again the psychological complaints of premenstrual tension were more common in the neurotic subjects. The schizophrenics complained significantly less and, surprisingly, the depressives complained no more commonly than did the control women.[12] This makes it unlikely that the electrolytic changes of depressive illness are identical to the putative biochemical changes of premenstrual tension

states, but radio-isotope ion tracking, such as Coppen and Shaw[13] undertook in depressions, has yet to be done in the premenstrual syndrome.

Meanwhile, the nature of menstrual irritability remains enigmatic except in so far as it may be related to a lowered threshold of complaint in women who score highly on the neuroticism scale of the MPI. This factor may resemble the 'pain-proneness' reported to underlie patients who complain of psychogenic pain,[14] or may be connected with acquired personal and cultural attitudes to menstruation.

1. Tilt, E. J. (1851) and Stieve, H. (1943): *Op. cit.*[2]
2. Heiman, M. (1959): J. Mt Sinai Hosp., **26**, 56.
3. Dalton, K. (1960): Brit. Med. J., **1**, 1425.
4. *Idem* (1959): *Ibid.*, **1**, 148.
5. *Idem* (1964): Proc. Roy. Soc. Med., **57**, 262.
6. *Idem* (1960): Brit. Med. J., **2**, 1647.
7. Sweeney, J. S. (1934): J. Amer. Med. Assoc., **103**, 234.
8. Bickers, W. (1958): Virginia Med. Mth., **85**, 613.
9. Bruce, J. and Russell, G. F. M. (1962): Lancet, **2**, 267.
10. Kessel, N. and Coppen, A. (1963): *Ibid.*, **1**, 61.
11. Coppen, A. (1965): Brit. J. Psychiat., **111**, 1133.
12. *Idem* (1965): *Ibid.*, **111**, 155.
13. Coppen, A. and Shaw, D. (1963): Brit. Med. J., **2**, 1439.
14. Engel, G. L. (1959): Amer. J. Med., **26**, 899.

DIE VERANDERENDE GESLAGSVOORKOMS VAN DIABETES

Die gangbare opvatting by Europese en Amerikaanse geneeshere is dat diabetes aansienlik meer voorkom by vrouens as by mans.[1,2] Die voorsprong op hierdie gebied by die skone geslag is toe te skryf aan die groter voorkoms van diabetes wat in volwassenheid begin, soos gesien na die ouderdom van 30 - 40. Pyke[2] en Fitzgerald en sy medewerkers[3] kon skynbaar bo enige twyfel met syfers en argumente aantoon dat die hoë voorkoms van middeljarige diabetiese vrouens verklaar kan word op grond van pariteit—elke swangerskap lei tot meer diabetiese geneigdheid totdat die volledige beeld van die toestand teenwoordig is. Hierdie aantreklike teorie het egter die feit veronagsaam dat daar in die meeste van die Kaukasiese lande van die wêreld nie 'n oorwig van vrouens onder diabetiese lyers is nie.[4] Trouens, die bewyse (wat ongelukkig nie afdoende is nie) dui op 'n oorwig van mans in baie tropiese lande.

Daar is meer treffende bewyse wat daarop dui dat hierdie oorwig van Kaukasiese vroulike diabetiese lyers gedurende die vroeë twintigste eeu 'n verbygaande rariteit is, selfs onder Blankes. Gedurende die afgelope eeu het diabetes waarskynlik meer onder mans as onder vrouens voorgekom in Brittanje en op die Vasteland van Europa.[5-7] Pavy[7] het byvoorbeeld, volgens syfers uit sy private praktyk in Londen, 200 mans tussen die ouderdomme van 40 en 49 behandel, teenoor slegs 79 vrouens. Gestandardiseerde sterftesyfers vir Engeland en Wallis, volgens die rekords van die Registrateur-generaal, toon ook 'n sterftesyfer aan diabetes wat tweekeer so hoog is vir mans as vir vrouens (gedurende omstreeks 1860).

Sedert die begin van hierdie eeu was daar 'n vaste styging in die sterftesyfer aan diabetes by vrouens, soos aangetoon deur die statistieke van verskeie Europese lande.[8] Malins en sy medewerkers[9] het gegewens van hul diabetiese klinieke gepubliseer wat die aantal nuwe gevalle aantoon in 5-jaar groepe van 1930 tot 1964. Aan die begin van hierdie tydperk was die oorwig van vrouens alreeds duidelik en daar was geen noemenswaardige verandering van die mans/vrouens verhouding tot 1955 - 59 nie, toe die vermeerdering van die vroulike diabetiese lyers begin afneem terwyl dié van die mans 'n bestendige toename getoon het. Gedurende 1945 - 49 was die mans/vrouens verhouding bv. 0·48 terwyl dit in 1960 - 64 0·90 was. Die standardisering van die syfers ten opsigte van die toepaslike sensussyfers, het feitlik geen verskil aan die verhoudings gemaak nie. Daar kan natuurlik sekere moontlike foute voorkom, soos Malins *et al.*[9] aantoon. Waar daar in 'n stad meer as een kliniek bestaan, kan praktisyns byvoorbeeld verkies om jong diabetiese pasiënte na een van die klinieke en ouere pasiënte na 'n ander kliniek te verwys. Ligte gevalle en ouere diabetiese pasiënte mag dalk glad nie na 'n hospitaal verwys word nie. En mans mag traer wees om na die hospitaal toe te gaan omrede van die verlies van tyd en geld omdat hulle uit die werk uit bly.

Sover bekend was geen een van dié oorwegings enigsins van toepassing op die diabetiese kliniek van die hospitaal van waar Malins se gegewens verkry is nie. Selfs al sou dit die geval wees, sou dit moeilik wees om te verstaan waarom daar 'n verandering sou wees gedurende die afgelope 20 jaar wat een geslag meer as die ander raak.

Verdere ontleding van Malins se syfers toon dat die verandering die gevolg is van 'n sneller toename in die aantal mans wat diabetes ontwikkel het sedert 1950, veral tussen die ouderdomme van 35 en 64. Hierdie toename in die aantal manlike diabetiese pasiënte kan waarskynlik nie toegeskryf word aan vermeerderde roetine-ondersoek van die gemeenskap nie, alhoewel daar verwag kan word om 'n groter aantal manlike as vroulike diabetiese pasiënte tussen die ouderdomme van 50 en 69 te ontdek.[10]

Die verklaring vir hierdie verandering bly nog duister. Malins meen dat daar moontlik minder vrouens mag wees wat gedurende die jongste tye met diabetes gepresenteer het, maar daar bestaan nie genoegsame bewyse dat dit die geval is nie. As dit waar sou wees, sou dit ook nie verklaar kon word op grond van 'n afname in oorgewig of veranderings wat betref pariteit onder vrouens nie. Malins eindig deur die waarskuwing uit te spreek dat daar baie versigtig te werk gegaan moet word wanneer formele genetiese ontledings gemaak word van gegewens wat versamel is gedurende tye waarin betreklike vinnige veranderings voorgekom het.

1. Joslin, E. P., Root, H. F., White, P. en Marble, A. (1959): *Treatment of Diabetes Mellitus*, 10th ed., p. 33. Londen: Henry Kimpton.
2. Pyke, D. A. (1956): Lancet, **1**, 818.
3. Fitzgerald, M. G., Malins, J. M., O'Sullivan, D. J. en Wall, M. (1961): Quart. J. Med., **30**, 57.
4. Tulloch, J. A. (1962): *Diabetes Mellitus in the Tropics*. Edinburgh: E. & S. Livingstone.
5. Bouchardat, A. (1875): *De la Glycosurie ou Diabète Sucré*. Paris.
6. Saundby, R. (1891): *Lectures on Diabetes*, p. 30. Bristol: John Wright.
7. Pavy, F. W. (1885): Lancet, **2**, 1033.
8. Harris, H. en MacArthur, N. (1951): Ann. Eugen., **16**, 109.
9. Malins, J. M., Fitzgerald, M. G. en Wall, M. (1965): Diabetologia, **1**, 121.
10. Report of a Working Party appointed by the College of General Practitioners (1963): Brit. Med. J., **2**, 655.