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NEUROTIESE SIMPTOME IN ORGANIESE SIEKTE

Neurose is 'n baie algemene siekte, en sluit ten minste 7 herkenbare kliniese sindrome in: Moegheid, of neurastenie soos dit vroeër genoem is; eenvoudige senuagtigheid, angsneurose en neurosirkulatoriese astenie; fobiese neurose; obsessiewe dwangneurose (vroeër psigastenie genoem); histerie; hipochondriase en reaktiewe depressie. Meestal het die pasiënte egter 'n mengbeeld van bogenoemde sindrome. Die moontlikheid dat daar 'n genetiese agtergrond vir hierdie simptome mag wees word vandag ernstig oorweeg en die feit dat verskeie psigofarmakologiese middels in die behandeling van baie van hierdie sindrome suksesvol is steun die teorie dat 'n tot dusver obskure biochemiese etiologie in baie gevalle mag bestaan.

Hierdie algemene simptome is dikwels vir die pasiënt hinderlik genoeg om hom te dwing om sy geneesheer te raadpleeg. So vind ons dat 'n paneel van vier algemene praktisyns vind dat 14% van hul pasiënte kla van simptome wat nie aan hul siekte toegeskryf kan word nie, en 'n ervare internis beweer dat 30% van sy konsultasies suiwer neurose of psigoneurotiese probleme is.[1]

Daar is twee gevolge van hierdie verskynsel wat nie altyd besef word nie: Vanweë die feit dat neurotiese pasiënte geredelik 'n dokter raadpleeg, word organiese siekte dikwels vroeër by hulle vasgestel. In so 'n geval word simptome van die neurose dikwels toegeskryf aan die organiese toestand. Die neurotiese simptome mag dikwels ook voorkom in organiese siekte, en die organiese siekte word behandel sonder dat die pasiënt se simptome noodwendig verbeter. Die klassieke voorbeelde van hierdie twee kategorieë is natuurlik hipertensie en anemie.

Hipertensie is meestal asimptomaties; die simptome wat wel die pasiënt by die dokter bring is gewoonlik te wyte aan die gevolge van die hipertensie, bv. linker hartversaking, iskemiese hartsiekte, serebrovaskulêre ongelukke of nierversaking. Die ander simptome, dikwels toegeskryf aan hipertensie, kom ook ewe dikwels as neurotiese simptome voor: hoofpyn, kragteloosheid, moegheid, senuagtigheid, slaaploosheid, winderigheid, hartkloppens en duiseligheid. Dit is te betwyfel of daar veel grond is vir die stelling dat enige tipe hoofpyn in hipertensie kan voorkom. Dit is miskien redelik om slegs die klassieke dowwe, kloppende oksipitale hoofpyn wat soggens by ontwaking reeds teenwoordig is, en wat deur die dag verbeter, direk aan die hipertensie te koppel. Die teksboek-stelling dat hipertensielyers besonder geneig is tot emosionele en psigiese probleme moet ook betwyfel word want dit is meestal by sulke neurotiese pasiënte dat asimptomatiese hipertensie gevind sou word. Die ewewigtige, flegmatiese persoon sonder neurotiese simptome sal bes moontlik eers by die dokter opdaag wanneer angina of nagtelike dispnee hom daartoe dwing. Die nadeel van hierdie verskynsel is dat die pasiënt dikwels deur sy dokter verseker word dat sy moegheid, hoofpyn of hartkloppens aan sy hipertensie toe te skryf is. Van daardie oomblik af beoordeel die pasiënt sy bloeddruk op sy simptome en die dokter sy bloeddruk op die manometer. Wanneer laasgenoemde dan sê: 'Jou bloeddruk is nou so-te-sê normaal' word dit dikwels begroet met geskokte ongeloof: 'Maar dokter my hartkloppens/hoofpyn/moegheid is dan nog net dieselfde'. Wanneer ons 'n simptoom aan hipertensie koppel en dit is nie duidelik aan die gevolge van hipertensie te wyte nie, is dit noodsaaklik om in die eerste plek die neurose te behandel, en indien nodig ook die hipertensie. (Die klaarblyklike betekenisvolle hipertensie in 'n jong persoon word natuurlik voldoende ondersoek, en waarna ons hier verwys is veral die matige hipertensie in 'n middeljarige, neurotiese persoon.)

Die diagnose van anemie as oorsaak van moegheid, duiseligheid, hartkloppens, prikkelbaarheid, hoofpyn en kortasemheid is eweneens in die neurotiese pasiënt 'n groot probleem. Dit val te betwyfel of hierdie simptome aan anemie toegeskryf kan word as die hemoglobien bo 9 - 10 G./100 ml. is. Hierdie 6 simptome is weliswaar belangrike en algemene simptome van anemie, maar Wood en Elwood[2] kon geen korrelasie tussen die hemoglobienvlak en die simptome vind by 'n groot reeks van vroue met hemoglobien-vlakke tussen 10 en 14 G./100 ml. nie. By 360 Britse huisvroue wat vir 'n roetiene borskas röntgenfoto aanmeld, vind Berry en Nash[3] simptome van geringe aard by 94%. Onder hierdie vroue was daar 14% wat verklaar het dat hulle 'onder die merk' voel, 37% het gekla dat hulle makliker kortasem word of meestal kortasem is, en 43% het gekla van chroniese moegheid. Die hemoglobien-vlakke was in al drie groepe dieselfde, en hoewel diegene met 'n hemoglobien onder 10·8 G./100 ml. meer simptome gehad het as die res, was die verskil nie statisties betekenisvol nie. Die les hieruit te leer is weer eens dat ons die hemoglobien konsentrasie van 9 G./100 ml. in 'n vrou moet ondersoek en volgens oordeel behandel, maar ons moet nie noodwendig enige verbetering in simptome verwag tensy ons die meegaande neurose ook behandel nie.

'n Derde simptoom wat soms weer te geredelik aan 'n neurose toegeskryf word is hiperventilasie. Dit is waar dat herhaalde aanvalle van lighoofdigheid of flou-gevoel sonder bewussynsverlies dikwels aan hiperventilasie toegeskryf kan word, en dat in die emosioneel-versteurde pasiënt borskaspyn van onskuldige oorsprong hiperventilasie mag teweegbring, maar hiperventilasie mag soms ook ontstaan as gevolg van angina pectoris en miokardiale infarksie, soms ook as gevolg van longembolis of in serebrovaskulêre ongelukke.

Dit is dus nodig om simptome nie te skerp af te baken as kenmerkend van 'n bepaalde toestand nie, en om by die bevinding van relatief geringe organiese afwykings nie die simptome te geredelik aan die organiese siekte te koppel nie. Miskien het die ou Vader van Geneeskunde dit in gedagte gehad toe hy gesê het: 'Die ondervinding is bedrieglik ... die oordeel is moeilik'.

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2. Wood, M. N. en Elwood, P. C. (1966): Brit. J. Prev. Soc. Med., **20**, 117.
3. Berry, W. T. C. en Nash, F. A. (1954): Brit. Med. J., **1**, 918.

BILHARZIA AND ITS PROGRESS IN SOUTH AFRICA

After the first Southern African discovery of *Schistosoma haematobium* at Uitenhage by Harley in 1864[1] numerous other workers reported the disease and by 1872[2] it was recognized as endemic in the Eastern Cape Province and Natal. It was widespread in the Rustenburg and Brits districts in the Western Transvaal in 1890, and shortly after this Middelburg, Schweizer Reineke and Klerksdorp were incriminated. The first intimation that *S. mansoni* was endemic came from Turner in Natal in 1908,[3] who described the clinical and pathological aspects without differentiating the parasites.

In 1938 Porter[4] depicted the geographical distribution of the two diseases and their snail hosts, mentioning that the snail distribution was wider than that of the disease. Since then the geographical distribution of both *S. haematobium* and *S. mansoni* has been defined a little more accurately and, contrary to popular belief, bilharzia is less widespread today than Porter showed, with no evidence anywhere of geographic spread.

S. haematobium occurs over the whole of the Transvaal north of latitude 26° south, with the exception of certain highveld areas in the east. Isolated foci are found at Wolmaransstad, Klerksdorp and Potchefstroom and in the northern and western suburbs of Johannesburg. The disease is endemic in the middle and lowveld of Swaziland, in Natal east of the Drakensberg and the coastal belt of the Transkei and Eastern Cape as far south as the Humansdorp district. In the Eastern Cape it would appear to be decreasing.

S. mansoni is found in the Transvaal north of the Zoutpansberg and in the whole of the Eastern Lowveld, with one or two isolated foci in the central Transvaal. In Swaziland it occurs in the lowveld as far south as about Big Bend. In Natal the disease is probably focal in distribution and, as far as is known, does not extend south of Durban—but the position is by no means clear.

The distribution of the snail intermediate hosts is more widespread than the disease, due probably to many factors, but the belief that the presence of the snail host automatically incriminates an area as endemic is not true: there are numerous areas, notably in South West Africa, Western Transvaal, Northern Orange Free State, Southern Transvaal, Northern Natal and the Transkei where snail hosts are found without the corresponding schistosome. Whether the schistosome could flourish in these areas is doubtful, provided ecological conditions remain static. However, development in non-endemic areas and areas of low endemicity might result in drastic ecological changes producing suitable conditions for the propagation of the parasite.

Infection rates of *S. haematobium* in Bantu children vary from about 10% to over 90% in the Eastern Transvaal lowveld and Northern Zululand. Low rates are related to good water supplies, scarcity of human population or low temperatures. High rates are always related to communal swimming in hot areas.

Infection rates of *S. mansoni* vary from about 30% to over 90% in the Transvaal. Low rates are associated with low human population density, improved water supplies and sometimes better agricultural methods. High rates are invariably associated with high population density, unprotected water supplies and a consequent high degree of faecal pollution of water. There is little relationship between the numbers of snails and bilharzia infection rates.

Although the two diseases differ from each other in the causal organism, snail hosts, site of pathology, epidemiology, response to treatment and therefore treatment, they do not differ in one very important aspect: both are primarily diseases of the Bantu—*S. haematobium* of Bantu children and *S. mansoni* of Bantu children and adults. Consequently, effective general control measures must in the first place be directed towards protecting these people.

To many the control of bilharzia means the application of molluscicides. Thousands of tons of these chemicals have flowed down rivers, streams and irrigation canals, or have been applied to dams and pools. The fact that so many of them have been developed and been given extensive laboratory and field trials is fair testimony to the interest shown by chemical firms and other people working on control. Thousands of hours have been spent, at enormous expense, evolving methods of application and working out population dynamics of the snail hosts, yet it is remarkable that no country has adopted molluscicides on an over-all national basis. That they have their place cannot be denied. But in a developed country like South Africa the over-all use of a control measure which completely disregards the human host in his changing environment, disregards the basic reason for the increase of bilharzia.

Development has aggravated bilharzia by directly increasing Bantu population densities on irrigation schemes, round White townships and in Native Reserves, with no corresponding improvement in water supplies. Experimental field observations indicate that infection rates are governed largely by water supplies and usage and human population densities. In other words bilharzia is merely a symptom. The true disease is bad water supplies. Fortuitously the change from a scattered rural to a congregated environment has brought about a situation where an improvement of the presently bad Bantu water supplies is a distinct possibility. It is also a practical and urgent necessity throughout the country. In endemic areas piped water on a communal basis, for everyday household use, is essential and must be supplemented by cheap swimming baths—which, on a *per capita* basis, are neither unrealistic nor expensive—and by fencing or other means to prevent human contact with potentially dangerous water near their homes.

These permanent measures alone have shown that the symptoms are alleviated slowly. Quicker relief once curative measures for the disease have been instituted, would appear to lie in mass therapy.

1. Harley, J. (1864): Med.-Chir. Trans., **47**, 55.
2. Batho, R. (1872): Army Medical Department Report (London), **12**, 502.
3. Turner, G. A. (1908): Parasitology, **1**, 195.
4. Porter, A. (1938): Publ. S. Afr. Inst. Med. Res., **8**, No. 42.