South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 10 March 1956
Weekly 2s. 6d.

Vol. 30 No. 10

Kaapstad, 10 Maart 1956
Weekliks 2s. 6d.

EDITORIAL

LESSER KNOWN FEATURES OF DIABETIC NEUROPATHY

Last week we discussed the occurrence of pain in diabetes;[1] we now return to the subject and refer to other manifestations of diabetic neuropathy which are of less common occurrence.

The autonomic nervous system has recently received considerable attention in diabetics.[2, 3] Apart from the complaints probably referable to disordered function of this system (impotence, diarrhoea, bladder disturbances, 'trophic' changes in the limbs) it has been found that a fair percentage of diabetics suffer from attacks of giddiness caused by postural hypotension, and show widespread loss of sweating and defective thermal regulation in the skin without vascular disease.

The pupillary reaction may be abnormal, and while the complete Argyll-Robertson pupil is rare in diabetics yet it has been occasionally reported.[4, 5]

Isolated or multiple cranial-nerve palsies, particularly oculomotor, have often been described in diabetes and are usually accepted as a part of the diabetic picture.[6, 5, 7] There is, however, some doubt expressed about this, and Bailey[8] has recently reported figures from the Mayo Clinic which do not support this concept. Thus in his series only 10 of 276 patients with external ocular palsies were diabetic, and of these all were elderly, and most hypertensive. We have certainly seen ocular pareses in more than 10 diabetics, all of which cleared up completely within several weeks. This total recovery seems to be invariable in diabetics, often as the carbohydrate state is better controlled. In this event, especially where a cerebral angiogram has been normal and the Wassermann test negative, it is difficult to conclude that the diabetes is in all cases merely incidental.

Pain is apparently uncommon in cranial-nerve affection, and when it does occur it is usually in the distribution of one or other branch of the trigeminal nerve,

VAN DIE REDAKSIE

MINDER BEKENDE KENMERKE VAN DIABETIESE NEUROPATIE

Verlede week het ons die pyn wat by suikersiekte voorkom, bespreek;[1] ons verwys nou na ander manifestasies van diabetiese neuropatie wat interessant en van belang met diagnose is.

In die jongste tyd is heelwat aandag bestee aan die outonomiese senuweestelsel van suikersiektelyers.[2, 3] Benewens moeilikhede wat waarskynlik aan stoornisse in die werking van dié stelsel toe te skryf is (diarree, onmag, blaasmoeilikhede, trofiese veranderings in die ledemate), ly taamlik baie suikersiektepasiënte aan aanvalle van duiseligheid wat aan liggaamshoudinghipotensie te wyte is en alhoewel daar geen tekens van vaskulêre aandoening is nie, sweet hul deurgaans minder en is die termiese regulering in die vel ondoeltreffend.

Die pupilreaksie mag abnormaal wees en hoewel 'n volmaakte Argyll-Robertson-oogappel selde by suikersiektelyers gesien word, word dit nogtans af en toe gerapporteer.[4, 5]

Enkel of veelvoudige verlamming van skedelsenuwees, veral okulomotories, word dikwels in gevalle van suikersiekte beskrywe en word gewoonweg beskou as deel van die suikersiektebeeld.[6, 5, 7] Daar bestaan egter 'n mate van onsekerheid hieroor en Bailey[8] het onlangs Mayokliniekgegewens aangehaal wat nie hierdie beskouing steun nie. Aldus was daar in sy reeks van 276 pasiënte met eksterne okulêrverlamming net 10 suikersiektelyers—almal bejaard met 'n besondere hoë bloeddruk. Ons het definitief okulêrparese by meer as 10 suikersiektelyers gevind. Dié parese het in al die gevalle, na verloop van 'n paar weke, geheel en al verdwyn. Hierdie volkome herstel skyn die reël by suikersiekte te wees en herstel tree dikwels gelyktydig met die verbetering in koolhidraatbeheer in. Dit is moeilik om te glo dat die suikersiekte altyd net bloot toevallig was veral as die serebraalangiogram normaal en die Wassermanntoets negatief vertoon het.

Oënskynlik word pyn selde ondervind deur pasiënte wie se skedelsenuwees aangetas is en indien wel dan is dit gewoonlik in een of ander takverspreiding van die drieling senuwee en gaan dit gepaard met 'n mate van gevoelverlies in die betrokke gebied.[9]

together with some loss of sensation in the appropriate area.[9]

In diabetics, perforating ulcers in the feet, gangrene with apparently good vascular supply, and Charcot joints (usually tarsal), seem to be caused by a multiplicity of factors. These include a loss of sensation with consequent repeated unheeded traumata producing damage to the feet, paralysis of the sympathetic nerve-supply, and infection. From a practical point of view an ill-fitting shoe, athlete's foot and personal pedicure are most important and the undesirability of these should be pointed out to all diabetics from the very start. A chiropodist should perform the pedicure. Unlike the state of affairs in tabes, the Charcot joint in diabetes often has an infective element in it.[10, 2]

It is well known that diabetic neuropathy may mimic tabes, with lightning pains, pupillary changes, perforating ulcer, absent deep-pressure pain, lost reflexes, impotence, urinary difficulties, Rombergism, and increased protein in the cerebrospinal fluid. Cases occur in which it is difficult to determine whether the neurological state is diabetic or tabetic.

Diarrhoea, which is intermittent and mainly nocturnal, usually with faecal incontinence, and often interspersed with periods of severe constipation, is a recognized complication of diabetes,[11, 12] and is believed to be related to damage to the autonomic nerves of the gut. If this is so then it seems logical to try hexamethonium or banthine. Crude liver extract (recommended empirically by Joslin) avoids the hazard of severe ileus, which has been seen after banthine.

The frequency with which impotence occurs with the onset of diabetes in the male, disappears after a few weeks' treatment (often on diet only), and is unaccompanied by features of peripheral neuritis, leads one to doubt a neuropathic causation. Certainly, however, it may also appear later in the course of the diabetes. It is far commoner than is generally realized and provides a virgin field for research.

An atonic bladder, with loss of sensation of fullness, consequent incontinence, and infection, is a serious, late and usually irreversible complication. Transurethral resection of the bladder neck may be necessary.[13] The spinal cord itself is probably affected (the 'cord bladder').

Involvement of the dorsal nerve-root explains the tabetic-like syndrome.[14] Pathological examinations have shown that the cord itself may be diseased, though the exact type of change is not at all clear.[15, 16] Clinically two types of cord disturbance (apart from 'cord bladder') are described. One is almost purely motor, a sort of chronic anterior poliomyelitis, occasionally with Babinski reflexes,[17, 18] the second is mixed and resembles subacute combined degeneration. In view of the rarity of these, it will obviously be difficult in any given case to be sure

In suikersiektegevalle is perforerende voetsere, gangreen met bevredigende vaskulêre toevoer, en Charcot-gewrigte (meesal tarsaal) skynbaar te wyte aan menigvuldige faktore o.a. die verlies van gevoel. Hierdie faktor gee aanleiding tot herhaaldelike traumata waarvan geen notisie geneem word nie en wat die oorsaak is van voetbeserings, verlamming van die simpatiese senuweetoevoer en infeksie. Dit is om praktiese redes uiters belangrik om alle suikersiektelyers uit die staanspoor te waarsku teen die nadelige gevolge van skoene wat nie goed pas nie, huidskimmelsiekte aan die voete (*athlete's foot*) en voetversorging (soos die verwydering van liddorings, toonnaels en eelte) deur die pasiënt self. Voetversorging moet aan 'n voetkundige oorgelaat word. Andersins as in die geval van uittering is daar dikwels in die Charcot-gewrig van suikersiekte 'n besmetlike element aanwesig.[10, 2]

Dit is alombekend dat diabetiese neuropatie 'n toestand van uittering (*tabes*) kan naboots, met skietende pyn, pupilveranderings, perforerende sere, verlies van reflekse, onmag, urienmoeilikhede, Rombergisme, afwesigheid van diepliggende pyn, toename van proteïen in die harsing- en rugmurgvloeistof. Dit is in sommige gevalle moeilik om te bepaal of die neurologiese kondisie aan suikersiekte of uittering toe te skryf is.

Diarree (gewoonlik met ontlasting-inkontinensie) wat met tussenpose, en hoofsaaklik in die aand, verskyn en dikwels deur tye van ernstige hardlywigheid onderbreek is, is 'n erkende komplikasie van diabetes.[11, 12] Die vermoede is dat dit in verband staan met beskadiging van die outonomiese senuwees van die derm. As dit wel die geval is, skyn dit logies om hexamethonium of banthine te probeer. Ongesuiwerde lewerekstrak (empiries deur Joslin aanbeveel) vermy die risiko van 'n ernstige dermknoop wat al na banthine waargeneem is.

Die frekwensie waarmee onmag by mans voorkom met die aanvang van diabetes, na 'n paar weke se behandeling verdwyn (dikwels net as gevolg van die dieet), en sonder dat dit gepaard gaan met tekens van periferale senuweeontsteking, laat mens aan 'n neuropatiese oorsprong twyfel.

Dit mag egter ook vanselfsprekend in 'n latere stadium van diabetes voorkom. Dit kom baie meer dikwels voor as wat algemeen besef word en die veld bied navorsingsgeleenthede aan vir die baanbreker.

'n Slap blaas, met verlies van die gevoel dat die blaas vol is, gevolglike inkontinensie en besmetting, is 'n ernstige, gevorderde en gewoonlik onkeerbare komplikasie. Dit mag 'n transuretrareseksie van die blaasnek vereis.[13] Die rugmurg self (die ‚*cord bladder*') is waarskynlik aangetas.

Aantasting van die rugsenuweewortel kan die tabesagtige sindroom verklaar.[14] Patologiese ondersoeke het getoon dat die murg self aangetas mag wees, presies op welke manier is nog glad nie duidelik nie.[15, 16] Klinies word 2 tipes murgsteurings (afgesien van die ‚*cord bladder*') beskrywe. Een tipe is amper suiwer motories, 'n soort van kroniese anterior poliomiëlitis, somtyds met Babinski-reflekse.[17, 18] Die tweede is gemeng en lyk na subakute gekombineerde degenerasie. Met die oog op die seldsaamheid van sulke gevalle, sal dit klaarblyklik moeilik wees om in enige gegewe geval seker te wees dat die etiologie by suikersiekte berus, alhoewel Garland

that the aetiology is truly diabetic, although Garland has described 12 cases of the motor type, all seen fairly recently.

The protein of the cerebrospinal fluid may be increased considerably, especially in those cases with more severe symptoms and signs. Root reports the protein as high as 440 and 435 mg. per 100 ml.[7] The colloidal-gold curve is also abnormal and conforms to no pathognomonic pattern. There is no increase in cells. It is important to realize that a raised protein in no way helps in the differential diagnosis from, say, tabes or the Guillain-Barré syndrome.

It is plain that diabetes is an excellent mimic. Nevertheless the great diagnostic pitfall lies in the too ready assumption that any complaint in a diabetic is caused by the diabetes.

1. Editorial (1956): S. Afr. Med. J., **30**. 208.
2. Martin, M. M. (1953): Lancet, **1**, 560.
3. Odel, H. M., Roth, G. M. and Keating, F. R. (1955): Diabetes, **4**, 92.
4. Jordan, W. R. (1936): Arch. Intern. Med., **57**, 307.
5. Rundles, R. W. (1945): Medicine, **24**, 111.
6. Collier, J., (1929): Proc. Roy. Soc. Med., **23**, 627.
7. Joslin, E. P., Root, H. F., White, P. and Marble, A. (1952): *The Treatment of Diabetes Mellitus*, 9th ed., chap. 17. Philadelphia: Lea and Febiger.
8. Bailey, A. A. (1955): Diabetes, **4**, 32.
9. Jackson, W. P. U. (1955): Brit. Med. J., **2**, 408.
10. Hodgson, J. R., Pugh, D. G. and Young, H. H. (1948): Radiology, **50**, 65.
11. Bargan, J. A., Bollman, J. L. and Kepler, E. J. (1936): Proc. Mayo Clin., **11**, 737.
12. Sheridan, E. P. and Bailey, C. C. (1946): J. Amer. Med. Assoc., **130**, 632.
13. Emmett, J. L., Daut, R. V. and Sprague, R. G. (1949): J.
14. Williamson, R. T. (1924): Practitioner, 112, 85.
Urol., **61**, 244.
15. Woltman, H. W. and Wilder, R. M. (1929): Arch. Intern. Med., **44**, 576.
16. Griggs, D. V. and Olsen, C. W. (1937): Arch. Neurol. Psychiat., **38**, 564.
17. Goodman, J. I., Baumoel, S., Frankel, L., Marcus, L. J. and Wassermann, S. (1953): *The Diabetic Neuropathies*. Illinois: C. Thomas.
18. Garland, H. (1955): Brit. Med. J., **2**, 1287.

onlangs 12 gevalle van die motoriese tipe beskrywe het, almal taamlik resent.

Die proteïen van die harsing- en rugmurgvloeistof mag aansienlik vermeerder, veral in pasiënte met ernstige simptome. Root gee proteïensyfers aan wat selfs so hoog as 440 en 435 mg. per 100 ml. is.[7] Die kurwe vir kolloïdale goud is ook abnormaal en stem nie ooreen met enige kenmerkende patroon nie. Daar is geen selvermeerdering nie. Dit is belangrik om te besef dat die vermeerdering van proteïene van geen hulp met onderskeidende diagnose tussen, laat ons sê, uittering (*tabes*) of die Guillain-Barré-sindroom is nie.

Dit is duidelik dat suikersiekte 'n baie goeie na-aper is. Nietemin is dit 'n groot diagnostiese strikval om te geredelik aan te neem dat elke kwaal waaraan die suikersiektepasiënt ly aan die suikersiekte te wyte is.

1. Van die Redaksie (1956): S.-Afr. T. vir Geneesk., **30.**
2. Martin, M. M. (1953): Lancet, **1**, 560.
3. Odel, H. M., Roth, G. M. en Keating, F. R. (1955): *Diabetes*, **4**, 92.
4. Jordan, W. R. (1936): Arch. Intern Med., **57**, 307.
5. Rundles, R. W. (1945): Medicine, **24**, 111.
6. Collier, J., (1929): Proc. Roy. Soc. Med., **23**, 627.
7. Joslin, E. P., Root, H. F., White, P. en Marble A. (1952): *The Treatment of Diabetes Mellitus*, 9de ed., hoof. 17. Philadelphia: Lea en Febiger.
8. Bailey, A. A., (1955): Diabetes, **4**, 32.
9. Jackson, W. P. U. (1955): Brit Med. J., **2**, 408.
10. Hodgson, J. R., Pugh, D. G. en Young, H. H., (1948): Radiology, **50**, 65.
11. Bargan, J. A., Bollman, J. L. en Kepler, E. J., (1936): Proc. Mayo Clin., **11**, 737.
12. Sheridan, E. P. en Bailey, C. C., (1946): J. Amer. Med. Assoc., **130**, 632.
13. Emmett. J. L., Daut, R. V. en Sprague, R. G., (1949): J. Urol., **61**, 244.
14. Williamson, R. T. (1924): Practitioner, **112**, 85.
15. Woltman, H. W. en Wilder, R. M. (1929): Arch. Intern Med., **44**, 576.
16. Griggs, D. V. en Olsen, C. W. (1937): Arch. Neurol. Psychiat., **38**, 564.
17. Goodman, J. I., Baumoel, S., Frankel, L., Marcus, L. J. en Wassermann, S. (1953): *The Diabetic Neuropathies*. Illinois: C. Thomas.
18. Garland, H. (1955): Brit. Med. J., **2**, 1287.