ENDEMIC SYPHILIS OR YAWS?

A REVIEW OF THE LITERATURE FROM SOUTH AFRICA

J. F. MURRAY, M.D.

Pathologist, South African Institute for Medical Research, Johannesburg

The first reference to the treponematoses in Southern Africa appears to be a statement by Lichtenstein[1] that, during his travels in Southern Africa between 1802 and 1806, he could find no evidence of syphilis amongst the Xhosas. Livingstone[2] in his reference to the Bechuana in 1857 stated that the disease was 'unable to maintain itself in any permanent form in persons of pure African blood anywhere in the centre of the country', but by 1885 Warren's expedition into Bechuanaland[3] found the Natives badly infected. From 1881[4] and onwards the disease appears to have spread rapidly and by 1887[5] the district surgeon of Bedford stated that more than half of 5,529 Natives examined by him were syphilitic.

An interesting and possibly a unique aspect of syphilis in South Africa is the statement by Mitchell[6] that 'a Chief in the Northern Transvaal was so impressed by the ravages of syphilis amongst his people and with the efficacy of inoculation or vaccination against smallpox, that he caused some 300 or 400 members of his tribe to be inoculated with material from an infective case of syphilis'. The results are not recorded.

The first clinical description of a case of treponematosis which we have been able to find in the early South African medical journals is by G. A. Heberden[7] of Barkly West, who described a case which he diagnosed as 'Frambesia or yaws.' The lesions, which were papulo-pustular, condylomatous, and polymorphous in nature occurred mainly on the head, neck, and face but were also scattered about the body and in the axillae and folds of the skin. The patient, a Bechuana, stated that he had seen many similar cases in Bechuanaland. Although Heberden classified the case as one of yaws it seems probable, in the light of modern knowledge concerning the treponematoses, that it was one of endemic syphilis. For 20 years there raged in the South African medical literature a lengthy, wordy and sometimes acrimonious controversy as to the exact nature of the treponematosis which was widely spread in Southern Africa and particularly in Bechuanaland. So heated did the discussion become that when McArthur and Thornton[8] asked the question, 'Is it necessary for there to be a primary chancre? Might it not be possible for the disease to be spread without a chancre at all?' Mathias[9] replied 'that the days of well authenticated miracles are well past and will probably never

come again, even in the interests of the District Surgeons of South Africa.'

On the one hand Mathias,[9-12] Hugo,[13, 14] Ricono[15] and Knobel[16] stoutly maintained that the condition was yaws, whereas McArthur and Thornton,[8] Cairns[17] and Walker[18] with equal confidence stated that the disease was syphilis. Mathias,[10] one of the most prolific writers on the subject, was emphatic that the condition was yaws. When confronted with Jonathan Hutchinson's dictum that yaws is 'syphilis modified by race and climate' Mathias thundered that 'it was only Hutchinson's great reputation as an authority on syphilis that maintained the existence of such an error for so many years, and made the attempts of others to preach the truth on this subject practically futile, and I believe that it is this alone which today accounts for the fact that yaws is practically not recognized.' The controversy is interesting in view of the fact that even today, 40 years later, despite our increased knowledge, dispute still exists about the exact relationship between these two treponematoses.

The opinion of McArthur concerning its exact pathogenesis varied from time to time although he was one of the most accurate observers of the condition. In his publication of 1911[8] he emphasized that the disease was transmitted from patient to patient by direct or indirect non-sexual contact and that it occurred mainly in childhood. Indeed, he gave an accurate and detailed report which differed in no important respect from the description of endemic syphilis by Hudson[19] in Iraq 25 years later. Although McArthur regarded late heredo-syphilis as an important feature in relation to the disease in Natives he thought that contact transmission was even more important. By 1922,[20] however, he had apparently modified his opinion and regarded heredo-syphilis as so prevalent that direct modes of transmission seemed to be of little importance among the Bechuanaland Natives. This opinion he maintained throughout the remainder of his publications and his weighty opinion continued to have an effect upon the approach to the subject of all medical men in South Africa.

Summarizing the South African literature over the last 50 years the following picture emerges.

INTRODUCTION AND SPREAD OF SYPHILIS

The Bantu in Southern Africa before their contact with the European appear to have been free of syphilis (Sax,[21] Mitchell[22]). Contact with the European introduced the disease amongst them and its spread was accelerated by the opening of the Kimberley diamond mines.[4] Possibly as a result of their proximity to the mines and of the high proportion of the male population which undertook labour at the mines the disease became particularly prevalent amongst the Bechuana. McArthur and Thornton[8] state that in 1904, of a population of 68,000 in Bechuanaland (excluding Gordonia), 12,000-14,000 were in employment on the Kimberley diamond mines annually.

According to Kark[23] living conditions of Africans in the Kimberley area were conducive to the spread of syphilis. Drunkenness was common, compounds were often filthy, and there was a rapid movement of men to and from the diggings. He emphasizes that a large number of men were living under abnormal social conditions and that promiscuity and prostitution were rife. As a result, by the turn of the century, the population was apparently heavily infected with syphilis, so much so that a Commission to report upon contagious disease amongst the Natives was appointed by the Government in 1906. The Commission[4] found that syphilis appeared to have been introduced somewhere in 1881, probably from Kimberley, and thereafter became widely prevalent so that its incidence varied from 'very little' to 'over 80% in some parts of the Zoutpansberg'. The Commission commented upon the varying incidence of the disease in different parts of the country but stated it to be 'enormously prevalent in the adjacent part of Bechuanaland'. They noted that the number of cases of syphilis and venereal disease treated at the Rietfontein Lazaretto in Johannesburg increased from 58 in 1900 to 659 in 1906.

At an intercolonial conference held in 1906 it was stated that syphilis was prevalent throughout the Native and Coloured races, that in some areas it was extremely prevalent and was increasing, that it was frequently imported by labourers returning from labour centres, that it was occasionally spread by servants to White persons, especially children, and that when it came under observation it was usually in the late tertiary state.

Almost all authors were convinced of the wide prevalence of syphilis amongst the Natives, but Leipoldt[24] expressed some doubt upon this point in 1920, mainly on the grounds that any estimate of incidence amongst the Bantu was seriously handicapped by the absence of accurate medical statistical information.

SEROLOGICAL FINDINGS

The first report upon the serological status of a Bantu population was that of Pijper,[25] who carried out Wassermann tests on 500 Coloured people at the Pretoria Hospital. The patients were of all ages between 6 and 60 and the sexes were about equally represented. The great majority were pure Natives but there were 'also some half-castes and mixtures of all kinds'. *Prima facie* cases of syphilis were excluded, but no further selection was made. The majority of the blood specimens were taken from traumatic surgical cases and from non-syphilitic patients from the medical wards and out-patient department. Many relations and visitors also contributed to the number of specimens. The results showed strongly positive reactions in 36·8% of the specimens and a further 11·0% which were definitely positive. In a further series of 40 apparently completely healthy Natives from Bethal district he found 12·5% positive Wassermann tests.[25]

Subsequent serological surveys in Southern Africa have shown a relatively high seropositivity rate, although the actual rate has varied considerably in different parts of the country. Schultz,[26] in 1926 obtained 16·6% strongly positive results in 242 Native males. In 1927, at the South African Institute for Medical Research,[27] specimens from 1,200 healthy Native mine labourers showed seropositivity rates as follows: Xhosa 2%, East Coast 7%, Pondo 8·5%, Bechuana 22%, Basuto 29·5%.

In 1937-39 Native males examined at the pass office in Benoni[28] showed 0·5% suffering from infective venereal disease. There were 5·0% showing superficial signs of old infections and 28·0% had a positive Wassermann reaction. The seropositivity rate among non-European ante-natal clinic patients in the same town was 35-42%.

Kark,[23] in 1949, concluded that few countries could have a higher incidence of syphilis than South Africa. Kark and

Le Riche,[29] in 1938-39, found 23·6% positive Wassermanns amongst African school children. Purcell,[30] in 1940 indicated that 'the incidence of syphilis in the Union is enormous'. Kark,[23] in 1949, reviewed the South African literature on seropositivity in various African groups and showed that it varied from 2·0% to 47·8%. The Annual Medical Report[31] of the Bechuanaland Protectorate Government for 1946 reported a seropositivity rate of 64% in 377 out-patients at Mahalapye.

After the introduction of serological tests the controversy whether the condition was yaws or syphilis appears to have died away in the South African literature and the high seropositivity rate amongst the Bantu was accepted as evidence of syphilis.

But it is not uncommon to find in private conversation that doubt is expressed about the interpretation of positive serological tests as evidence of syphilis. It has been felt by many that, owing to the numerous well-known biochemical aberrations found in any series of Bantu sera, many of the reactions might be biological false positives.[29] No method of testing this suggestion has been available until recently, but an investigation into this aspect of the problem has been proceeding at the Institute for Medical Research during the last 2 years and will be reported in detail in due course. Suffice it to state that the use of the *Treponema pallidum* immobilization (TPI) test and of a battery of serological tests for syphilis (STS) has confirmed the high seropositivity rate found by previous workers amongst the Bantu and has shown that, if one accepts a positive TPI as indicative of past or present treponemal infection, then a high rate of such infection exists amongst the Bantu.

CLINICAL MANIFESTATIONS

Reference is made above to the fact that the first case of treponematosis recorded in the early South African medical journals was described as yaws and subsequent publications were divided on the issue whether the common treponematosis seen in Southern Africa was syphilis or yaws.

The clinical picture (Ricono,[32] Garrow,[33] Hugo,[13, 14] Mathias,[9-12] Walker,[18] Heberden,[7] McArthur and Thornton,[8] Knobel,[16] McArthur[20]) was characterized by the presence of a papulo-pustular eruption (sometimes acuminated), condylomata in the flexures and moist areas of the skin, mucous patches in the mouth and throat, and periostitis, particularly of the legs and arms.

A notable fact was that these signs were seldom preceded by a primary chancre. Garrow[33] mentions that the condition sometimes originated sexually and then spread innocently, as in a group of farm workers which he described. McArthur and Thornton[8] refer to 27 cases spread commensally amongst children in a children's home in Kimberley. They regarded many cases of the condition as hereditary but they admitted that the usual stigmata of hereditary syphilis were absent. While they regarded sexual spread as important in the urbanized African, they felt that in the rural Bantu indirect spread by the use of common utensils and clothing was much more important. In the tertiary-stage destructive gummatous lesions occurred in the skin, nasopharynx and bones, but the viscera were seldom affected, cardio-vascular complications seldom occurred, and there were, apparently, no remote central-nervous-system complications such as tabes dorsalis and general paralysis of the insane. The destructive lesions of the nasopharynx sometimes became very extensive[14] and resembled the cases of mutilating endemic syphilis which have been described by Jones.[34] The lesions, which were clearly the tertiary lesions of endemic syphilis, were sometimes described as late manifestations of congenital syphilis (Hill-Aitken[35]).

Another feature of the condition was that not infrequently the lesions spontaneously healed and all authors affirm the rapid response to such antisyphilitic measures as potassium iodide, iodiform ointment, mercury and (at a later date) arsenical injections.

The association of a favoid condition of the scalp was, by some authors, regarded as a syphiloderm (McArthur and Thornton,[8, 20] Marshall and Wilson,[36]) but others regarded it as a favus infection (Mitchell and Robertson[37], Murray *et al.*[38]).

Those who felt that this treponematosis was yaws based their opinion upon the following facts:

(*a*) That a primary chancre was seldom observed,

(*b*) that there was little or no evidence of sexual spread,

(*c*) that it occurred most frequently in childhood,

(*d*) that there were no multiform eruptions,

(*e*) that the internal organs were seldom affected,

(*f*) that there was no iritis,

(*g*) that no central-nervous-system sequalae such as general paralysis of the insane or tabes dorsalis occurred,

(*h*) that patients readily recovered under external applications such as iodiform ointment, and some without any treatment at all, and

(*i*) that no hereditary manifestations were found in the offspring.

It is now known that all these arguments are equally applicable to endemic syphilis (Hudson,[19] Grin,[39] Guthe and Willcox[40]) and it is clear that the condition which was so widely spread amongst the Bantu of Southern Africa was endemic syphilis and not yaws. In recent years clinical descriptions of endemic syphilis have been published from Southern Africa (Murray *et al.*,[41] Willcox,[42] Taylor[43]). The disease still, apparently, occurs in the more remote parts of the subcontinent and is identical with the treponematosis described in earlier writings as yaws.[9-16] This is not to say that yaws has never occurred in Southern Africa, but the only authentic examples of it which appear in the South African literature (Hackett,[44] Bensusan[45]) clearly indicate that the disease had been imported from one or other of the endemic yaws areas in tropical Africa.

ETIOLOGY AND PREVENTION

The introduction of better hygiene of schools and of water supplies is, apparently, in itself sufficient to diminish the incidence of endemic syphilis in a primitive community. This has been exemplified in Southern Africa, where the incidence of the condition was found by Murray *et al.*[41] to be appreciably higher in the more backward areas of Bechuanaland as compared with the more advanced sections of the community.

It is of interest to note that, although penicillin was as yet unknown, McArthur and Thornton[8] as early as 1911 realized the value of mass therapy in the control of endemic

syphilis. They recognized the response of cases to minimal therapy and suggested that 'the difficulty of dealing with syphilis will disappear, for all that will be necessary is for each patient, and for any who react to the serum tests, to be injected with the solution of the new drug (606). And if the serum diagnosis cannot be simplified so as to become a practical measure, but Ehrlich's remedy stands the test of time, we consider that, in order to eradicate the disease, the Government might perhaps be justified in seeking from the Legislature powers to order the injecting of the new drug into every member of the known syphilitic families in this area'.

In rural areas of the Union standards of living amongst the Bantu have apparently advanced sufficiently to diminish appreciably the incidence of endemic syphilis amongst them. Even in the Native urban townships, insanitary and unhygienic though many of them are, the incidence of endemic syphilis is relatively low and only minor outbreaks occasionally occur. (Taylor,[43] Sachs,[46] Murray *et al.*[41]). The place of endemic syphilis in the South African community is apparently being taken by the classical venereal form of the disease, but the incidence of the latter, together with the occurrence of occasional sporadic outbreaks of endemic syphilis, is sufficiently high to maintain a high seropositivity rate amongst the Bantu of Southern Africa.

No satisfactory comparative studies have been carried out in the Union to determine the relative incidence of seropositivity in different sections of the Bantu population, such as school children compared with non-school children, rural compared with urban Bantu, various age groups, and so forth. A careful study of this type would give valuable information on the part syphilis is now playing in the Bantu populations of the Union of South Africa.

SUMMARY

The literature on syphilis in the Bantu of Southern Africa has been reviewed and the opinion is expressed that the condition frequently described in the past as yaws was, in fact, endemic syphilis. Cases of true yaws have been infrequent and have occurred only when imported from the tropical areas of Africa. The high rate of endemic syphilis in the past and the continuance of it in some foci, together with the occurrence of classical venereal syphilis, has led to a high seropositivity rate amongst the Bantu.

Use of the TPI test has confirmed that the high seropositivity rate found in the Bantu of Southern Africa is due to treponemal infection.

REFERENCES

1. Lichtenstein, H. (1812): Quoted by Sax, S. (1952): S. Afr. Med. J., **26,** 1037.
2. Livingstone, D. (1857): *Missionary Travels and Researches in Southern Africa*, p. 128. London: John Murray.
3. *Warren's Expedition.* Official Report. Quoted by McArthur, D. C. (1922): Amer. J. Syph., **7,** 569.
4. *Report of the Contagious Diseases Commission* (1907): Pretoria: Government Printer.
5. *Report of the Select Committee on Reports on District Surgeons* (1888): Quoted by Anning.[28]
6. Mitchell, J. A. (1926): Med. J. S. Afr., **21,** 210.
7. Heberden, G. A. (1898): S. Afr. Med. J., **5,** 274.
8. McArthur, D. C. and Thornton, E. N. (1911): S. Afr. Med. Rec., **9,** 18.
9. Mathias, J. (1911): *Ibid.*, **9,** 64.
10. *Idem* (1910): *Ibid.*, **8,** 102.
11. *Idem* (1911): *Ibid.*, **9,** 128.
12. *Idem* (1911): *Ibid.*, **9,** 174.
13. Hugo, D. de V. (1911): *Ibid.*, **9,** 41.
14. *Idem* (1915): *Ibid.*, **13,** 107.
15. Ricono, M. (1911): *Ibid.*, **9,** 215.
16. Knobel, J. B. (1916): *Ibid.*, **14,** 151.
17. Cairns, P. T. (1911): *Ibid.*, **9,** 67.
18. Walker, P. H. (1911): *Ibid.*, **9,** 182.
19. Hudson, E. H. (1937): Trans. Roy. Soc. Trop. Hed. Hyg., **31,** 9.
20. McArthur, D. C. (1922): Amer. J. Syph., **7,** 569.
21. Sax, S. (1952): S. Afr. Med. J., **26,** 1037.
22. Mitchell, J. A. (1917): S. Afr. Med. Rec., **15,** 186.
23. Kark, S. L. (1949): S. Afr. Med. J., **23,** 77.
24. Leipoldt, C. L. (1920): Med. J. S. Afr., **16,** 25.
25. Pijper, A. (1921): S. Afr. Med. Rec., **19,** 302.
26. Schultz, H. J. E. (1921): Med. J. S. Afr., **21,** 209. Quoted by Anning.[28]
27. A. R. S. Afr. Inst. Med. Res. (1927): P. 56.
28. Anning, C. C. P. (1948): Leech, **19,** 9.
29. Kark, S. L. and le Riche, H. (1944): Manpower, **3,** 1.
30. Purcell, F. W. F. (1940): S. Afr. Med. J., **14,** 453.
31. Ann. Med. Rep. Bechuanaland Protectorate Govt. (1946).
32. Ricono, M. (1916): S. Afr. Med. Rec., **14,** 83.
33. Garrow, A. (1915): *Ibid.*, **13,** 89.
34. Jones, L. G. G. (1953): Brit. J. Vener. Dis., **29,** 104.
35. Hill-Aitkin, C. J. (1908): Transv. Med. J., **3,** 310.
36. Marshall, J. and Wilson, G. A. (1949): S. Afr. Med. J., **23,** 783.
37. Mitchell, J. A. and Robertson, G. W. (1915): S. Afr. Med. Rec., **13,** 28.
38. Murray, J. F. *et al.* (1957): S. Afr. Med. J., **31,** 657.
39. Grin, E. I. (1952): Bull. Wld. Hlth. Org., **7,** 1.
40. Guthe, T. and Willcox, R. R. (1954): Chron. Wld. Hlth. Org., **8,** 37.
41. Murray, J. F. *et al.* (1957): Bull. Wld. Hlth. Org., **15,** 975.
42. Willcox, R. R. (1951): S. Afr. Med. J., **25,** 501.
43. Taylor, W. N. (1954): *Ibid.*, **28,** 176.
44. Hackett, C. J. (1953): 1*st International Symposium on Yaws Control*, Wld. Hlth. Org., p. 158.
45. Bensusan, A. D. (1952): S. Afr. Med. J., **26,** 389.
46. Sachs, S. B. (1953): Personal communication.