THE HISTORY OF SYPHILIS IN UGANDA

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SYNOPSIS

The circumstances of an alleged first outbreak of syphilis in Uganda in 1897 are examined and attention is drawn to certain features which render possible alternative explanations of the history of syphilis in that country. It is suggested that an endemic form of syphilis was an old disease of southern Uganda and that protective infantile inoculation was practised. The country came under the observation of European clinicians at a time when endemic syphilis was being replaced by true venereal syphilis. This process has now been completed, endemic syphilis has disappeared, and venereal syphilis is now widespread and a more serious problem than ever. This theory explains the observations of other writers and reconciles the apparent discrepancies between various reports.

Until comparatively recent times the country now known as Uganda was cut off from the rest of the world. The Nile swamps to the north, the impenetrable Congo forest to the west, the mountains and the upland plateaux with the warrior Masai to the east, and the other immense difficulties of African travel, had protected the country from intrusion. In the southern lacustrine areas there had developed the remarkable indigenous kingdoms of Bunyoro and Buganda. These became conscious of the larger outside world about 1850, when a Baluch soldier from Zanzibar reached the court of the King of Buganda, the Kabaka Suna. This first traveller was followed by the Arabs of the coast, and as the Baganda became aware of the outside world, so did the outside world become aware of the remarkable inland kingdoms and strove to reach them. Such a discovery had hardly been paralleled since the exploits of Cortes and Pizarro.

The first European to enter Uganda, the explorer Speke, arrived in 1862. Others followed, the most important from the social, political and medical points of view being H. M. Stanley. His expedition in 1888 for the relief
of Emin Pasha, cut off by the Mahdist revolt from his government in Egypt, was of momentous consequence to Uganda. Stanley travelled from the west coast, cutting his way with the greatest difficulty through the Congo forest. By this expedition it would seem that he introduced into Uganda both human trypanosomiasis and the chigger flea, and possibly other diseases.

The latter years of the nineteenth century in Uganda, until the proclamation of a British Protectorate in 1894, were full of social and political troubles with much fighting between the various factions; the Protestant, Catholic, and Moslem communities were struggling for supremacy, and a great loss of social cohesion and a loosening or abandonment of previous customs and traditions took place. There was an influx of strangers into Uganda—Sudanese, Arabs from the coast, Indian and other troops as well as British, and members of other communities.

Some of the early explorers were medical men or had, inevitably, medical interests, but the concern of most of the early physicians was with the health of troops and government officials rather than with the medical needs of the indigenous population. It was not until 1897 that the famous missionary doctor, Albert Cook, came to Uganda to devote his labours to the native people. He soon realized the seriousness of the great trypanosomiasis epidemic which was reaching tremendous proportions. In 1902 a Sleeping Sickness Commission was sent from England and this brought to Uganda a galaxy of medical talent thus early in its history—Bruce, Castellani, Low, Nabarro, and others. The trypanosomiasis epidemic was still under study when attention was drawn to the ravages of syphilis in the population, and help was sought from England. In 1906 Colonel Lambkin, of the Royal Army Medical Corps, a leading British expert in venereal diseases, came to Uganda. He studied the local conditions, made a report, and drew up a scheme for the control of syphilis based largely on the use of intramuscular injections of mercurial compounds. This scheme was put into operation gradually by Keane and others in the years before the First World War, which brought operations to a standstill. After the war the antisyphilis campaign, in a modified form, was restarted, and ever since, syphilis has been considered a disease of major importance in Uganda, as indeed it is.

Now, with such a short, clear-cut history, and with the documentation provided by keen and skilled observers, it might seem an easy matter to follow the history of syphilis in Uganda from the time it first came under European observation to the present day. No searching in mediaeval archives, no pondering of the exact meaning of Greek or Latin phrases, is required. Yet, in fact, the history of syphilis in Uganda is most confusing. Contradictions and inconsistencies abound, and the older and more recent observations can be reconciled only with difficulty. The writer first became aware of this when engaged with colleagues in a study of congenital syphilis
in Uganda, and it impressed him even more strongly when he tried to assess the syphilitic lesions seen at autopsies on Uganda Africans.

The Early Evidence

Speke,22 who had to give much time to medical work among the Baganda at the Kabaka Mtesa’s court, hinted at seeing syphilitic lesions when he first entered the country a decade or more after the first Arabs from the coast had arrived. Wilson & Felkin 27 noted that “syphilis is extremely common”, an observation based on their experience between 1877 and 1879, and one which is of great weight because Felkin was a physician. Cook 6 stated quite definitely that

“between 1897 and 1907, the latter being the year of Colonel Lambkin’s investigations, the incidence of syphilis increased rapidly. In 1921 we re-examined the question and on purely clinical grounds, or indeed by merely getting the medical histories of large numbers of natives, we came to the conclusion that 80% of these people had, at one time or another, suffered from syphilis”.

The dates mentioned are worth notice. In 1901 Moffat 19 noted the fact that although gonorrhoea was common, stricture of the urethra was “exceedingly rare”, again a point of considerable importance; but he went on to say that

“syphilis is very common, with secondary and tertiary symptoms of all kinds . . . many cases of advanced syphilis come under observation in which there has been no previous treatment and in consequence the manifestations are often much more severe than those now generally seen in civilized countries . . . the commonest affections of bones are periostitis and ostitis generally of a syphilitic nature.”

The most clear-cut descriptions are those of Lambkin,17 who described an “outbreak of syphilis on virgin soil”. He considered that the disease had been introduced into Uganda by the Arabs about 1850, but had not become widespread until about 1897 when there was a sudden outbreak among the Baganda. (This year, 1897, was the year Albert Cook arrived in Uganda.) Lambkin considered that half the Baganda were affected, that in other parts, for example, Ankole, up to 90% were affected, and that the infantile mortality rate was 50%-60% because of syphilis. He described it as “mutilation everywhere”, as seen in the British Army in 1806 in Portugal. He wrote:

“in the primary form the true Hunterian chancre is the rule and this, as often as not, takes on a phagedaenic character resulting in wide destruction of the surrounding parts. The second stage is characterized by widespread and confluent eruptions, ulcerations of the mucous membranes, laryngitis, iritis, periostitis and joint affections, profound anaemia, cachexia and general disturbance of the nutrition”.

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* The country of Uganda is now divided into several provinces, which include the ancient kingdoms of Buganda on the north-west corner of Lake Victoria, and of Bunyoro-Kitara to the further north-west area. The inhabitants are distinguished by the prefix “Mu” in the singular and “Ba” in the plural. Thus Buganda is inhabited by the Baganda.
The tertiary stage was characterized by early rupial syphilides which extended rapidly over the body and limbs; osteo-arthritis lesions with severe osteoscopic pains, periostitis and necrosis of bones, breakdown of eyelids, ears, and nose. On the other hand, parasyphilitic lesions were not common. No mention was made of cardiovascular syphilis. Lambkin remarked on cases with loss of sexual power and early tabetic lesions but did not give details. He said that congenital syphilis was common, usually shown in boys and girls about 8-10 years of age as bilateral osteo-arthritis of the knees and caries of the bones of the face and skull. Interstitial keratitis with corneal opacities and choroiditis were common.

After this dramatic report, the Uganda Government took active steps to deal with syphilis in the Protectorate, starting in the Kampala area and later extending its activities to the Masaka area, whence Keane in 1912 described the situation. Tertiary syphilis in his experience was relatively uncommon, and the lesions relatively unimportant—for example, psoriatic lesions and chronic ulcerations, etc.—but nervous system lesions were very rare. The primary stage, too, was rarely observed, the vast majority of cases being seen in the secondary stage, and congenital syphilis was common, of a severe type and clinically closely resembling secondary syphilis. The clinical features of secondary syphilis were the dry or pustular skin rash, or cauliflower-like excrescences, mucous throat patches with masses of granulation tissue, ulcerations of the palate, arthritic and osteal lesions, pains in the ribs and chest, and well-marked condylomata. Treatment was by the intra-muscular injection of Lambkin’s mercurial cream, over a period of 14 weeks in $\frac{1}{6}$-grain doses. This produced marked and immediate changes; not uncommonly, all signs disappeared before the sixth dose without return during an observation period of 15 months. Congenital syphilis responded equally well to abdominal inunctions of 10% mercury ointment (British Pharmacopoeia), and tertiary skin ulcers to Lambkin’s calomel cream thinly smeared on lint and applied to the ulcer.

It will be noted that there are some discrepancies between the accounts given by Lambkin and by Keane. The latter recognized that the excellent response to mercurial therapy was

"at variance with recognized views and may be a feature of this disease in these people. Its obvious bearing on the practical problem of the suppression of the disease here is of immense importance for such persons are incapable of propagating acquired syphilis and are presumably immune from reinfection. They may become the subject of tertiary syphilis but that is a personal matter and not of public concern."

The campaign continued until the outbreak of the First World War forced a temporary check. In 1914 it was estimated that the population of Buganda was about 700,000. The number of cases treated per annum, as quoted in the Government reports, rose from 3690 in 1911 to 7412 in 1913. From native returns, it was estimated that the deaths from syphilis were
between 1750 in 1911 and 2287 in 1913, but little importance can be attached to these figures.

The antisyphilis campaign continued after the war, but little was written about syphilis until, in 1927, Webb & Halliday discussed the signs and symptoms of late syphilis in Buganda, based on the records of Mulago Hospital, itself founded as a venereal disease centre. The cases had been recorded between the years 1921 and 1926, and a Wassermann reaction was positive in all. The authors analysed the lesions found in 4000 men and 3711 women, classifying them under 11 main headings. In 2854 there were only subjective symptoms, and 890 had latent syphilis. Ulcerative conditions were present in 3015, the ulcers being usually single and occurring on the legs. Syphilis was regarded as responsible for 80%-90% of all ulcers seen in the hospital and clinically indistinguishable from non-specific ulcerations. Destructive ulcerative processes differed only in degree and virulence from the simple ulcerations and were seldom seen in women, but gummata were as frequent in women as in men. Dermatitis was seen in 1093 cases and was the commonest single lesion, but only 29 had rupial lesions. Ulcerative mucous membrane lesions occurred in 49 cases and non-ulcerative lesions in 82. Visceral syphilis was uncommon, as were neurosyphilis and syphilitic circulatory system disease. Stigmata of congenital syphilis were rarely seen, a feature which excited comment; 68 cases only were observed: 17 with keratitis, 11 with saddle noses, 16 with Hutchinson’s teeth, two with fissured lips and 22 with sabre tibiae.

This valuable paper is of great importance; the authors were cautious in their interpretations and did not discuss the previous literature. It seems that the greatest stress was laid on the Wassermann results, and the reliability of the Wassermann reaction was perhaps overstressed and overweighted, particularly in tropical Africa. There may well have been a high incidence of seroreactors, and in leg ulcers this probably led to many cases of tropical ulcer being regarded as syphilitic, for tropical ulcer in Buganda is much less common in adult females than in males, and Webb & Halliday noted that gummata occurred as frequently in females as in males.

Cook discussed the figures produced by Webb & Halliday in relation to antenatal syphilis as seen at Mengo Hospital, Kampala. He estimated the infant mortality rate at 300 per 1000 live-births, and thought syphilis a major cause of this; the percentage of his antenatal cases with a history of syphilis was over 60. In 1936 Cook stated that at Mulago Hospital, where Wassermann reactions were done as a matter of routine, there were corresponding antenatal figures; over 60% showed evidence of syphilis, although in 46% the infection was latent, and was diagnosed by serological reactions only. Cook claimed good results from the treatment of antenatal cases with mercury perchloride (grains 1/48 to the fluid ounce thrice daily throughout pregnancy).
Although Cook, Lambkin, and Keane had maintained his position, it will be noted that the discrepancies existing between the reports of Lambkin and Keane had been accentuated by the report of Webb & Halliday, and many further differences were disclosed by the review of Loewenthal in 1939. He saw little evidence of congenital syphilis in either the child or the adult population. Primary lesions were common and severe, visceral gummata and palatal perforation were rare, cutaneous gummata were uncommon, but cardiovascular syphilis and neurosyphilis were common. Billington studied neurosyphilis in Uganda and found tabes dorsalis to be rarely seen, but all other forms of neurosyphilis were frequently encountered. In 1947 Muwazi, Trowell & Davies discussed congenital syphilis; among 1000 carefully studied children only seven proven cases and six probable cases were seen. Congenital syphilis could not be considered a common cause of infantile deaths; it was extremely difficult to diagnose because of unreliable maternal histories, the frequency of fostering children, and the difficulties caused by malnutrition and sickle cell disease. Davies in 1947 discussed the post-mortem incidence of syphilis at Mulago and the type of lesions seen. The autopsy incidence was about 12%, with cardiovascular lesions in about 60% of syphilitic cases; the remainder were mostly neurosyphilitic cases. No more recent evidence has been brought forward to alter these views.

Before discussing these reports a word must be said about yaws. Castellani, who was in Uganda with the Sleeping Sickness Commission, states that "in Uganda and the region of the Great Lakes it is occasionally met with", an opinion which is most authoritative and which is supported by Moffat. He noted that, although yaws was not common in the lacustrine areas, it was widespread in the northern and western parts of the Protectorate, that is, away from the area we are considering, the Kingdom of Buganda. Hackett contrasts Buganda, where yaws was rare, with the northern part of Uganda, where it was common. These reports thus all correspond over the periods under consideration in concurring that yaws is not, and was not, common in the lacustrine areas.

**Discussion**

The pioneers of medicine in Uganda worked under difficult and often disheartening conditions with courage and enthusiasm; it would be quite unfair to criticize them personally from the comfort of an armchair and in the light of later fuller knowledge. We have, however, the right to assess the history of syphilis in Uganda on the basis of their writings, and it is evident that there are many discrepancies—so many as to require a careful reassessment of the evidence provided. It is equally obvious that syphilis now is a very different disease in Uganda from what it appeared to be, in its manifestations, fifty years ago. How has this come about? At one time
it was suggested that yaws and syphilis were confused. This theory is untenable. The evidence we have of the distribution of yaws in Uganda at that time is clear-cut, internally consistent and in line with recent evidence. It seems therefore impossible that they could have been mistaken to the extent compatible with the evidence available. Other possibilities must be explored first.

Was there an epidemic of syphilis in Uganda?

This question may be taken as a starting-point. Since the time of Lambkin, who was strongly supported by Cook, the belief that there was such an epidemic has not been challenged. Lambkin gives no figures, but merely gives percentages of up to 50% infected in Buganda, and up to 90% in Ankole. These are fantastically high and can be accepted only with caution. Some actual figures are quoted by Groen: "A. R. Cook reports that about 14.8% of the 13,000 patients treated as outpatients in 1908 were syphilitic according to the Mission Hospital in Mengo, Uganda; 11.4% of the patients admitted to hospital in the years 1903-1907 had syphilis". These figures are far from the percentages given by Lambkin, and are very similar to the figures given by Davies, based on autopsy studies. Such figures no doubt underestimate the incidence of syphilis in the population but Cook's figure of 14.8% was based on outpatients. We have no previous figures for comparison, so we cannot tell how this concords with the observations of Wilson & Felkin, previously cited.

We can take up other features of Lambkin's report. He thought that syphilis had been introduced by the Arabs about 1850 but that there had been an explosive outbreak about 1897. Therefore in seeking for an explanation of the outbreak he had to find some change about this time which would account for this rapid dissemination. His investigations convinced him that the major causes were the introduction of Christianity and the abolition of the punishments formerly meted out for immoral offences committed by either sex. In other words, the abandonment of polygamy and of the old restrictions on the liberty of women were the chief causes of the outbreak. Curiously enough, or perhaps not so curiously, Lambkin was supported in these views by both the Protestant and Roman Catholic missionaries and some of their prominent converts. J. Roscoe (quoted by Lambkin), an authority on the Baganda, went into some detail in support of Lambkin's views, saying that up until about 12 years previously the women of the tribe had been kept in strict surveillance and confinement; "in fact so strictly was this adhered to, that they were more like prisoners than anything else — hence immorality and promiscuous intercourse did not exist".

Before criticizing this attitude, attention may be paid to two other possibilities, one of which Lambkin regarded as doubtful. First, the habits of the Bahima tribe, a cattle-tending people, heavily indicted by Roscoe
and others as responsible because of their curious sexual habits. "Thus after a woman is married all sexual restrictions are thrown to the winds. She may welcome to her bed any of her husband's friends or relatives with impunity." Strangers and travellers were also so accommodated. Lambkin does not specify why he considered it doubtful if such easy-going habits contributed to the outbreak. He probably could not bring himself to suppose that these were recently acquired habits, nor that they should have suddenly become operative about 1897.

There was another cause which Lambkin discovered, of exceptional interest and importance, which throws much light on the position. He found that a practice existed "of deliberate vaccination of healthy infants with the syphilitic virus from affected persons, the reason given for the practice being that syphilis communicated in this way during infant life conferred immunity to it in the adult". Missionaries confirmed the existence of this custom; Father Laane had preached on more than one occasion against the practice of wrapping infants a few days old in clothes smeared in syphilitic discharges. Observers agreed that it was a common custom in some parts; Sir Apolo Kagwa, an outstanding Muganda leader, stated that it was "well known"; some suggested that although it was common in Bunyoro it was not practised in Buganda, but this point seems not to have been finally established.

The existence of this practice is of great significance and begins to expose a flaw in the whole of Lambkin's argument. Surely this is no new custom—and it was evidently widespread—that had grown up in twelve years, nor even in fifty, and its existence throws doubt on the views both of Lambkin and of his missionary associates. It argues long familiarity with some treponemal disease.

Before discussing this, an allied consideration deserves attention. Neither Cook, Lambkin, nor Keane suggests that there was any social stigma attached to syphilis in Uganda, either in the "congenital" form known as "munyo" or in the adult "kabotongo". Muwazi, Trowell & Davies noted that there was no stigma attached to "kabotongo", that it was probably regarded as a non-venereal disease, and that only in recent years were modern educated people in Buganda realizing that there was any social opprobrium attached to syphilis. Keane hints at the same lack of stigma, and even at the lack of any eagerness for treatment; unless sent for treatment by the chiefs, patients suffered quietly at home, not concealing the disease. It is difficult to reconcile this with the missionary viewpoint, adopted by Lambkin, that the emancipation of the women was the cause of the explosive outbreak. The Baganda were much too intelligent not to realize the connexion between venereally acquired syphilis and promiscuous intercourse, even if it were not forcibly pointed out to them by the missionaries themselves. Even if one doubts that there was such a lack of immorality and promiscuous intercourse
as Roscoe suggests, a rigidity in sexual morality is usually accompanied by a censorious attitude to the victims of venereal disease, and by an accompanying sense of shame on the part of some of the victims. The absence of such a stigma argues long familiarity with a disease frequently, if not usually, acquired non-venerally. M. Rosental’s description of syphilis in 1924 among the Kalmucks and Kirghiz in Astrakhan (Russia), quoted by Groen, is relevant here. He says “the inhabitants of the steppes who are in a low state of civilization have no comprehension of the dangers of infection. Only in a few cases is the infection transmitted by sexual intercourse, usually syphilis is innate or is transmitted by mouth based on the peculiar centuries-old habits of the Kalmucks”. He goes on to describe their habits of passing pipes from mouth to mouth, and indeed such methods of spread, for example, by the smoking of communal pipes of which the mouthpieces were infected, were recorded in Uganda by Cook.

There is therefore much to suggest that syphilis was not a new disease introduced by the Arabs to the inhabitants of Uganda, but one of long standing, often, if not usually, acquired extra-venerally. The custom of infantile vaccination argues long familiarity, as does the fact that the people realized that, if inoculated in infancy, the adult was spared entirely, or the disease was less severe (cf. Fiji), for the practice must have taken many years to become an established fact, acceptable to the “medical profession” indigenous to the country. These “doctors” should not be regarded as crude witch-doctors but as medical men who, in some respects at least, were remarkably advanced. This is shown for example, by Felkin’s description of an operation for Caesarian section which he witnessed by invitation in Bunyoro. This was only a few years after Lister had moved to London. It is perhaps of significance that Felkin saw this operation in Bunyoro, where, according to Lambkin’s informants, infantile syphilitic vaccination was most widely practised and accepted.

It seems therefore impossible to accept, in the light of the evidence available, that syphilis was a new disease attacking a virgin population in 1897-1909. We have the clear testimony of Wilson & Felkin that it was common in the late 1870’s, and the less clear evidence of Speke in the 1860’s. Lambkin admits that it had been present for about 60 years before his time and had “prevailed in a mild form”, but how could this be, if it was a true syphilis attacking a virgin population? Would not an explosive outbreak, as in Europe in 1493 onwards, have come much more suddenly? Was there, in fact, an explosive outbreak, an epidemic, of syphilis? No doubt the troublesome years from 1885 onwards, with the religious wars and the forced evacuations and migrations of whole communities, were responsible for some increase in syphilis in a population among whom it was previously common. But by 1897 the country was becoming pacified, and the last serious fighting was the
mutiny of the Sudanese troops in late 1897; from 1899 peace prevailed generally in Buganda and the country settled down to consolidation and peaceful production. Had the epidemic occurred in the years 1885-1895 the theory would seem more credible; but it occurred in the years during which the country was at peace, while the population were becoming increasingly alarmed at the spread of sleeping sickness and were beginning to seek medical help. The year 1897 has been frequently mentioned, and it is perhaps significant that this was the year in which that forceful personality, Albert Cook, arrived. He had been busy treating patients during his long march from the coast, and on arrival he flung himself into medical work among the indigenous peoples. His work was temporarily interrupted by the Sudanese revolt, but he was soon back in his famous hospital at Mengo. He quickly became aware of the ravages of disease among the native people and soon recognized the importance of syphilis. No doubt, as so often happens, the more he looked the more he found, and he became convinced that syphilis was a major problem, as indeed he proved it to be. He also became convinced that an epidemic of syphilis was in progress, but of this there is little real evidence. It is not supported by his own figures; it is not in consonance with other evidence; and it seems on many grounds inherently improbable. It is no disrespect to the memory of this fine and able physician to suggest that he was mistaken, that there was no real epidemic of syphilis, and that in so far as there was an "epidemic" he himself was the origin in that he created, out of the partial knowledge of the previous years, a full knowledge of the ravages of the disease and by his energy and enthusiasm convinced others that it was an "epidemic spread" in an "explosive outbreak". His great personality makes this not improbable.

If the arguments outlined above are accepted it is possible to see the history of syphilis in Uganda in a different light from that usually accepted and to reconcile the discrepancies previously alluded to. It seems permissible to postulate that the Baganda had been familiar for a very long time with a disease resembling in many ways venereal syphilis but usually spread extravenerally, and without any stigma attached to it among a people whose moral standards were perhaps somewhat severe, if not quite so severe as some writers accept. It was frequently seen in childhood, and it had been noted that those who escaped attack until adult life were more severely affected. Thus there were advantages in getting the disease when young and on this was based a practice of infantile inoculation with the disease. Since this was considered worth while, it argues that the disease was very common. What disease could fit this picture? Yaws is a possibility, as in some areas protective infantile inoculation is practised.11 But we have evidence that yaws was not common in Buganda, and this clear statement is supported by other evidence. The likelihood is that some form of endemic syphilis had prevailed for many years.
Endemic and venereal syphilis

We know much more about endemic syphilis than was known in Lambkin’s time, and many accounts of it have been written. We know that forms of endemic syphilis occur in Rhodesia, and in Bechuanaland, under the name of “njovera”, and in Sindebele for “dichuchwa”. There is no doubt that it exists or has existed in different parts under a variety of names. The whole question of endemic syphilis has been exhaustively reviewed by Groen. Some of the remarks made by Willcox seem peculiarly apposite. Speaking of the Karanga people he says

“although they have no other word for syphilis and often use the same, there is some indication that it is appreciated that the juvenile njovera is a different condition which is acquired in a different manner from that of the syphilis of the towns; the latter even being stated to have arisen only since the coming of the white man. The word ‘njovela’ used in Sindebele for syphilis, does not appear to have the double meaning and is employed indiscriminately for both conditions.”

In describing the clinical picture Willcox says

“the disease is characterised typically by the secondary lesions of genital or ano-genital condylomata and split papules at the angles of the mouth. The regional glands adjacent to the condylomata enlarge to a syphilitic pattern. Mucous patches may also exist on the lips and buccal mucous membranes and there may be sore throat and laryngitis. Large papules or actual condylomata in the axilla are also not rare. Those on the body are not so common and are of a raised yellow crusted or framboesiform and of a scanty scattered nature.”

The secondary lesions were frequently accompanied by osteoscopic pains. The infection was often acquired in early childhood and extragenitally. Willcox considered it a form of extra-venereal syphilis, spread by close contact and possibly by flies, resembling bejel. The later lesions were more yaws-like, palatal ulcerations but not goundou were seen, but periostitis, ganglion formation, plantar lesions, and gummatous ulceration were all encountered. Among the neighbouring Batonga people the same disease occurred, known as “siakwelele”. Although the disease is on the decline, most older persons questioned admitted to a childhood attack, and the serological positivity rate was very high—up to 75% in some areas.

The review by Groen goes in detail into numerous reports of endemic syphilis. Only a few points pertinent to Uganda will be mentioned. The frequency with which the disease occurs without a primary lesion is stressed, and scars can rarely be located. But where primaries do develop they are often phagedaenic. The secondary lesions are usually severe with much bone and joint pain. The severity of the skin lesions is emphasized and such lesions are said to be more frequent, more florid, and more severe in hot countries, as moist condylomata, severe pustular (especially in Negroes), impetiginous, ichthyoid and rupeial forms. In under-
nourished peoples the severe pustular forms are more common. Mixed secondary and tertiary lesions are often combined, such as ruipial lesions and marked mucous changes, and there is an unusually early appearance of frequent severe tertiary lesions. The skin changes are the most prominent throughout, but deep ulcerations of the face, ears, lips, and eyelids are frequent, with great bone destruction in the face, nose, and limb bones. Visceral and neurosyphilitic lesions are rare, particularly tabes dorsalis. The majority of cases are affected extragenitally. In some areas the disease is severe, in others mild. Spontaneous healing without therapy is well known, and in other cases the lesions may become stationary, cause little trouble, and do not affect the general health. The response to therapy has been variable, but small dosages of mercurials have been effective in the past. The lesions now respond well to penicillin.25

If the history of syphilis in Uganda is looked at on the basis that endemic syphilis is of long standing in the lacustrine areas, then most of the difficulties melt away and the discrepancies in the various accounts can be reconciled. The descriptions of Groen and of Willcox bear a close resemblance to the accounts given by Cook, Lambkin, and Keane. Endemic syphilis was of long standing in Buganda, widely disseminated, usually acquired extravenereally and prevailing widely in a mild form. It was not thought of as a venereal disease and no social stigma was attached to it. The Arabs came into the country in the years about 1850, followed by the Europeans and others. At some time in this period true venereal syphilis was introduced. Its spread may have been limited by the social customs of the day, but it slowly began to replace the endemic syphilis. It would seem that, when Cook, Lambkin, and Keane were working in Uganda, true venereal syphilis was replacing endemic syphilis and the differences between the conditions were not recognized, since all were probably influenced by the unitary views of Hutchinson.15 That venereal disease was spreading is suggested by the remarks of Moffat 19 on gonorrhoea previously quoted, for he found gonorrhoea to be common but urethral strictures to be very rare, “perhaps on account of the great size of the parts”. Within a few years, and certainly by the nineteen-twenties, this was entirely changed and urethral stricture was a condition of great frequency and importance.8 The increasing frequency of gonorrhoea may well have convinced the early workers that syphilis was spreading in a similar way, which was no doubt true, but the extent was almost certainly exaggerated. That treatment with small dosages of mercurials was so successful supports this interpretation of the wider prevalence of endemic syphilis in those days. We may therefore conclude that the early workers saw mostly endemic syphilis with some increasing admixture of true venereal syphilis.

But with treatment and with advancing civilization, endemic syphilis in the clean and clothed Baganda people declined rapidly, to be replaced
behind a legacy of the chronically affected and a high positive serological rate, which has been such a puzzle to subsequent investigators. For despite the views of the earlier workers and the high index of serological positivity in antenatal cases, congenital syphilis is uncommon and has been so since the early nineteen-twenties. The high Wassermann reaction rate powerfully influenced Webb & Halliday in their studies. It would appear that they were dealing with a considerable residue of endemic syphilis cases and a considerable number of cases of true venereal syphilis. By the nineteen-thirties the late sequelae of true venereal syphilis—cardiovascular and neurosyphilitic lesions—were occurring in considerable numbers, as Loewenthal noted. By then it would seem that endemic syphilis had died out, but small pockets may have persisted in rural areas. Still, there would be a considerable number of cases with serologically positive reactions as sequelae of an attack of endemic syphilis in childhood. It is of some interest that when the question of high incidence of serological positive reactions in the mothers and the infrequency of congenital syphilis was discussed in Uganda, Scott Brown suggested that these features could best be explained on the assumption that the mothers themselves were congenital syphilitics. This acute suggestion did not find favour at the time, but if it is altered to suggest that the mothers were old victims of endemic syphilis it is a very probable explanation of the discrepancy.

If the theory of the history of syphilis in Uganda is accepted, then the present position becomes clearer. The discrepancies in the previous accounts can be explained, the slur placed on, and accepted by, the Christian missions and the faith they propagate can be removed, and the blame attached to the women of Buganda can be diminished and be more widely shared. But a paradox arises in that with the "debunking" of the previously accepted views of the history of syphilis in Uganda and a diminution of the importance previously attached to it, the menace of syphilis in Uganda becomes greater. The older belief in the enormous frequency of congenital syphilis is seen to have been based on a misapprehension but the menace of true congenital syphilis now becomes greater. For in Uganda today endemic syphilis seems to have disappeared, its immunizing powers (whatever their extent or efficiency) have gone too, and we are left with a high incidence of true venereal syphilis and with all its grave complications and sequelae, with nervous and cardiac complications of great clinical frequency, and importance. The antisyphilis campaigns of the past have altered, but have not solved, the problem, which is now more serious than in the past.

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RÉSUMÉ

L'auteur examine les travaux antérieurs sur la syphilis en Ouganda et plus spécialement sur la "flambée" de syphilis qui se serait produite vers le début du vingtième siècle — selon des informations qu'il considère comme erronées.

L'auteur émet l'opinion qu'une forme endémique de syphilis aurait été connue depuis longtemps dans l'Ouganda méridional. Les populations auraient été familiarisées avec la maladie qui se propageait sans contacts vénériens dans la plupart des cas; on pratiquait une inoculation infantile prophylactique contre cette maladie. Des cliniciens européens ont commencé à observer le pays à une époque où la syphilis endémique était fort répandue encore, mais où la syphilis vénérienne véritable commençait à la remplacer. Ce processus est maintenant achevé, la syphilis endémique a disparu et la syphilis vénérienne véritable est maintenant très fréquente.

L'auteur soutient que cette conception de l'histoire de la syphilis en Ouganda explique les observations d'autres auteurs et aplani le discorde qu'on relevait entre les différentes études. Cette conception explique également un fait qui a suscité parfois de nombreux commentaires: le taux élevé de séropositivité des divers groupes de population.

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HISTORY OF SYPHILIS IN UGANDA