NOTICE OF ORDINARY MEETING

The fifth general meeting of the Society for 1976 will be held in the Museum Education Building, North Terrace, Adelaide at

8.00 pm Monday 26 July, 1976

AGENDA

1. Apologies.

2. Minutes of meeting held Monday 28 June, 1976 to be confirmed. Copies of these minutes are attached.

3. New members.
   The following new members have been elected to the Society.
   Ordinary membership:

   Mr. Eric Malcolm VIVIAN
   Mr. Robin RADFORD
   Mr. Darryl Kenneth BULLEN
   Mr. Harold Glen HOWARD
   Mrs. Barbara Thelma TAYLOR
   Mr. Peter STRAWHAM
   Mr. Denis COPLE
   Mr. Donald FINLAYSON

   34 Edgecombe Terrace, Rosslyn Park
   Flinders Medical Centre, Bedford Park
   P.O. Box 251, Unley
   232 Brighton Road, Somerton Park
   12 Downer Street, Plympton Park
   12 Beverley Road, Aldgate
   16 Berkeley Road, o'Halloran Hill
   58 Birman Crescent, Flagstaff Hill

   Associate membership:

   Mrs. Robin RADFORD
   Mr. Mark RADFORD
   Mrs. Judy STRAWHAM

   1A East Terrace, Blackwood
   1A East Terrace, Blackwood
   12 Beverley Road, Aldgate

   Papers and journals from other Societies will be tabled at the meeting.

5. Films.
   The Council is endeavouring to obtain the following films from the 'People of the Australian Western Desert' series by Ian Dunlop for screening at the meeting.
   Part 3 Sacred boards and an ancestral site
   Part 7 Spear-throwing, including stone flaking and gum preparation
   Part 9 Spinning hair string, getting water from a well and binding girls' hair
4. Subscriptions are as follows:

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The following is a reprint of an address given to the Society by the late Dr. C.J. Hackett at the Annual General Meeting, Monday 27 November, 1967.

'SOME ASPECTS OF TREPONEMATOSES IN PAST POPULATIONS'

Introduction

This title is rather too general to be dealt with adequately at one time. My experience is restricted to certain diseases - to a group known as trepomatores, which are due to infections with spiral organisms that under the microscope look exactly alike.

It was in 1927 when Draper Campbell invited me to join an Adelaide University Board for Anthropological Research visit to Central Australia that in Alice Springs I first became interested in bone disease in the Aborigines. We saw a woman named Randa with, perhaps, the most advance boomerang legs that have ever occurred. Not only were her legs markedly bowed forward but her thighs and forearms were similarly deformed. Sir John Cleland was also on this trip.

I cannot let this opportunity pass without paying tribute to these two men, Sir John Cleland and Draper Campbell, for the parts they have played in the reawakening and development of anthropology in Adelaide following the initial stimulus of Wood-Jones.

The next incident for me occurred in the spinifex and desert sheoak country south of the Mann and Musgrave Ranges in 1933 where Norman Tindale and I were on another Adelaide University anthropological trip. One morning on the return journey to Ernabella, the camels, despite tight hobbling, wandered further than usual and it was midday before the Aboriginal camel-men brought them back. Among a small party of Aborigines who had found us during this delay was a woman with a sick child. This little girl had boomerang legs and extensive scabbing and scarring of her face, chest and abdomen. It seemed possible that this association might provide the basis for study into the cause of boomerang legs.

This study was made possible the next year by a grant from the Sheridan Research Fund of the Adelaide University. Some radiographs were taken in
Alice Springs, after which Darwin, Wave Hill, Victoria River Downs and Bathurst Island were visited. In Alice Springs among the folk radiographed were some with enlarged bones with holes in them. The bones of Randa were also found. In a monograph published in 1936 it was stated that boomerang leg and these other changes were due to yaws. Subsequent studies make it very unlikely that yaws causes boomerang leg.

In 1938 MacKay published a paper on some 'pathological changes in Australian Aboriginal bones' based on a study of specimens, mostly from N.W. Victoria, in the Australian Institute of Anatomy in Canberra. He did not concern himself with boomerang leg. He excluded yaws as a cause of the other changes because he could find no evidence that it caused bone lesions. He concluded that 'The ultimate diagnosis must therefore ... rest between venereal syphilis, an endemic type of non-venereal syphilis, or some indigenous disease of which we have no medical knowledge, which also produces these striking pathological lesions in bone'.

During 1937-1939, with a grant from the Medical Research Council of Great Britain, a radiographic study of the bone lesions of yaws was made in Uganda, which left no doubt of their frequent occurrence in a population of high infection rate of 80% or more. About 10% of this population had active skin or bone yaws. As has already been said, no indication was found in over 400 patients studied that yaws caused boomerang legs. The publication of this study was delayed by the war until 1951.

In 1942 Root had published a very useful paper on the bone lesions of endemic syphilis in Deir ez Zor in Syria. It appears that the bone lesions of venereal syphilis, endemic syphilis and yaws in adults do not greatly differ.

The Treponematoses.

We must now return to the treponematoses. Some folk would at this stage produce a large table of the differences between these diseases to distinguish each from the others. Equally as many would deny them all. Two or three points will be mentioned that can be readily recognised. Venereal syphilis is transmitted among adults as a venereal disease and the other two among children as simple contagious diseases. Venereal syphilis may occur anywhere where there are susceptible adults, especially in large towns and cities, but yaws and endemic syphilis occur only in rural populations, the former in humid warm areas and savanna country and the latter in hot arid desert-like areas.

The organism, treponemes, found in all these, under the microscope look alike, and cannot be cultivated in the laboratory, but they will all infect rabbits and hamsters (Turner and Hollander 1957). From these studies, it appears that the treponemes of yaws and venereal syphilis have opposite characters while those of endemic syphilis have intermediate ones.

I have said there are three different diseases and shall speak of them as such. There are some folk, however, who feel strongly that there is only one species of treponeme and that it causes only one disease which may appear differently under different environmental conditions and that the disease manifestations may change and revert in a century or so, or in a year. Hudson (1962) is the present doyen of this point of view and his written much about it.
A fourth human treponematosis, pinta, of the Americas from Mexico to Brazil must now be mentioned. It causes only skin colour changes, while yaws and endemic syphilis cause changes in the skin and bones, and venereal syphilis in the heart, arteries and brain, in addition to the skin and bone. Its organisms will not, however, infect animals.

**History of Syphilis**

Syphilis was first recognised by that name about 1515 and it was said not to have occurred in Europe, or in fact anywhere else, before 1495. One should not forget that the complete disease picture of syphilis was not known until the 19th century. Yaws was not fully recognised until about the end of the last century, and endemic syphilis and pinta until the 1920’s.

The history of venereal syphilis is confused and controversial. The usual story in the history books of medicine is that Columbus and his men brought venereal syphilis to Europe from Hispaniola in 1493 and that Spanish mercenaries fighting with the Neapolitans and/or the French forces of Charles VIII besieging Naples in 1495 were responsible for the initial outbreak of the disease. Finally, Charles returned over the Alps and the disbanding of his army spread the disease throughout Europe. A recent account has named the man who first brought syphilis to Scotland – from Paris of course.

Nearly all the above statements have been challenged from historical records. Accidental errors have crept in and intentional falsifications have been suspected. It seems that an American origin was not proposed before 1520 and it has been said that this was promoted by the Fugger family of bankers who had the monopoly for the importation from the Caribbean islands of guaiac bark, which was one of the earliest remedies for syphilis. The high death rate in this first outbreak is quite unlike venereal syphilis in any other country or time.

A possible explanation of the apparent outbreak is that the mode of life of that period was particularly favourable for the venereal transmission of disease. The occurrence of many patients made it possible for the doctors to observe the course of the disease in individual patients so that symptoms previously thought to be separate diseases were recognised as stages or aspects of one disease. At the same time an epidemic of a real killer, such as plague or typhus, may have provide the background of high mortality.

In Persia, India, China, and Japan, there is the same story; syphilis was brought by European travellers and explorers early in the 1500’s; descriptions of it are to be found in early written records.

**Geographical distribution of the treponematoses**

After 400 years of fruitless textual controversy, it seemed that with the new knowledge from this century it was time to attempt a new approach to the historical problem of syphilis. For this purpose the geographical distributions of the four treponematoses were taken. It was hoped that this might at least suggest where further work was needed.

A series of maps will be found in a publication of 1963 on the origin of the treponematoses. Adequate and accurate published data are unfortunately not available, hence the first map is based on corrections of published data, from
information gained in many discussions and from visits to a number of countries.

The distributions needed first are of disease in indigenous populations in their natural stable state and in separate immigrant populations. Thus allowance had to be made for recent internal population movements and for the effects of modern chemotherapy. Some of you may find errors in some of my assumptions due to my inadequate knowledge. Of particular interest, however, is the general sequence of suggested events rather than the proposed dates.

The first map attempts to show the distribution of the treponematoses about the year 1900. The most important points are; pinta only in the Americas, yaws in the tropics and endemic syphilis in arid areas north and south of the tropics, with venereal syphilis in many other places. Minor deviations from these broad statements will not be touched upon now.

The next map (2) is an attempt to indicate possible distributions at Columbus's time and are derived from generalizations from the first map.

A map of the distributions of climates would show the agreement of that of endemic syphilis with hot arid regions.

The third map is based upon the peripheral (Western) distribution of pinta which suggests that it might have been the first treponematosis to arise in man - and to have spread throughout the world by, it is proposed, about 15,000 B.C. It would then have been isolated in the Americas by the flooding of the Bering Strait with the melting of the polar ice caps - although the present sea levels may not have been reached until about 4,000 B.C. Alas, there is no known eastern accompaniment of the marsupials in Australia and Tasmania by pinta.

The next map (4) is based upon the assumption that somewhere in the Afro-Asian land mass certain mutants appeared in the treponemes that gave rise to yaws which might have extended throughout the world it could reach by about 10,000 B.C.

Then about 7,000 B.C. (map 5) climatic changes due to the retreat of the last glaciation gave rise to the present extensive intra-continental arid zones which favoured treponeme mutants leading to endemic syphilis (Lamb 1963).

It is difficult to see how otherwise endemic syphilis north of the equator could have reached southern Africa - and probably central Australia. It will be recalled that animal experimental infections suggest that endemic syphilis probably developed from yaws and in turn gave rise to venereal syphilis.

The next change, (map 6), it is suggested, came with the development of urban populations in South West Asia or the Eastern Mediterranean, where the standard of living and clothing perhaps reduced the possibilities of treponeme transmission to the close venereal contact of adults -and suitable mutants were selected. This might have been about 3,000 B.C.

The last step ( map 7), was the carriage of venereal syphilis throughout much of Europe by the Romans up to the early centuries of the Christian era - and to susceptible populations throughout the world by Europeans after the century of great geographical discovery of Columbus and Vasco da Gama. Yaws was also carried about in African slaves.
Of the irregularities in the distributions in the first and last maps, the most noteworthy is (or was until a few years ago) the presence of endemic syphilis in Bosnia which probably came there from Anatolia with the Islamic invasion of the 14th century. This was before Columbus's return!

From all this two clear indications for further study arise. One is for the collection of treponemes from areas where mutation might have been important - and to store these in the frozen state in a suitable place such as the International Treponeme Bank in Baltimore. When the pathological treponemes can be cultivated and their antigenic characters studied, much real evidence might be obtained.

The other is to improve the diagnosis of disease in dry bones by the development of diagnostic criteria - to replace authoritarianism and guesswork. Then the occurrence of diseased bones in burials in different parts of the world may provide relevant information.

It is this aspect that has interested me for the past few years.

Lesions in old bones

There have been many publications on the lesions in old bones. Some are of little value and the diagnoses and/or dating of others have often been challenged. In none have the diagnostic criteria used been defined.

A French worker, Pales, in 1930 in a chapter on prehistoric syphilis said no one with experience and qualifications will make a diagnosis of syphilis in a dry bone without many reservations, while folk interest in the implication of a bone being syphilitic will make the most uncrritical statements. In 1965 Pales told me he thought that the position had not changed since 1930.

Williams, in the early 1930's, visited all the medical museums in Europe with more than five syphilitic skulls - preparatory to studying all the old bones in Europe claimed to be syphilitic - and also many of those in the United States of America. He did not define his diagnostic criteria and last year and unsuccessful attempt was made to trace his notes through his old University in Buffalo, U.S.A.

His conclusions were that no pre-Columbian bones with syphilitic lesions had been found in Europe and only a few had been found in America. This interesting aspect, however, can not here be followed further except to say that the first paper describing syphilitic lesions in pre-Columbian bones in U.S.A. was by Joseph Jones in about 1876. In the past, accuracy of dating pre-Columbian bones has added to the problems of assessing their medical historical value.

Diagnostic criteria

During the past two years, while working from the Department of Medical Anatomy (Dr. H.A. Sisson) of the Institute of Orthopaedics, London, all the medical museums in London have been visited and bones with lesions likely to be due to infectious disease such as venereal syphilis, pyogenic osteomyelitis, tuberculosis etc., were studied - and photographed - then the museum notes were looked at. The first two categories, venereal syphilis and osteomyelitis, were the most numerous and in the first quarter of this century were frequent in hospital patients. Over 400 bones were studied by the use of 1400 photographs of each specimen after sorting into individual bones, skull, tibia and femur, were divided into those with similar changes and then the label 8.
diagnoses were seen - no attempt was made to diagnose the changes present. The changes in the long bones were composed of expansion and destruction.

Of 39 tibiae with expansion along (and no destruction), 34 were labelled syphilis. Of 18 with extensive expansion and destruction none was labelled syphilis but all were labelled osteomyelitis (almost certainly of pyogenic origin). Of 8 with local expansions and sinuses, 5 were labelled syphilis. The other tibiae of the 140 studied do not detract from the value of these findings since they included fractures, specimens without museum diagnoses and small groups of 2.5 specimens with the same diagnosis.

Of 171 skulls and calvaria, 66 were set aside because of absence of notes and the few specimens with the same diagnosis, of which 9 with osteomyelitis was the largest, and 12 labelled syphilis in which the changes did not resemble those in any remaining 105 labelled syphilis. The changes in this large group appeared to form a series starting with clusters of pits in which the pits coalesced to leave cavities opening on the surface which sometimes extended through the whole thickness of the bone.

These superficial cavities might themselves join with adjacent ones to form a characteristic picture. The irregular framework of original bone remaining between the cavities then becomes thickened and rounded and finally appeared to fill the intervening gaps to produce a nodular surface. This with time often flattens. All the changes can be recognised in the descriptions of the mid-19th century such as ulceration, caries sicca and stellate scars, but previously their evolution was difficult to understand.

Large depressions might remain - and on top of all these late (healed) changes the original clustered pits might reappear as a relapse.

On these syphilitic skulls often appear areas of dead bone - several inches in diameter. These probably result from the ulceration of syphilitic lesions through the skin and pyogenic infection entering through the sinus thus produced. One should not forget that many of the skulls and long bones referred to entered museums during the 1800's and that the many specimens in the Hunterian Collection were indexed before 1797; they came from the poorer part of city populations.

That briefly summarises the position of the study to date. More bones must be seen to ensure that the changes outlined above do not occur in other diseases. It should be stresses that label diagnoses have been spoken of. These are poor things to a statistician. Of the 105 skulls only 6 had some authenticity of diagnosis - depending upon information outside the bone itself; while only 1 of over 100 tibiae had this.

Observations in the South Australian Museum, through the kindness of Dr. W.P. Crowcroft, and in Melbourne (Professor L.J. Ray, Department of Anatomy, University of Melbourne) and Perth (Western Australian Museum) have covered about 1500 skulls and several times as many long bones. Practically all the changes seen in these aboriginal bones fall into the types of lesion labelled syphilis in European skulls and long bones - except for the large areas of dead bone in the London syphilitic skulls.

In the London long bones is a group of changes labelled osteomyelitis and almost certainly due to pyogenic infection mostly in childhood. These are
completely absent in the Aboriginal bones seen. When medical teams first went into the previously unadministered parts of West New Guinea in the 1950's, the only bone lesions found were those due to yaws; none was due to pyogenic infection.

It is very improbable that the Australian Aborigines suffered from venereal syphilis before European discovery, and for as long after as they continued to contract one of the other treponematoses in childhood - which they still do in some out-of-the-way rural places. Venereal syphilis is probably not prevalent in Aborigines at the present time in many parts of Australia.

The findings of the brief bone survey in Adelaide and Melbourne indicate that the Aborigines over a wide area in the past suffered from a treponematoses, but not from pyogenic osteomyelitis.

More bones need to be studied in Great Britain and perhaps Europe to provide adequate material upon which to base criteria. The observations of the past few weeks have, however, indicated that the London studies are in the right direction as far as the Australian bones are concerned. Whether they will be of the similar value in the diagnosis of pathological lesions in pre-Columbian bones in America may be learnt in Washington in a few months time.

If the hoped for diagnostic criteria do not appear from the further London studies, the direction of the studies will have to be changed to the recognition of patterns of bone changes in one population for comparison with those in another.

There are two further aspects to which I would like to refer.

One is a curious tunnelling change which occurs in bones buried for more than 300-400 years and which in time may lead to their complete destruction. Reference is, of course, not made to the chemical changes that occur in buried bones, nor to the importance of soil composition and water table levels in the deterioration of bones. Wedd (1864) and Roux (1887) have described these changes and had attributed them to a fungus. These changes are obviously post-mortem and extend into the cortex form the surfaces. Their particular interest is that they could greatly reduce the value in old bones of diagnostic microscopical structures that might be related to a particular disease. That the microscopical structures of diseased dry bones might have diagnostic value is doubted by some and denied by other bone pathologists.

The other point is that in Britain bones with changes like those of syphilis and osteomyelitis are said not to be seen until the later Middle Ages. Finally in a large venereal disease clinic in a large African city where the prevalence of venereal syphilis was high, no patient with syphilitic bone lesions was seen in ten years!

It is hoped that this very brief summary has provided interest in some of the problems of some diseases in some past populations.
REFERENCES


Rost, G.S. (1942): Roentgen manifestations of bejel ("endemic syphilis") as observed in the Euphrates River Valley. Radiology, 38, 320.


