Theses Digitisation:

https://www.gla.ac.uk/myglasgow/research/enlighten/theses/digitisation/

This is a digitised version of the original print thesis.

Copyright and moral rights for this work are retained by the author
A copy can be downloaded for personal non-commercial research or study, without prior permission or charge
This work cannot be reproduced or quoted extensively from without first obtaining permission in writing from the author
The content must not be changed in any way or sold commercially in any format or medium without the formal permission of the author
When referring to this work, full bibliographic details including the author, title, awarding institution and date of the thesis must be given
A CULTURAL, PHYSICAL AND PATHOLOGICAL STUDY

OF THE

WESTERN DESERT ABORIGINES OF AUSTRALIA

A thesis presented for the Degree of Doctor of Medicine, University of Glasgow.

By John Morrison, M.B., Ch.B.
DECLARATION

I declare that the work presented in this thesis has been performed by me; that the thesis has been written entirely by my own efforts, and that all references to any other person's studies has been fully acknowledged in both the text and the references.

John Morrison, M.B., Ch.B.

1st Sept. 1973
INDEX
GENERAL INTRODUCTION

PART I

THE CULTURAL TRADITIONS OF THE WESTERN DESERT ABORIGINES

Introduction

The Environment of the Western Desert Aborigines

Conception and Birth

Infanticide, Geriatricide and Cannibalism

Diet and Nutrition

Initiation Rites

Marriage and Kinship

Medicine and Magic

Social Behaviour and Punishment

Art, Craft and Language

Death and its Association

THE INFLUENCE OF WESTERN CIVILISATION ON THE CULTURAL TRADITIONS OF THE WESTERN DESERT ABORIGINE

MEDICAL PROBLEMS DUE TO THE CULTURAL TRADITIONS OF THE WESTERN DESERT ABORIGINES

PART II

THE PHYSICAL CHARACTERISTICS OF THE WESTERN DESERT ABORIGINES

Introduction
<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Physical Features of the Western Desert Aborigines</td>
<td>122</td>
</tr>
<tr>
<td>The Blood Cells of the Western Desert Aborigines</td>
<td>150</td>
</tr>
<tr>
<td>The Blood Groups of the Western Desert Aborigines</td>
<td>164</td>
</tr>
<tr>
<td>Other Genetically Inherited Characteristics of the Western Desert Aborigine</td>
<td>174</td>
</tr>
<tr>
<td>The Biochemistry of the Western Desert Aborigines</td>
<td>179</td>
</tr>
<tr>
<td>Physiological Observations in the Western Desert Aborigines</td>
<td>186</td>
</tr>
<tr>
<td>DISCUSSION OF THE PHYSICAL CHARACTERISTICS OF THE WESTERN DESERT ABORIGINES</td>
<td>192 - 228</td>
</tr>
<tr>
<td>Comparison of Western Desert and Other Aboriginal Groups</td>
<td>194</td>
</tr>
<tr>
<td>Comparison of Western Desert Aborigines and the White Population of Australia</td>
<td>219</td>
</tr>
</tbody>
</table>

PART III

THE PATHOLOGY OF THE WESTERN DESERT ABORIGINES 229 - 413

<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Introduction</td>
<td>230</td>
</tr>
<tr>
<td>Congenital Abnormalities</td>
<td>233</td>
</tr>
<tr>
<td>Traumatic Disease</td>
<td>237</td>
</tr>
<tr>
<td>Infectious Disease</td>
<td>243</td>
</tr>
<tr>
<td>Diseases of the Cardio-Vascular System</td>
<td>326</td>
</tr>
<tr>
<td>Diseases of the Respiratory System</td>
<td>330</td>
</tr>
<tr>
<td>Diseases of the Digestive System</td>
<td>337</td>
</tr>
<tr>
<td>Diseases of the Urinary System</td>
<td>348</td>
</tr>
<tr>
<td>Diseases of the Blood and R.E. System</td>
<td>352</td>
</tr>
<tr>
<td>Diseases of the Male Genital System</td>
<td>357</td>
</tr>
<tr>
<td>Diseases of the Female Genital System</td>
<td>361</td>
</tr>
<tr>
<td>Diseases of the Endocrine System</td>
<td>369</td>
</tr>
<tr>
<td>Topic</td>
<td>Page</td>
</tr>
<tr>
<td>----------------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>Diseases of Metabolism</td>
<td>371</td>
</tr>
<tr>
<td>Diseases due to Deficiencies</td>
<td>374</td>
</tr>
<tr>
<td>Diseases of Allergy and Collagen</td>
<td>378</td>
</tr>
<tr>
<td>Diseases due to Physical and Chemical Agents</td>
<td>380</td>
</tr>
<tr>
<td>Diseases of the Skin</td>
<td>384</td>
</tr>
<tr>
<td>Diseases of the Locomotive System</td>
<td>390</td>
</tr>
<tr>
<td>Diseases of the Nervous System</td>
<td>395</td>
</tr>
<tr>
<td>Diseases of the Eye</td>
<td>403</td>
</tr>
<tr>
<td>Diseases of the Ear</td>
<td>409</td>
</tr>
</tbody>
</table>

**DISCUSSION OF THE PATHOLOGY OF THE WESTERN DESERT ABORIGINALS**

The Pathology of the Western Desert Aborigines in Comparison with other Aboriginal Groups 417

The Pathology of the Western Desert Aborigines in Comparison with the White Australian Population 425

**SUMMARY AND CONCLUSIONS** 440

**ACKNOWLEDGEMENTS** 447

**BIBLIOGRAPHY** 448

**APPENDIX** 488

A. Personal Publications concerning Aborigines 489

B. List of Publications concerning Aborigines acknowledging Authors Assistance 493
<table>
<thead>
<tr>
<th></th>
<th>MAP 1</th>
<th>Western Australia - Western Desert Area</th>
<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>MAP 2</td>
<td>Area of Medical Practice in Western Desert</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>MAP 3</td>
<td>Distribution of Western Desert Aborigines</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>MAP 4</td>
<td>Western Australia - Population</td>
<td>8</td>
</tr>
<tr>
<td>5</td>
<td>MAP 5</td>
<td>Distribution of White Population - Western Desert Area</td>
<td>9</td>
</tr>
<tr>
<td>6</td>
<td>MAP 6</td>
<td>Western Australia - Vegetation</td>
<td>10</td>
</tr>
<tr>
<td>7</td>
<td>PLATE 1</td>
<td>Western Desert Cunringas</td>
<td>65</td>
</tr>
<tr>
<td>8</td>
<td>PLATE 2</td>
<td>Western Desert Message Sticks</td>
<td>66</td>
</tr>
<tr>
<td>9</td>
<td>PLATE 3</td>
<td>Western Desert Bullroarers</td>
<td>67</td>
</tr>
<tr>
<td>10</td>
<td>PLATE 4</td>
<td>Western Desert Weapons</td>
<td>68</td>
</tr>
<tr>
<td>11</td>
<td>PLATE 5</td>
<td>Western Desert &quot;Humpy&quot;</td>
<td>76</td>
</tr>
<tr>
<td>12</td>
<td>MAP 7</td>
<td>Exploration of Western Australia</td>
<td>86</td>
</tr>
<tr>
<td>13</td>
<td>PLATE 6</td>
<td>Fair Hair in Western Desert Aborigines</td>
<td>195</td>
</tr>
</tbody>
</table>
1. Adult Males - Mean Height in Relation to Age  & 137
2. Adult Males - Mean Weight in Relation to Age  & 138
3. Adult Males - Body Proportions  & 139
4. Adult Males - Digital Formulae  & 140
5. Dermatoglyphic, Darwinian Tubercle, and Ear Lobe Patterns  & 141
6. Adult Females - Mean Height in Relation to Age  & 142
7. Adult Females - Mean Weight in Relation to Age  & 143
8. Adult Females - Body Proportions  & 144
9. Adult Females - Digital Formulae  & 145
10. Male Children - Mean Height in Relation to Age  & 146
11. Male Children - Mean Weight in Relation to Age  & 147
12. Female Children - Mean Height in Relation to Age  & 148
13. Female Children - Mean Height in Relation to Age  & 149
15. Haemoglobin Values  & 161
16. Total and Differential White Cell Counts  & 162
17. Blood Clotting Characteristics  & 163
18. Blood Group Distribution  & 173
19. Glucose Tolerance Tests  & 185
20. Mean Blood Pressure in Relation to Age  & 188
GENERAL INTRODUCTION

This thesis is based on my personal observations of the cultural, physical, and pathological characteristics of the Western Desert aborigines of Australia. My first contact with this group of aborigines occurred in 1958 when I commenced general practice in Kalgoorlie, a town of some 25,000 persons, situated at the south west corner of the Western Desert area. This town was the centre of medical treatment for the group of Western Desert aborigines who lived in Kalgoorlie, its close environs and along the trans-Australia railway line. It was apparent to me at an early stage during the two years spent in this town, that optimum treatment could not be given to these patients due to a lack of knowledge of their culture, physical standards and pattern of disease processes. There was a paucity of literature regarding this basic knowledge, not only in regard to the Western Desert aborigines, but in relation to Australian aborigines in general, and it was in an effort to improve the standard of medical treatment to the aborigines that I commenced the study that forms the basis of this thesis.

This study was continued during the subsequent three years when I was in general practice in Leonora.
Map 2. Areas of Medical Practice in Western Desert.
This town is situated 157 miles north of Kalgoorlie and the practice, which was single handed, covers an area of 250,000 square miles, and includes the major part of the territory of the Western Desert aborigines. It was in this practice area that the majority of this group of aborigines resided.

During this time I was most fortunate in having as my matron at Leonora, Miss Sadie Connor, M.B.E. who is half-aboriginal; who speaks the language fluently, and who knows all the aborigines in this area intimately. It was during these five years in the Western Desert area that most of the field work of this thesis was carried out.

Subsequently, I moved to Perth to various hospital appointments, but my interest in the Western Desert natives continued and I remained in close contact with these people and continued my research. During the past four years I have lived in Brisbane, Queensland and continued to correspond with people in the Western Desert area.

It is on the basis of these past fourteen years experience and study of the Western Desert aborigines, that I present this thesis which considers this ethnic
group from three major aspects.

The first of these is their culture and traditions, because all aborigines in this area are more or less bound by these influences and it is necessary to have some knowledge of them to be able to give adequate medical treatment. In this part I have not only presented the traditions and cultures of this specific group as I observed them, but have also considered how they either correspond to or differ from other aboriginal groups using the available literature on this subject for comparison. I also consider the influence that western civilisation has had on traditional customs.

The second part refers to the physical characteristics of the western desert aborigines, because without a knowledge of the normal parameters it is impossible to define the abnormal which is a fundamental necessity in diagnosis. My own studies in this area have of necessity been restricted to fairly simple tests due to the lack of laboratory facilities in the towns in which I worked, as in Leonora all haematological, biochemical, pathological and radiological tests were performed by myself as the sole medical
practitioner in this area. Again comparisons between the physical standards of the Western desert aborigines and such standards as have been published in regard to other groups of aborigines and the caucasian population are compared. The discussion relates to the differences which are apparent between the various groups and the evidence of a multi-racial origin of the Australian aborigine is examined.

The third section describes the pattern of pathology present in the western desert, established by my personal observations and again a comparison with other aboriginal groups and the caucasian population is presented and discussed.

In general, this thesis, which is restricted to full blood aboriginal group of the Western Desert natives unless otherwise specified, poses more questions than it answers and if this dissertation stimulates further study not only in the Western Desert area but amongst aborigines in general, then I will feel my efforts have been rewarded.
THE CULTURAL TRADITIONS
OF THE
WESTERN DESERT ABORIGINES
INTRODUCTION

The cultural traditions of the Western Desert aborigines have evolved over many thousands of years, the exact time being unknown and subject to the widest speculations. It is not surprising that the exact origin of these customs have been lost in the realms of antiquity and that the customs have been surrounded by a considerable amount of mysticism and tribal rite. They are now inter-woven with tribal law and form an important part of aboriginal life. It is unfortunate that many of the original accounts of aboriginal culture and tradition unduly emphasised this mysticism which in turn led to both bizarre and fanciful theories as to their original motivation. Despite the lack of proof on which these theories were based, repetition by other authors has given them an aura of fact.

It is my belief that most of the customs of the Western Desert aborigine have a teleological basis and represent social and cultural adaptations of this ethnic group to their peculiar environment, and it is in fact only when one considers the aborigine in relation to his environment that one realises the high degree of specialisation which he has attained.
Map 3. Approximate Distribution of Western Desert Aborigines.
THE ENVIRONMENT OF THE WESTERN DESERT

ABORIGINES.
Map 4.

Western Australia - Population.
The Western Desert area covers almost 300,000 square miles, forming almost one-third of Western Australia (975,920 square miles) and more than one-tenth of the total Australian continent, (2,971,081 square miles). It is thus more than three times the size of the British Isles. It is bounded in the west by a rough line joining Agnew, Leonora, Menzies, Kalgoorlie, Coolgardie and Norsemen. The southern border runs approximately 50 miles south of the trans-Australia railway line and the northern border runs approximately north-east by east, extending from north of Agnew, through Lake Wells, the Brown Range and Lake Christopher to the Northern Territory. The eastern border is bounded by the Central Australian desert. The Western Desert area therefore consists of parts of the Great Victoria Desert, the Gibson Desert and the Central Australian Desert. (Maps 1 and 2).

The full-blood aborigines in this area are distributed in a roughly 'C' shaped manner. The northern limb extending from Warburton Ranges in the east, through Cosmo Newberry, Laverton and Mount Margaret Mission to Leonora. From there the western limb extends south from Leonora through Menzies to Kalgoorlie and Coolgardie and then the lower limb
Map 5. Approximate Distribution of White Population in Western Desert Area.
extends westwards along the trans-Australia railway line to Cundulee. The northern and southern limbs of this 'C' are separated by the Great Victoria Desert. The full-blood aboriginal population in this area is shown in Map 3, but the population at each camp is variable according to the time of the season, and the mean distribution is based on my own experience and calculations. The general distribution of the white population of this area is shown in Maps 4 and 5.

The Western Desert region is based on Cambrian and pre-Cambrian rock formation according to Spate (1960), forming a flat tableland averaging 2,000 ft. above sea level. There are various rock outcrops rising to a maximum of 800 feet and these lie principally in the western part of this zone and as these are associated with mineral outcrops, this is where most of the towns and settlements have occurred.

The flora of this area consists of pure desert particularly to the east and north-east, which gradually goes through the transition stage of gibber desert, spinafex desert and salt bush plain as one travels west...
Map 6. Western Australia - Vegetation.
until in the most western areas there are scattered mulga forests. (Map 6).

The area is roughly enclosed by the 25th to 32nd degrees of latitude south and 120 to 130 degrees of longitude east. The average rainfall in this area is 8 – 10 inches at its western extremity, but decreases eastward to considerably less than 6 inches, but in many years is less than 2 inches. (Map 7). The temperature varies from a daily winter average of 50° Fahrenheit and the summer average of 100° Fahrenheit. However, the temperature may frequently fall to 45° Fahrenheit in winter and rise to 120° Fahrenheit in summer, these being shade temperatures. The diurnal swing is considerable and the summer night temperatures frequently fall to 50° Fahrenheit although the average is 70° Fahrenheit. During the winter nights it often falls to below freezing but has an average of 40° Fahrenheit. The humidity of the whole region is extremely low.

The combination of low rainfall, low humidity and high temperatures prevent permanent collections of surface water and all lakes which are shown on maps are actually salt pans which only fill during seasonal
rain, and even at that time are usually too saline to be drinkable. Potable water in the area is therefore restricted to a few variable rock catchments, a few surface springs and such underground water that can be reached by digging wells or sinking bores.

The natural fauna of the area consists of kangaroos, wallabies, very few wombats, a large number of various reptiles, particularly goannas and smaller lizards, and a number of non-venomous snakes, although a few rare venomous types may be seen. There are numerous birds particularly of the budgerigar and parrot families and a number of bustards and emus. The distribution of this game is very closely controlled by and related to the amount of seasonal rain and vegetation and the permanent water holes.

Traditionally the life of these people is geared to a constant search for water and the accompanying game and vegetation. This applies to all aboriginal tribes and each tribe seems to restrict itself to an area sufficient for its needs. Thus the coastal aborigines hunt relatively small areas and considerably supplement their diet with fish while the inland tribes cover large areas of ground in their
search for the essentials of life.

The nomadic state of the Australian aboriginals has generally been stated to be indicative of a primitive society. This however may not be a totally justifiable statement in relation to the Australian aborigine as none of the indigenous fauna in Australia has yet been proven to be capable of domestication and none of the indigenous flora capable of cultivation. It is therefore, apparent that the nomadic state has been forced on the aboriginal has a necessity rather than as a failure to develop a potentially agricultural way of life.

It is against this environmental background, with its absolute bar to attaining what is generally considered a higher degree of civilisation, (as was emphasised by McCarthy (1957)), that one must consider the traditions and cultures of the Western Desert aborigines in particular, and the Australian aborigine in general.
CONCEPTION AND BIRTH.
CONCEPTION

Spencer and Gillen (1899, 1904) and Spencer (1914) stated that the aborigines of Australia did not relate sexual intercourse with subsequent pregnancy and that the aborigines believed that conception only occurred when women visited one of the sacred tribal fertility areas. This statement has also been repeated by Love (1936), Elkins (1938) and Abbie (1960).

In my experience of the Western Desert aborigine, this is not true and the natives have a very real knowledge of the connection between sexual intercourse and subsequent pregnancy. I have questioned many people in this area about this statement including Matron Corner and they all expressed equal disbelief that the aborigines did not connect the two events. It is my belief that these authors have been misled by the aboriginal's natural shyness in discussing what they consider private affairs with strangers and in addition I believe the wrong emphasis has been placed on the fertility areas which to the aborigine represent the same function as a place of worship does in the white race. There is certainly no doubt that all aborigines do connect the two events and I consider it unrealistic to suppose that a race whose very survival
depended on accurate observation and knowledge of nature, would not have had the intelligence to correlate the two events.

It has also been stated by many anthropologists of whom Foelsche (1881), Love (1936), and Elkins (1938), and by a number of medical authorities including Basedow (1919, 1932) and Cleland (1928) that abortion, both spontaneous and acquired, is common in Australian aborigines. However, none of the anthropologists attempt to correlate this statement with their previous statement that the aborigines lack knowledge of the cause of conception and in fact Foelsche (1881) is the only anthropologist who describes the method which is used in the Northern Territory aborigines. This is the extremely doubtful one, of tying a tight belt around their waists. Among the Western Desert aborigines I never found any evidence of procured abortion and close questioning of the full-blood and part-blood aborigines in this area has not indicated that there is any traditional knowledge of procuring abortion by either mechanical or chemical methods and I believe the incidence as stated in the literature is greatly overrated. I have also noted that the incidence of spontaneous abortion in this area is much lower than in the white population.
Another fallacy believed by many white people is that the aboriginal woman does not know when she is pregnant, a belief that has probably arisen from the statement that they don't understand the significance of sexual intercourse. This is certainly erroneous in my experience in the Western Desert area, because although the woman will not confide to strangers that she is pregnant, she will frequently inform some other female and I have often been informed by Matron Corner that the woman thinks she is pregnant and will relate it to change in her menstrual cycle, even in the very early months, and from the basis of clinical examination and from the definite time of the last menstrual period as given by the more sophisticated aborigines, I have found that the duration of pregnancy is the same in both the white and aboriginal races and that the pregnancy and labour is usually uncomplicated in the true, nomadic aborigine.

The birth itself is surrounded with various taboos and customs in the Western Desert area. When the woman's time is due she leaves the main camp accompanied by parous female assistant who will attend to her needs during the confinement. The time spent in the isolated camp will depend on the main body of
the tribe. If the main body is relatively settled they will remain away for several days, to a week, but if the tribe is moving rapidly the expectant mother will only be separated for one or two days before overtaking the rest of the tribe. Possibly the most significant aspect of the selection of the situation for the maternity camp is that as far as is known, it has not previously been used as a camp site. This in itself tends to ensure the sterility of the area and the site is occupied immediately before labour commences. This short duration of stay in the maternity camp in the Western Desert region is different from that described by Love (1936) who stated that the time spent in the maternity camp is of the order of several weeks. He was however referring to the more coastal groups of Western Australian aborigines. These tribes are much more settled and much less nomadic and therefore less likely to travel any great distance from the expectant mother. In the Western Desert area, however, within a matter of two days, she may have over eighty miles to walk in order to catch up the main body of the tribe.

At the maternity camp which is completely taboo to all male members of the tribe, a fire is made and the woman awaits the onset of labour. In the Western
Desert area this camp is in fact not usually occupied until the initial contractions are starting. The woman labours in a squatting position with the assistant sometimes helping with both the birth of the child and the delivery of the placenta by pressing the abdomen. As far as I can gather, she does not actually help to extricate the child from below. Normally the child is not separated from the placenta until after it is delivered, but on occasion the cord may be severed earlier if the third stage is delayed for any reason. Separation of the cord is accomplished by rubbing the cord between two flattened stones (not jagged or sharp), which is an important point in assuring haemostasis, leaving 8 - 12 inches of the cord attached to the baby. The baby is then covered in cold ashes which stick to the vernix giving it an insulated and sterile covering. The placenta is either buried or burnt, and the baby is usually immediately put to the breast. While in the confinement camp the mother follows no particular diet. She seems to recover almost immediately from the birth of the infant. It is not uncommon for the women to start several hours after the delivery of the infant to catch up with the tribe who may be thirty or forty miles ahead of them.
At Warburton Range's mission it is now not uncommon for the women of the tribe to use the Warburton Mission Hospital as a confinement camp, coming in when they have started their contractions, delivering in the traditional squatting manner and leaving the hospital immediately after delivery.
INFANTICIDE, GERIATRICIDE AND CANNIBALISM
INFANTICIDE.

Collins (1798), Curr (1886), Love (1936), Elkins (1938) and Kaberry (1939) all comment on the prevalence of infanticide amongst various groups of Australian aborigines, and I have no doubt it was certainly practiced amongst the Western Desert aborigines, although to a decreasing extent, while I was a general practitioner in that area. I base this statement on there being only one known set of existing twins in the whole of the Western Desert region, prior to 1962, and that that set who were then ten years old had been adopted by a white family. In my own experience the incidence of twin pregnancy in aboriginals is similar to that in the white community and there is no other method of accounting for the dearth of twins in this region apart from the fact that they had either been killed or they had died from illness, and it is difficult to believe that the latter had accounted for all twins born. This belief is supported by the fact there were very few children in the older age groups in the nomadic families who were separated by less than three years of age, although this was a common occurrence in the more urban groups. Other personal communications with people who were interested in this area, particularly Matron Corner,
attested to the fact that it was considered extremely bad luck amongst the Western Desert aborigines to have twins and that normally they would kill one of them. It was also considered equally bad luck to deliver a child while breast feeding one and this second baby would be killed. The method of disposal was to smother the baby at birth.

It is not difficult to understand why this custom has arisen when it is remembered that the aboriginal child is normally breast fed for at least three years and frequently for considerably longer. As McCarthy (1957) has pointed out, the aborigines had no method of boiling or stewing food prior to the arrival of the white man, as he had no knowledge of pottery manufacture. The food was therefore eaten raw, baked or grilled. This type of cooking coupled with the naturally hard and tendinous nature of their available diet, meant that children had to be breast fed until their dental development was sufficient to cope with the normal adult diet. Although the custom of stewing and boiling food has now been assimilated into their way of life, the custom of prolonged breast feeding is still maintained, probably because of simplicity and the assurance of a reasonably constant supply of nutrition (milk). A feature that is not
present in their normal diet. Among the aborigines the combination of assuring a satisfactory milk supply for the growing child and the necessity of carrying this child long distances until it is sufficiently mobile to keep up with the rest of the tribe, meant that the birth of another child within two or three years of the preceding one, added considerable strain on the resources available to the mother. To attempt to bring up a subsequent child while one was still being breast fed, under these circumstances, jeopardised the lives of both children. In the case of twins, it is easy to see how little chance the mother had of coping with breast feeding two children of three years of age, plus carrying them the necessary distances, and again both lives would be jeopardised. The natural method then of protecting the elder child was to kill the younger at birth and this custom has now been a part of the aboriginal life for centuries. There is, however, no evidence that the custom is used indiscriminately to remove children that are undesirable, either due to physical deformity or from parental neglect and it seems to me that the custom has been restricted entirely to those who have been born within two or three years of the previous child or from the result of a twin pregnancy. It is interesting that the full-blood
nomadic aboriginals will not admit to the possibility of twins being born and they disclaim all knowledge of such a possible event occurring. Presumably the fact that two do not survive allows them to ignore the possibility of two babies being born. With twins, however, it appears normal to allow the male to survive if they are different sexes, otherwise it is usually the second that is disposed of. As far as I can gather in the Western Desert area, the method of disposal is as stated earlier by suffocating the child in the sand, although Foelsche (1881) mentioned that it may be accomplished by pressing a hot coal in the mouth of the infant, a method which was apparently used in the Northern Territory, but I have not heard of this in the Western Desert area. I have no doubt that the infanticide rate is decreasing rapidly in the Western Desert area and the reasons for this will be discussed later.
GERIATRICIDE.

This practice has been referred to by Foelsche (1881), Curr (1886) and Love (1936). From their descriptions referring to the aborigines of the Northern Territory and the northern part of Western Australia respectively, it appears that the act was passive rather than active, and consisted simply of leaving the elderly person, usually with some supplies, when they were too feeble and infirm to maintain the pace of the tribe. Generally the elder women were sacrificed before the elder men who were helped because of their status as the most experienced and knowledgeable members of the group. I could find no evidence of this practice in the Western Desert aboriginals but it is reasonable to assume that it was probably practiced in the earlier days and that those members of the tribe who were unfit to maintain the normal pace would be left behind. Today this most probably does not occur as the elderly members of the tribe are left at missions and they remain there for the rest of their days. In this situation, as will be described later, the elder members of the tribe are an important source of material supply due to their money earning capacity as "pensioners."
Love (1936) stated that the aborigines of the Northern Territory when they crossed a possibly alligator infested river, proceeded in the order of the old women first, then the old men, the middle aged and then the young, believing that the first to cross are the most likely to be attacked and therefore sending the most dispensable members across first. It is my belief that geriatricide was in fact never practiced to any extent among the Western Desert aborigines, and only occurred when a member offered direct threat to the survival of the tribe, and not just a general lack of usefulness. This is supported by the fact that I have seen a number of blind and maimed aborigines who accompany the tribe in its nomadic wanderings. These people certainly cannot contribute to the general well-being of the group, but by reason of the fact that they can manage to keep up, albeit with some help, they do not offer a direct threat to its existence and under these circumstances these individuals are treated with great kindness and affection.
CANNIBALISM

Most of the early authorities on Australian aborigines comment to a greater or lesser degree on the prevalence of cannibalism. Certainly, a number of these reports appear to be highly coloured and Elkins (1938) in his review of the subject stated that cannibalism was only practised as part of the burial ritual, when close members of the deceased's family would taste, rather than eat, portions of the body, usually the inside of the thigh and the mammae. This practice was fairly widespread, but evidently not popular, and it decreased rapidly after Australia was colonised and there have been no recent reports of the custom continuing.

I have certainly never heard of the practice amongst the Western Desert aborigines and can find no account of it having been practised in this region. Foelsche (1881) has commented on reports of children and babies being eaten, but there is no substantiating evidence. It is however, possible that in times of great stress, this may have been an occasional event. He also stated however, that the custom was certainly not widespread and was deprecated by any aborigine questioned as to its occurrence.

The customs of infanticide and geriatricide do not appear as inhuman on close examination as they appear
on casual inspection, as they have both appear to have been founded on the basis of what is best for the survival of the race. Both these customs have probably been over emphasised in the literature and used as examples of the primitive nature of these people, when in fact these customs represent one facet of the social adaptation of the aborigine to their peculiar environment and it is apparent that as the environment is changing, so also are their customs, and both of the above habits seem to be decreasing. It is apparent that cannibalism, as it is normally understood, has never been a feature of aboriginal culture and there is certainly no evidence of its existence today.
Of all the Australian aborigines, the Western Desert natives have the least variety of diet. The game in this area consists of kangaroos which are relatively uncommon, the more common rock wallaby which is restricted to various rock outcrops, emus which are dispersed over the whole area and the plain turkey which in the Western Desert region has diminished greatly in numbers due to being shot out by white hunters. Small marsupials such as possums, and the more occasional wombat are also hunted. The male members of the tribe generally spend most of their time in tracking this game with infinite patience. In drought and dry seasons, the game is reduced in numbers and do not breed, as both the kangaroo and wallaby as well as the quokka which was initially described by Shields (1958) may retain the fertilised ovum for some time (in the case of the quokka, for 198 days) if conditions are adverse before development of the ovum proceeds. In fact further development will not proceed unless conditions have improved. However, when the quantity of game is reduced in dry seasons, to a certain extent this is offset to a hunter's advantage by the animals congregating around the available waterholes, dispersing when the next rain occurs.
The women aided by the children forage for smaller animals and edible vegetation. These smaller animals consist of lizards, which vary from the small mountain devil, 2 or 3 inches long, to goannas which may measure up to 6 feet in length, the occasional native cat and since the white settlers arrived, rabbits. Several species of goanna are present in this area. These are caught either by burning a patch of ground or being chased by the dogs till they take refuge in a tree when they will be killed by the women. However much of the female effort in providing food is spent on digging up various edible tubers and gathering the berries and nuts of desert bushes. A variety of grass seeds and the seeds of mulga trees are collected, dried and powdered into flour. Various analysis of the flour produced by these seeds has shown them to have a high protein content, generally of the order of 20%, as shown by Davidson (1957) and Elphinstone (1963). Grubs, particularly wichety grubs, are collected, roasted and eaten. Honey ants are a delicacy, the attached sack of honey which is greatly prized, is bitten off, and eaten. Wild honey is also collected, and bees will be followed to their hive and driven off with smoke while the honey is removed. All species of eggs are gathered, ranging from the large
emu egg to the small bird's egg, and smaller birds are also trapped.

No fruit grows in this area, but a form of wild tobacco is obtainable in the Rawlinson Ranges to which expeditions are made to collect it. It is then dried and mixed with woodash, and ordinary tobacco if available and chewed. This appears to be a type of narcotic similar to the "pitcheri" described by Cleland (1939) and is used by the more eastern tribes.

The food is usually gathered in the morning and early afternoon to be cooked in the later afternoon at camp. After cooking it is carefully divided according to custom, with the relatives (both tribal as well as blood) all sharing in whatever has been collected. The cooking is simple and is such that there is virtually no waste. The game is eviscerated through a longitudinal abdominal incision, the intestines being given to the dogs, while the liver, heart, spleen and pancreas are divided amongst the people, the liver generally going to the hunter who also gets the major share of the food. The abdominal incision is closed by a long wooden skewer, and the
whole animal, including the skin is laid in a prepared hotpit, then covered with warm earth and stones, and the fire is rebuilt on top of it and it is allowed to cook in its own juices. This cooking destroys hair and feathers. When cooked, the animal is carefully removed and reopened, the juice being drunk, all the flesh being eaten, the small bones are chewed up and the larger bones are broken open and the marrow sucked out. Any remaining refuse, the skin and the clean bones, are given to the dogs. Birds and reptiles are similarly cooked and eaten.

Since the advent of the white man, stewing in containers is now quite common, although in the eastern part of the Western Desert the majority of food is still cooked by traditional methods.

Water is obtained from the well, soaks and waterholes known to the tribe and are supplemented and even replaced in times of drought by fluid obtained from animals and succulent roots. A number of the desert trees also store water and these can be incised and water collected from them. In the dry beds of creeks hibernating frogs which contain a store of body fluid and water may be dug up and this fluid used in
place of drinking water. Wells may be also temporarily dug in the course of dry creeks and the seepage of water into these is frequently used by the aborigines to augment their supplies. Although the coolamons (previously described) are the traditional method of carrying water while on walkabout, billy cans are now more frequently used, but even so, only small quantities lasting one to the most, two days, are taken by the aborigines.

In general the standard of nutrition of the desert aborigines is adequate, but in times of shortage, it is obvious that malnutrition may develop. Although overt malnutrition is rare, it is probable that subclinical states do occur more frequently than is realised. The usual diet as outlined above, is a high protein, low fat and low carbohydrate diet. Minerals are supplied from the drinking water, most of which has a considerable mineral content and salt is easily obtained from both the clay pans and the drinking water. Calcium and phosphorous requirements are probably well covered by the normal habit of eating bones, egg shells, whole grain mean and the seeds of native plants. Most of the vitamins are supplied from the same source and from the game livers
heart and lights which also supply their iron intake. Under natural conditions, this tough food combined with the grit attached to it in cooking, is according to Wilson (1954) the main cause of the attrition of the teeth of the desert aborigine. The grit is also probably useful for keeping them free from decay as caries is uncommon in the nomadic aborigine. Although this nutrition is probably adequate during health, there are various phases of life in which it apparently becomes inadequate. Three major phases in which diet is probably effective are initially during early childhood when breast feeding is probably not adequate and the child is not capable of eating the normal nomadic diet. The second phase is again during childhood when apart from the above reasons, the child also has recurrent infections (as is detailed later under pathology). This is often following by a period of mal-absorption and under these circumstances, considerable wasting will occur despite an apparently reasonable intake of food. The third phase is in old age, when a combination of tooth attrition and dental caries in conjunction with loss of teeth results in difficulty in the older members chewing and assimilating the rather tendenous and tough food. Under these conditions nutritional deficiencies may occur.
Problems of the adaption of the aborigine to a more western diet are discussed later.
INITIATION RITES.
An integral part of aboriginal life is the various stages of initiation and the accompanying rites. There are many descriptions of these rites in various tribes of aborigines in Australia and although Curr (1886) and most of the subsequent authors placed most emphasis on the physical mutilation that accompanied these ceremonies, I am in agreement with Love (1936) that the major part of the rite is the increased status due to the knowledge imparted to the initiate in the period prior to the ceremony and that the physical side is less important and is simply an identifying mark of the status which this particular person has reached. None the less the physical part forms a climax to each period of training and in itself marks an important stage in aboriginal life.

Amongst the Western Desert aborigines both the males and females have separate initiation rites. Those associated with the male are much longer; and more complex; occurring in various stages, and in general have much more ceremony than the female rites.

**MALE INITIATION RITES**

In the Western Desert region the most important initiation ceremony is that of circumcision. This rite is preceded by a long period of training for the initiate, and he and his mentor (one of the elder men of the tribe) live separately from the rest of the aborigines, fending for themselves in the bush. During this time the young man is taught
both hunting techniques, and the folk lore of the tribe. The corroboree of circumcision marks the end of this training period and may be held for one or more young men.

The novitiates are usually just past puberty and therefore some 13 years of age. The aborigines have no definite method of estimating time and a decision as to who will be initiated is decided at a meeting of elders, who agree empirically which boys are physically ready to be initiated, and there may therefore appear to be some disparity between the true ages of these boys and the time at which they are initiated.

The ceremony associated with circumcision in the Western Desert region is identical to that in Central Australia and this has been well described by Love (1936), Elkins (1938) and previously by Spencer and Gillen (1896-1904). The operation consists of pulling forward the prepuce and removing the redundant skin with several cuts from a sharp instrument such as flint, a glass sliver or old razor blade, as the aborigines have no hesitation in using any available instrument. The blood is caught in a receptacle and used during the remainder of the ceremony for decorative purposes (as described under Arts, Crafts and Languages). Haemostasis is attempted by covering the raw area with wood ash.

After a further period of training in early adulthood
another ceremony is undertaken by the Western Desert aborigines at which stage the penis is sub-incised. This also is accompanied by a corroboree and is in many ways marks the true attainment of adulthood in this region, as under traditional law a woman will only marry a man who is already sub-incised. In this ceremony the penile urethra is split open dorsally from the meatus backwards for a variable distance. It is common to repeat this sub-incision at subsequent ceremonies for blood letting, and many of the older aborigines have a sub-incision which extends from the meatus to the scroto-penile junction.

**BLOOD-LETTING**

This is a common part of any aboriginal ceremony and is widely used in the Western Desert area. The most frequent site for obtaining blood is from the superficial veins of the lower arm, usually the median cubital, cephalic and basilic veins or their tributaries. The flow and quantity of blood obtained is regulated by ligatures applied above and below the site of the venepuncture. By middle age, most men of the Western Desert area have numerous venesection scars on both arms as either are used indiscriminately. As mentioned above, extension of the sub-incision is another site from which blood is collected. The blood may be caught and mixed in a receptacle ( ) with blood from other individuals to signify brotherhood, and it is also used for decoration or
as a fixative to allow feathers and hair to be attached to the skin.

**CICATRIZATION**

In the variable period between circumcision and subincision, a male is usually adorned by a number of cicatrices denoting his tribe. In the Western Desert area these scars are simple and usually consist of five or six horizontal stripes across the ventral aspects of the chest, with occasionally a few vertically placed scars in the deltoid region. In these lacerations keloid scarring is promoted by rubbing in earth and wood ash into the wounds and this has led many people to believe that keloid is a normal consequence of incisions in an aborigine.

**OTHER RITES**

Other rites which I have seen practised by the Western Desert aborigines are tooth tapping in which the left upper central incisor tooth is removed and nasal seatum perforation.

In the former the operator peels back the gingival margin with a small pointed stick, and then presses a short stout stick against the base of the tooth and both loosens it and removes the tooth with several blows from a stone. This custom is much less prevalent than it has been, and the loss of this tooth is generally restricted to the much older
Nasal septum perforation is also declining in popularity and is infrequently seen amongst the middle aged people. Many of the older aborigines, due to collapse of the nasal septum, have a particularly heavy cast to their countenance. I have not in my time in the Western Desert region seen any evidence of this practice in the younger adult men.

The tribal tradition of circumcision and sub-incision are restricted to the more central areas of Australia, the area of practice or the latter being smaller than that of the former. Various theories have been advanced as to its motivation and Love (1936) considers it to be a return to a matriarchal society while Taplin (1879) believed it was a form of contraception. Roheim (1925) stated that he believed sub-incisions represented an attempt by men to add to their own masculinity the magical reproductive powers of women, by combining in their masculine genitalia the external characters of the female; and Ashley-Montague (1937) agreed with this interpretation. Abbie (1951) used the custom of circumcision by the Australian aborigines as a method of trying to date their origin, but more recently (1969) agreed with Cawte (1969) that both circumcision and sub-incision is performed in imitation of the kangaroo.

I find all these theories fanciful and believe that
since both these practices are restricted to the dry areas of Australia, circumcision has arisen as a method of preventive medicine, and that this particular operation is performed to prevent the complications of balanitis which are extremely common in this area. It is not uncommon for circumcised men in this area to develop meatal ulceration and meatal stenosis and I believe that sub-incision has originated as a simple dorsal slit of the urethral meatus which has gradually been extended. The details of this argument and the comparison with the local white population in the same area in Australia has been presented in the Medical Journal of Australia and a copy of this paper is given in Appendix 1. (Morrison - 1967).

In my experience both circumcision and sub-incision are decreasing in prevalence amongst the Western Desert aborigines and it is not uncommon for the more urbanised full-blood aborigines to have the circumcision performed medically, while is is relatively rare to see sub-incisions (amongst the younger males) except in the nomadic group of aborigines. Blood letting is still extremely common but cicatrization is less common than previously and both tooth-tapping and nasal septum perforation are also extremely rare in the young adult males as noted above.

FEMALE INITIATION RITES

The initiation rites in the female Western Desert
aborigines are much less colourful and have much less importance placed on them than their male counterparts.

There is no history of circumcision ever having been practised amongst the female aborigines and I could find no evidence of the practice in the Western Desert area. Circumcision however, is performed in females at about the age of puberty. There is not the same comparable importance of this ceremony with male circumcision and it is achieved by a ritual rupture of the hymen with a sharp stick or stone, and is usually performed by a female relative. No male attends this ceremony which is usually extremely brief and not marked by a corroboree. The theoretical objectives of this operation is not always achieved as I know an aboriginal woman who stated that her husband had in fact to perform a perineotomy on her after they were married because she was "too small".

The only other common rite performed on females in the Western Desert area is that of cicatrization and this is usually a very simple, single, horizontal scar running between the breasts. Both tooth-tapping and nasal septum perforation have been practised in the past but in my experience are now extremely rare.

There is no equivalent in the Western Desert area to the operation which Foelsche (1881) noted in the Northern Territory among the female aborigines, where the distal two phalanges of the index finger of the left hand were removed.
by tying a tight ligature around the joint. This custom was supposed to increase their dexterity in inserting their fingers into the hole made by their digging stick, but I could find no evidence of this custom having ever existed in the Western Desert region. According to Roth (1897) introcision was practised in Queensland where it was performed by males and Kaberry (1939) reported introcision in the Kimberley region where it was performed by female relatives.
MARRIAGE AND KINSHIP
MARRIAGE

The laws covering marriage between Western Desert aboriginals are very strict, most marriages being arranged between parents soon after the birth of a female child to a much older male child or adult male, and the marriage itself not taking place until the girl has passed puberty. Marriage is only allowed between certain groups, the definition of these groups depending on their particular totem and the tribe to which they belong. In the Western Desert area, the laws of kinship are such that marriages occur between second cousins in a patriarchal lineal manner and the offspring are of a moiety of the fathers totem.

Although the bride is promised to the husband shortly after birth, relationships do not occur between the man and woman until after the girl has passed through puberty and been initiated.

The Western Desert aborigines are basically monogamous, and the husband and wife usually remain together for the duration of their joint lives. However, it is not uncommon for the elderly men to be allowed to take more than one wife. In this area, the maximum I know is 4, and each of the wives of this man appeared to be separated by approximately 15 years of age. Under these polygynous situations, the first wife maintains authority over the younger ones.
Polygamous marriages in the Western Desert region may arise by two methods. The first of these is that the elder men, probably as a sign of status, are granted more than one wife, and these are acquired at intervals. It is probable that these women are acquired largely to support the man, as a wife is the main forager for food in the family unit, (apart from actual hunting of game) and from this point of view, these additional wives are a method of helping the elders to cope with the environment, particularly when he is no longer in a position to hunt himself and his first wife is getting too old to forage efficiently. The second method of acquiring extra wives, is by inheritance, as it is the normal custom if a man dies, that his brother assumes the responsibilities of his family, acquiring both his wife and children and looking after them as if they were his own.

Adultery, except when practised under strict tribal custom, is not permitted but it does occur. It is usually revenged by the husband spearing the offending male and beating his wife, although some authorities state that both of the offenders may be killed, however, in my experience, I have not seen this occur. Occasionally the woman is given to the other women of the tribe for punishment and this may be more severe, and I have seen an aboriginal woman with a short spear passed through the posterior fornix into the peritoneal cavity as a result of such sentencing. The degree
to which the adultery is punished is often dependent on the virility of the husband, and it is not uncommon for the older men with several wives to be unable to pass the traditional sentence on an offending male or to satisfactorily chastise the offending wife.

Adultery is probably not uncommon in traditional camps where there are usually an excess of single mature men, and it is this group in particular, in my experience, that make advances to the younger wives of the tribal elders.

Adultery is however permitted under certain tribal circumstances. These are, that as a mark of respect and hospitality, the husband may offer his wife to his tribal brothers or even to strangers who are visiting the camp. It is also the practice at certain corroborees, for free relationships to occur as long as the laws of kinship are maintained.

**LAWS OF KINSHIP**

The aboriginal tribe is a rather loosely knit group of aborigines who recognise a certain similarity amongst themselves, who in the Western Desert area refer to themselves as the "Wongai".

Within this structure, there are a variety of clans, and the basis of each clan is a number of kinship groups.
It is these kinship groups which show the true functional
element of the social organisation of the tribe. Each
kinship group is divided into two moieties which are repre­
sented by various totems and these moieties are reduplicated
in both the clan system and the tribal system. Under this
system, marriage is never permitted between two persons of
the same moiety. Marriage is only permitted between two
individuals of different totems and different moieties. In
the case of the Western Desert aborigines, the kinship is
based on a patrimonial descent and any offspring of the union
takes his father's totem and moiety. This system, within the
limitations of the numbers available, does however, ensure
an adequate mixing of genes and if examined in detail, is
very similar to the marriage laws of Judaism, as can be seen
in the figure opposite.

TRIBAL GOVERNMENT

The governmental system of the tribe is based on a
gerontocracy. All decisions are reached by a panel of elders
who control the total tribal welfare. The various clans and
kinship groups also have similar gerontocratic structures,
which because of the wide-flung nature of the groups have
reasonably autonomous powers.

In a group of people who have no written language and
in whom the wisdom and knowledge of the centuries is handed
down by word of mouth, it is apparent that the elders do have most knowledge and it is this that gives them their particular power in the tribe. Although the tribal elders are male, the elderly women also have some considerable say in the running of domestic affairs and can exercise in my experience a considerably "lobbying" power on the male council.

Genealogical tables of aborigines are often difficult to construct because of the kinship system, as the child will not only regard its true father as its father, but also all the male members of the same totem and similarly regard all the married females of the same totem as its mothers.

Variations in the marriage laws and kinship laws occur in various other tribal groups of Australian aborigines. For example certain groups in the Northern Territory, have marriage laws based on a matrimonial descent and in some the details as to the accepted bounds of relationship are less strict than in the Western Desert area. The social organisation of the northern tribes in Central Australia have been described, in detail, by Spencer and Gillen (1899) and Love (1936), and Kaberry (1939).
MEDICINE AND MAGIC.
The Western Desert aborigines, in common with all Australian aborigines, consider illness to be due to supernatural forces and a tribal magician is usually consulted in dealing with this. The magician, one of the tribal elders, occupies his position either by inheritance through his father or by an apprenticeship if he is considered suitable. He is consulted not only for treatment, but to reverse the evil spirit back to the human agency that is considered to be using it. More recently, this power has waned with a gradual breakdown of tribal structure but he is still frequently used in conjunction with the local doctor, and he still occupies an extremely important status in the eastern part of the Western Desert region.

His remedies are simple, depending on various incantations and some sleight-of-hand when he will pretend to extract from the painful area various stones and sticks. The patients will frequently relate how many of these objects have been removed and the number usually depends on the severity of the paid. Cawte (1965) gives a very graphic description of such a ceremony in the Kimberley region of Western Australia. Both the medicine man and the aborigines themselves do simple massage for muscular pains. The common treat-
ment of pains in the spinal region is by the patient lying prone while either the medicine man or a helpful relative walks up and down his spine.

Simple surgical skills were understood by the aborigines prior to the advent of the white man and are still practised today, like those for treating snake bites, a treatment which is almost identical with our own and has been described by Foelsche (1881) in reference to the aborigines of the Northern Territory. This treatment which is also used in the Western Desert area, consists of ligatures which are applied proximal to the bite, and the area is deeply lacerated by several incisions with a sharp stone or even bitten out by another aborigine if one is present.

Cautery is also used by the aborigines and in the Western Desert area this is the normal treatment for tooth ache, a burning coal being dropped or pressed into the cavity of the tooth. Curr (1886) describes the use of hot coals for the treatment of conjunctivitis, the glowing coal being passed in front of and close to the cornea, a remedy that must have considerably increased the complications of his condition.
Simple control of bleeding by applying wood ash to encourage haemostasis; a knowledge of milking and compressioning veins to increase or diminish blood flow; and simple digital pressure is similarly well understood and used in everyday practice in the Western Desert region. More complicated operations of circumcision, subincision and posterior perineotoky have already been described. In my experience these are practised with considerable skill, and complications are remarkably rare.

Amputation of the distal two phalanges of the index finger by tying tight ligatures of cobweb and hairstrands around the joint to constrict the circulation has been described by Spencer and Gillen (1899) and previously by Foelsche (1881) in relation to the Northern Territory aborigines although I have seen no evidence of this practice in the Western Desert area. Dinning (1949) has offered proof that some elementary principles of fracture treatment were understood by the aborigines, a fractured leg being treated by leaving the person at the waterhole with the leg straightened and buried in a mud cast for 6 weeks. The recovery of the patient is of course dependent on the tribe staying in the region and keeping him
supplied with food, and apparently this method of
treatment has only been used by the peripheral tribes
in Australia. I could find no evidence of its use
in the Western Desert aborigines. It is probably in
this region that the extreme unlikelihood of the
tribe being in the same locale for six weeks has
negated this form of treatment, although fractures
of other limbs are treating by splinting.

It is common technique of treating any illness
or injury by the application of human-hair bands.
These are worn over the affected part as head bands
for headaches, armbands for joint aches, bracelets
for wrist aches and waist bands for abdominal pain.
For more general disturbances, the hair band is worn
around the neck. I could find no evidence in the
Western Desert area of the aborigines of this region
of ever treating headache by lacerating the forehead,
as Hackett (1936) has reported to be the case in the
more eastern aborigines, and Ford (1942) noted it to
be a common practice in the Northern Territory.
SOCIAL BEHAVIOUR AND PUNISHMENT.
The Western Desert aborigines have a friendly, happy disposition and show affection to their children and respect to their elders. Any afflicted members of the tribe are well looked after and I have seen a blind man who for some 40 years had been looked after by his relatives, taking his place on walkabouts and being guided by another member of his family holding the end of a six foot pole. Similarly, men and women who are lame are assisted in the marches and are well fed by the other members of the community. Both true psychotic illness and psychological upsets are in my experience extremely rare in a full blood aborigine in their natural environment, although behaviour problems in the children and acute psychosis may appear when they are forcibly moved to another habitat.

Time is not considered important and they have no exact method of dating events, except by reference to other events, although within this framework, they can be remarkably accurate.

Within the tribal structure, all possessions are shared to a greater or lesser degree and envy is apparently non-existent. Theft appears to be extremely rare and in my experience of this region, any possession
can be left unguarded, regardless of their potential usefulness to the aborigine.

Superficially there appears to be a lack of individual leadership amongst the Western Desert aborigine, most decisions being reached by a council of the elders. However, it is obvious that certain individual personalities do rise above the rest even in these council meetings. The apparent lack of leadership, coupled with the failure of aborigines who have been partially assimilated to succeed in western civilisation has led Abbie (1951) to postulate that it is possible that the genetic make up of leadership has been lost in Australian aborigines. However, this is not my experience in the Western Desert area and the failure of assimilation is a much more complex problem than simple genetic make up and will be considered later. Again in my experience there is certainly no truth in the fairly widely held belief that the aborigine has an intellectual capacity inferior to that of the white person.

The Western Desert aborigine considers it extremely bad manners to be asked a direct question by a stranger and if this occurs, it is also considered
politeness by the aborigine to give the answer which he thinks is wanted. It is this tradition which has led a number of authorities to state that the aborigine is untruthful, and this custom applies not only to the Western Desert, but other Australian aborigines.

In the Western Desert family, the husband and wife relationship is similar to that of the white person, the man being the head of the family, but the wife having greater or lesser influence depending on her personality. The mother-in-law holds a particularly powerful position commanding considerable respect as well as authority over her son-in-law, although it is the custom for neither to converse directly with the other, all social intercourse being carried on through the medium of a third party which may be either an animate or inanimate object. There are also considerable tribal strictures regarding social intercourse between the sexes of the same moiety group.

Most of the serious inter or intra-tribal disputes are settled on the basis of a spear fight. These may involve from two comtatants to almost the whole male membership of the tribe, but regardless of the number of participants, they follow a set pattern, initially with the comtatants facing and abusing each other. Both sides
gradually work themselves into a passion and eventually the spears are thrown. As soon as blood is drawn, the combat is over, regardless of the number of participants involved, although under the circumstances it is not unusual for several members to be speared simultaneously.

Despite the apparently dangerous aspects of these battles, fatalities are relatively rare, as tribal law demands an opponent may only be speared in the anterolateral aspect of the thigh. This results in a fairly mild flesh wound, with no immediate danger to life. If, as occasionally occurs, an opponent is accidently speared in a more vulnerable spot, the aggressor will be punished by the tribe. Immediately blood is drawn, the fight is over, the combatants help each other, and all animosity is forgotten.

It is generally believed that if an aboriginal accidentally kills another, then his own life is forfeit. This may be so in theory, but it certainly does not occur in practice in the Western Desert area. To my knowledge, four fatalities have occurred in the Western Desert area following spear fights. The first occurred some 40 years ago, when a man killed his brother and it is stated that he was eventually blinded by the tribal elders as punishment. I have already referred to this person and noted that he was well looked after by the tribe, following
this punishment. The other three cases occurred more recently, one just before, one during, and one after the time I was in Leonora. In all these cases, a man who had accidentally killed the other, immediately left the region, with his family and during their absence, each was sentenced to a set number of spearings by the tribal elders. In all three cases, each has served a prison sentence for manslaughter, and are now released and back in the Warburton Desert area. They have in fact been punished since their return and are now completely reassimilated to the tribal pattern.

Disputes between the Western Desert aboriginal women follow a similar pattern, with initially a vocal then a physical attack. The physical attack consists of the two disputants facing each other with nulla-nullas with which they alternatively hit each other on the head, the one left conscious being the winner. Again no animosity or attempt at revenge is held by the loser.

Domestic disputes between husband and wife usually commence with a vocal attack by the wife which is terminated by a beating with a nulla-nulla by the husband, or a more immediate physical attack if the argument is started by the husband.

Invariably, any person injured in these fights
will show great stoicism both during the receipt of the injury, and also during the repair and healing of the wound. Both men and women can suffer tremendous blows to the skull with little effect and most of the women's scalps are marked with numerous scars.

According to Curr (1886) a similar pattern of aggression was present in the major inter-tribal conflicts in earlier days and Love (1936) has reported the same pattern in the north of Western Australian. Both of these authors emphasise the rarity of a fatal outcome in any dispute.

In my experience in the Western Desert area, the reason for most fights was some transgression of the marriage laws and although, superficially the women of the tribe have an extremely hard time, this is not completely true, and their position is probably similar to that of the lower classes in the Caucasian race. The status of the aboriginal woman within the tribe has been dealt with in some detail by Kaberry (1939) who spent some time with the aboriginal women of the Kimberley region of Western Australia.
ART, CRAFT AND LANGUAGE.
Western Desert Churingas

Plate 1.
The art of the Western Desert aborigines is restricted to wood carving and a few rock paintings. Most of the wood carvings are limited to the making of "churingas" which are their sacred emblems. All young men make their own particular churinga for the initiation ceremony. These are of an abstract pattern, usually a number of whirls connected with an interweaving design. Each churinga represents a different animal and story, differing only in minor detail from each other and they are about 12 inches long and of a flattened oval shape as shown in Plate 1.

More exotic carvings are made by the elders for use in the other tribal rituals which are stored in sacred places to be used by successive generations, and may measure up to 10 feet in length. Message sticks are small oblong pieces of wood with a particular message carved in symbols on it to be carried from tribe to tribe. (Plate 2). I have not seen any carved stone artifacts in this region although other tribes in Australia do make them, as described by Curr (1886), Smyth (1878), Love (1936) and Elkins (1938). In all cases these churingas are hidden from the women of the tribe and it is generally believed that any
Western Desert Message Sticks.
woman who sees them will die. Rock paintings are extremely rare amongst the Western Desert natives, unlike those of the Ayres rock area, the Nullabor Plain and the more Northern parts of Australia. A few are to be found in a cave almost midway between the Warburton ranges and Laverton according to Canning (1961) and these consist of representations of humans and animals.

Corroborees are aboriginal gatherings to celebrate a particular event and they follow a stylised pattern developed over the generations. They generally consist of a particular ceremony, e.g. the initiation ceremony, followed by a series of set dances with particular participants which then continue into general tribal dance. In the Western Desert area the participants are generally male. All the dance patterns are simple in outline, usually depicting a particular act in the life of the aborigine, such as hunting an emu, or from their observations of nature, like the snake dance and kangaroo dance, or they may be of symbolic pattern invoking a particular spirit, such as the rain dance. The music accompanying these corroborees is simple, formed by rhythmic handclapping, drumming on a hollow log, and the noise of the bull-roarer. This last is an ovoid piece of wood, generally
Western Desert Bullroarers.

Plate 3.
carved, with a flattened and a convex side. This is whirled around on the end of a piece of rope and made of hair which as it rotates in a circle gives a deep, roaring noise. (Plate 3). The pitch and the rhythm of this can be altered and controlled by the performer. The didgeridoo is not used by the Western Desert aborigine although it is in use in Queensland and the Northern Territory.

Elaborate decorations are often worn at the corroborees consisting of designs painted on the body and face with white ochre, or designs made from emu feathers and stuck to the body with blood, the blood having been previously gathered at a ceremonial blood-letting. Head-dresses of emu feathers and plaited grass are also sometimes worn. The design for the decorations and head-dress depend on the particular ceremony.

Aboriginal crafts are simple and the Western Desert aborigines have the least sophisticated range of all tribes, probably due to the limitations imposed by their environment. They have no craft of pottery or cloth weaving (like all Australian aborigines) and they have no skill at basket weaving (unlike the coastal aborigines who weave baskets and lobster pots). The
crafts of the interior natives are restricted to the manufacture of weapons, utensils and personal adornments and shelters, which latter have already been described.

Weapon making is the major occupation of the adult male aborigines in the Western Desert area and consists of making spears, woomeras, shields, boomerangs, throwing sticks and nulla nullas. (Plate 4). The spears are made with great skill and ingenuity. They are 10 - 12 feet long and average diameter is one inch, and they are fashioned from the long thin roots of the salmon gum tree which grows in the wetter parts of the area in scattered groves. These are extremely sparse towards the eastern parts of the Western Desert area where they are only occasionally found along the edges of dried creek beds. The roots are generally selected after a heavy rain when the rushing creek waters have temporarily uncovered them. It is not uncommon for a group of young men to be sent several hundred miles to gather a supply for the tribe. I know of several occasions when the Warburton aborigines have travelled to Kookynie a round trip of almost a thousand miles to gather roots. The roots are carefully hand straightened while damp over a slow fire, the broader end is carved
Western Desert Weapons and Accessories.

Plate 4.
into a flattened head about six inches long by one and a quarter inches broad, tapering to a point which is fire hardened. A single barb is tied to the middle of the head by a kangaroo tendon while the centre of the tail is recessed to fit the spike of the woomera. Spears used in domestic arguments are not fitted with barbs. When spear shafts break, a frequent occurrence with these long thin weapons, they are repaired with kangaroo tendon by whipping. Spears of the Western Desert area are the simplest design of all aboriginal types, the more Northern and Coastal aborigines frequently using multiple pronged or multiple barbed spears with frequent variations of shell and stone heads.

The woomera is used to propel this spear, acting as an extension of the arm and giving greater velocity and control to its flight. It is generally two and a half to three feet long consisting of a hand grip, a body and a tail. The hand grip is bulbous and is made from the carved, expanded end of the body and to increase its friction and grip, is covered with a mixture of sand and spinafex resin. The body of the woomera is a deep concave tapered distally to a tail to which is fixed by kangaroo tendon, a short, stout spike, pointing towards the handle. The tip of this
spike engages a recess in the tail of the spear. Woomeras are restricted to the tribe using long spears and the Warburton range tribe is easily recognised by its slender appearance and the very deeply carved body. Sometimes a sharp flint is fixed to the handle to be used for sharpening the point of the spear.

The boomerang of the Western Desert aborigines is a plain, angled piece of wood sharpened along both edges. It is thrown at an object with a spin but does not return. These natives have no knowledge of returning boomerangs and it is purely used as a throwing weapon.

Both the throwing stick and the nulla nulla are simple, short weighty sticks that vary from two and a half to three and a half feet in length and are hurled or used as clubs. Both usually have a simple, grooved design carved on them in this region.

The shield is carved from a single solid piece of mulga wood, and is two and a half feet long by only five inches wide, tapering at both ends with a handle carved from solid wood at the back. The front is
usually carved with a simple design. (Plate 6). The shields are used to deflect spears, not to stop them, and he usually increases the effective cover given by the shield, by vibrating it rapidly in his hand, usually the left, while at the same time he fences for an opening to throw his own spear.

Stone knives and axes are occasionally made with chipped flint, the blades being tied to rock handles with sinew or kangaroo tendon.

The only common utensil made by the Western Desert aborigines are "coolamons" which are shallow wooden dishes in which water may be carried. These are oblong vessels and may measure up to seven feet in length, although the most common type is from one to two feet long.

Personal adornments in the Western Desert region are extremely few and simple, apart from those described to wear at corroborees. Generally when on true walkabout, no clothes are worn at all apart from a simple pubic tassel made of plaited hair, grass or bark, by both the adult males and females. Human hair artefacts are the most common type of adornment and
may be worn as a necklet, belt, headband or bracelet, and are believed to have healing powers. Small wallets of skin or bark and hair are used to carry churingas. The only other common personal adornment worn by the adult male, is a short stick or bone artefact through their nasal septum, and these today are usually only seen on ceremonial occasions.

The women make sharp pointed digging sticks about three feet long which they use to dig edible roots and turn over stones in their search for the smaller animal life.

The language of the Western Desert aborigines is "Wongi", with a vocabulary of some 10,000 words, according to Douglas (1962) and Blythe (1962). The grammatical construction is relatively complicated and many words are used to denote several objects, the precise interpretation depending on the construction and context. Within the Western Desert area several varieties of the Wongi dialect exist. The language is generally poor in abstract expressions but with very detailed terminology of the flora and fauna and in family relationships. One major difficulty in learning the language is the constant change which it undergoes as words which are the same
DEATH AND ITS ASSOCIATION
No event in the culture of the aborigines is so surrounded by fear, mysticism and ritual as that of death. The Western Desert aborigines believe that death releases the evil spirit of the person and that this spirit will continue to inhabit the region where the body is buried, and moreover, that the power of this spirit is directly proportional to the virility of the person during life.

Thus the death of a baby is of little consequence, the death of a child is considered to be more powerful and this is increased by that of an elderly woman and next an elderly man. The most powerful spirits are those that belong to the young or middle aged adults and of these the most potent is that of a vigorous young male. The power of the spirit is also considered to be inversely proportional to the period of decline of the individual and there is considerably less awe attached to those deaths following a long and lingering illness, compared with those that occur rapidly.

The Western Desert aborigines have an acute sense of impending death and the ritual associated with death frequently occurs before life is actually extinct. It can be disconcerting for the medical practitioner treating aboriginal patients, whom he feels are either starting to respond to treatment or at least holding their own to hear the relatives starting ceremonies and to know from personal experience that they are seldom wrong in their assessment. I can give no explanation
for this apart from some form of extra-sensory perception. On several occasions I have heard "wailing" coming from the aboriginal reserve (some two miles from the hospital) prior to the death of a patient, even though no aboriginal has been in contact with the patient for at least 24 hours.

The death ceremonies always commence with "wailing", a peculiar high pitched keening note, with alternatively rising and falling cadences which is used by all the aborigines to denote grief. The "wailing" starts among the immediate relatives but progresses throughout the camp until all the aborigines within the area join in. The duration of this "wailing" depends on the potency of the person deceased. The more potent the spirit the longer it lasts. The women of the camp play the major role in these ceremonies, congregating in a central huddle and keeping up an almost continuous noise while the men sit around the periphery and only occasionally join in. At the same time the women closely associated with the deceased person emphasise their grief by lacerating their heads with stones, often helped in this process by friends. The males do not usually inflict injuries on themselves but on occasion I have seen them stab themselves in the thigh with their spears.

While the initial ceremony usually lasts about 24 hours, it will recur spasmodically in the ensuing weeks as fresh members of the tribe either come into camp or are met by the
grieving relatives.

In the Western Desert area the deceased is buried in a shallow grave, the body trussed in a fully flexed position like a fetus and the grave filled with earth and covered with a mound of stones and branches to prevent wild dogs from disturbing it. During the burial the "wailing" continues but shortly afterwards the tribe will abandon the camp where the death occurred, either leaving or more frequently burning the deceased's "humpy" and possessions, and they will avoid that area for a considerable time which is usually several years.

With more static camps such as those associated with the missions the aura of the spirit of the person is believed to remain close to the body, and though his "humpy" and possessions may be abandoned, the main camp site will still be used if the body is buried sufficiently far from it. This distance varies from a few miles for a child to fifteen to twenty miles for an active adult male and it usually falls upon the mission superintendent to transport the body a respectable distance from the major camp. Despite the body being taken some distance from the camp, the majority of the tribe will prepare for walk-about and leave the main camp site for a period, usually varying from several weeks to several months duration. This traditional custom of destroying the deceased's possessions and avoiding the area of death is deeply ingrained in the Western Desert aborigines creating
some problems in their medical treatment which will be
discussed later. As an example of the deep seated nature of
this custom, I can quote the case of a half-caste aborigine
who was apparently well assimilated, with a regular job in
Leonora, and who owned a house in this town. When his son
was killed (in the house) by another aborigine the father
could only be restrained with difficulty from burning the
house to the ground. This episode caused considerable alarm
to the surrounding white population as the house was situated
in the middle of the town which consists mainly of wooden
buildings. Fortunately he was persuaded to desist, but never-
theless he and his family immediately abandoned the house and
moved to an adjoining town.

After the actual burial ceremony the body is left
undisturbed and the area in which it is buried is avoided for
approximately one year when a reburial service is performed.
In this ceremony the remains are exhumed and reburied in a
different site some distance away. This ceremony is brief
and not usually surrounded by ritual.

After death the person will never again be referred
to by name and his or her name if also used as a general
word in the language is dropped from the vocabulary in the
belief that the mention of the name will attract the spirit.

Burial services are apparently similar in all groups
of aborigines according to the descriptions given by Love (1936) in reference to the Northern Territory aborigines and Elkins (1938) and Spencer and Gillen (1894) in reference to the central Australian aborigines. The major differences which occur between the Western Desert aboriginals and these described groups are in detail only, although it would appear that ground burial is largely restricted to the interior of Australia while the northern and coastal aboriginals also practised tree burial in some areas but all groups also later rebury the bones. Cremation is not practised but in some tree burials a fire is lit beneath the body which may be incidentally charred in the process.

Although it is impossible at this stage to decide the original motivation of the various burial customs of Australian aborigines, it is apparent that they are a very useful public health measure and it may well be that teleologically this is their origin. If one considers that the extent of the area which is considered to be inhabited by the spirits is proportional (a) to the virility and (b) the shortness of the terminal illness, then if we replace "influence of spirit" with "virulence of infection" it is apparent how effective these measures could be in protecting the tribe, as severe infectious disease, in the middle aged adult (apart from trauma) is the most likely cause of death. There is evidence (as discussed in a future chapter) that
various epidemics of infectious disease swept the Australian continent prior to white colonisation and if one wants to avoid spread of infection there is probably no better method than to dispose of the body on the immediate site of death and to burn all material possessions, including housing and to immediately decamp from that area and to avoid it for a suitable length of time.

ASSOCIATIONS WITH DEATH

Closely associated with death are the well known customs of "singing" a person to death, "pointing the bone" and the Kadicha man. The two former customs are similar and stem from the aborigine's belief that all illness and death results from someone using spirits to injure them. Despite the wide belief in these customs I have never seen an example where it was believed that either of these methods had been used, although I was dealing with some of the least sophisticated of all the Australian aborigines. I believe that these are largely legends that have persisted in a similar manner to our own fairies and "little people" and though one may recount them as a possible fate, there are no concrete examples to be quoted.

The Kadicha man is a legendary giant, half man, half emu, who represents the natives own conception of "the grim reaper". Anyone seeing his tracks is in imminent danger of death. According to legend the Kadicha covers his tracks by
wearing special shoes which are a loose sandal made of human hair, emu feathers and blood. These shoes are made by the aborigines and worn by the magicians and certain of the elders on secret missions. It is said that no one can be tracked while wearing these shoes, but in my opinion it is more likely that no aboriginal is willing to track anyone wearing them. By heresay, it is stated that they are generally worn by elders commissioned to perform tribal murders passed as a sentence when a particularly serious offence had been committed and which they have decided merits the death penalty.

I have met two elders of the Western Desert region who are reputed to be Kadicha men. The first was stated by Blythe (1960) to have performed at least 5 executions, while the second gained his reputation by travelling from Cosmo Newberry to the Warburton Ranges, a distance of 300 miles, in under 5 days. Time being vouched for by a missionary. However, it was apparently not the distance travelled in this short time that gained him his reputation, but the fact that he was not seen by any other aborigine and had apparently left no tracks that were found by other aborigines. I cannot vouch for the veracity of either of these stories.

Tribal murders are said to be rare and they are apparently reserved for serious infringements of tribal laws. Elkins (1938) and Love (1936) state that they are carried out in other aboriginal groups by spearing, although Hopkins (1960)
stated that in the Western Desert area, he believed that they may be performed by piercing the root of the neck to penetrate the lung with a sliver of kangaroo bone. I have seen no example of this custom in the Western Desert region, although if it does exist, it is not unexpected that I would have no knowledge of the practice, although if it does occur, it must be extremely rare otherwise there would be more specific facts available.
THE INFLUENCE OF WESTERN CIVILISATION

ON THE

TRADITIONAL LIFE OF THE WESTERN DESERT

ABORIGINES
THE EARLY SETTLERS

According to Alexander (1960), the first European contact with Australia may have been made as early as 1527, by the Portuguese spice traders, although the earliest definite landing was by Dirk Hartog on the islands off the coast of Western Australia in 1616. Subsequent landings were made by ships of the East India Company, in the same region, but it was not until 1642 that Abel Tasman was ordered to explore the coast (and, incidentally, discovered Tasmania), that any active effort was made to study the nature of the continent and attempt to chart the coastline. However, his report was so discouraging, according to Cairns (1960), that the East India Company lost any further interest in Australia.

The first British contact was made by William Dampier (1688), and his voyage was rapidly followed by that of Wilhelm de Vlaming in 1696. The most important voyage territorially was that of James Cook in 1770, when he took formal possession of the east coast for the British Crown, and in this voyage and his subsequent journeys in 1772 and 1776 he explored and charted much of the coastline.

The first permanent settlement was at Botany Bay in 1787 under Governor Phillip, although this settlement was soon transferred to Port Jackson, the actual site of Sydney, from where, after considerable difficulties in establishing the
In considering the influences which western civilization has exerted on the Australian aboriginal in general and his way of life, one can place three separate major forces.

The first of these was exerted by the early colonisers, and this effect was most apparent in the coastal regions, where settlement first occurred.

The second major influence was, and to a greater or lesser extent still is, exerted by the various missionary groups who are active both in the coastal and also the internal regions.

The third, and probably the most effective influence, is that due to government legislation, with its provisions for providing both status and aid for the aborigines.

The Western Desert aborigines have been more fortunate than the majority of the native tribes, as their isolation and environment prevented them from coming into contact with the early settlers, and the first contacts were made when Australia was becoming a stable country. This advantage was improved by the rapid establishment of the missions very soon after contact had been made; and in fact for many of the more eastern tribal groups within the Western Desert, their first white contacts were the missionaries, and their present state is therefore largely due to the missionary influence followed by government legislation.
colony, exploration was undertaken.

Although George Vancouver in 1791 and d'Entrecasteaux in 1792 had explored the coast of Western Australia, it had been considered unfit for settlement, although in 1826 an expedition was despatched from Sydney to take formal possession of the west coast due to suspicions that the French were about to establish a base there. This was accomplished by Major Edmund Lockyer at King George's Sound (now the site of Albany), and this party returned to Sydney in 1831. Coincidentally with this expedition, Captain James Stirling had explored the west coast, including the Swan River, and on the basis of his report Thomas Peel formed a syndicate to settle immigrants in this region. Captain C.H. Fremantle had been despatched to take possession of the unoccupied part of Australia west of 120° and Stirling returned in 1829 as Lieutenant-Governor to be followed by parties of emigrants to found the first permanent settlement on the Swan River. While many of the early emigrants left to go to the eastern states, this settlement (now Fremantle and Perth) had been consolidated by 1835. In 1841, an abortive settlement by the Western Australian Company was attempted at Australind to the south, but the exploration of Gray and the Gregory brothers opened up a successful settlement at Champion Bay (Geraldton) on the north coast.

The initial exploration and settlement of Western
Map 7. Early Exploration of Western Australia.
Australia was therefore confined to the coastal belt. Impetus was given to the establishment of properties by convict labour between 1849 and 1868. The northern pastoral territories were explored by the Gregorys in 1863, but no contact was made with the Western Desert natives by any white persons until the great transcontinental exploration of Forrest in 1874 and Giles in 1875-76 and the more northern expeditions of Carnegie and Wells in 1896. These contacts were purely temporary, and some idea of the sparcity of the white population can be gained by the fact that the total white population of Western Australia in 1850 was 5,886, while in 1890 it had mounted to 32,290, nearly all of whom lived in the coastal belt.

However, the discovery of gold brought both a rapid increase in the white population and extended the areas of settlement towards the centre of the continent, the first of these discoveries being at Hall's Creek in 1881, and in the Yilgarn (200 miles east of Perth) in 1888. The much richer finds in the Murchison in 1891, Coolgardie 1892 and Kalgoorlie in 1893 rapidly attracted a population inland. The extension of the Kalgoorlie goldfields south to Norseman in 1894 and north to Leonora, Laverton and Gwalia in 1895, and the innumerable smaller fields in this region led to some 60,000 white people being settled on the western edge of the Western Desert region in the next three to four years.
The contact of the tribal natives of this region with white people was therefore not only abrupt but also overwhelming, and it was quite the opposite of the more gradual process which occurred in the pastoral areas of Australia, particularly in the north-west of Western Australia. This contact was almost entirely deleterious to the aborigines, as the settlers used the aborigines virtually as slaves, and many tales of the horrors perpetrated by the white people are still told.

Water being the most important commodity for survival, the early prospectors took almost any steps to find it, whilst the aborigines equally reticent about revealing it's location. It is stated that methods such as tying an aborigine in the sun and rubbing salt on his tongue until dehydrated then freeing and tracking him to the nearest water-hole were used to learn the location of these wells. The white population of the goldfields were predominantly male, but apart from the usual infectious diseases introduced by them, the employment of Chinese coolies as mine labour and Indians and Afghanistsans as camel drivers also introduced asiatic diseases, such as leprosy and hookworm, while the Kanakas brought the various polynesian diseases with them. The only control of the spread of these diseases was exerted by the climate.

The predominantly male population soon introduced
the aborigines to alcohol, while the Chinese also introduced the habit of opium, and venereal disease rapidly flourished in these circumstances. The general lack of hygiene due to shortage of water laid the foundations for the great typhoid epidemics which swept the goldfields in the early 20th century.

The Western Desert natives were soon divided into two groups. The first group were those native whose tribal territory lay within the areas of white settlement, or alternatively were attracted to the areas of settlement. This particular group were rapidly reduced to destitution, as the local game was shot out and the country denuded of timber for the purpose of building towns. At the same time, this group formed virgin soil for the spread of many infectious diseases, many of which they had not previously been exposed to. Such infections as tuberculosis, the exanthemata, venereal disease and the common cold, contributed greatly to the morbidity and mortality of these tribes. As their natural resources were diminished, so were they forced more and more to depend on the charity of the towns for sustenance, which in turn forced them to congregate in small, permanent camps on the fringes of the local settlements in insanitary conditions which increased the morbidity and mortality; while any work they performed was generally repaid in kind, by goods, alcohol or rations. The rations largely consisted of flour, tea and sugar, and with the major change of diet involved,
so the vicious circle of disease and malnutrition continued. Other goods given for work often consisted of implements such as axes and knives and clothing. The aborigines, having no social background of wearing clothes, naturally never washed them, wore them continuously, and freely exchanged them with each other, all these practices helping to disseminate disease amongst themselves.

The lack of necessity for sanitation in their indigenous, nomadic life, also made them incapable of coping with the static camps now forced on them. Lack of washing facilities and their ignorance of the contaminating effects of sputum also aided the spread of disease, especially of the gastro-enteric type, and tuberculosis. All these facts played important parts in decimating the native population within the areas settled by the Europeans.

The second group of aborigines, consisting principally of those in the eastern part of the Western Desert area, fared better. They remained free of actual contact with the white people until the missionary stations were formed, except for the occasional, more adventurous prospector who passed through their area. However, they did not remain entirely free from the influences of the early settlers, as they still met and co-mingled with the urban aborigines at corroborees, which led to the introduction of most of the infectious diseases to this part of the region. Fortunately, their still nomadic
life tended to confine the spread of disease, and their morbidity and mortality was not nearly as high as in the towns. Within a few years, the goldfield towns had largely stabilised. The boom days of towns appearing overnight at some rich strike and disappearing almost as fast passed. With the stabilisation of these towns, marked improvements in sanitation and water supply occurred, such as the completion of the Kalgoorlie water supply pipeline which extended 350 miles in 1903. The incidence of disease in both the white and native population decreased, while the influx of families into the area reduced the incidence of venereal disease.

Despite the dismal picture presented, the Western Desert aborigines were fortunate that the unstable period was so brief, and a large number therefore survived, unlike the Tasmanian aborigines all of whom were eliminated, and the natives of Victoria and New South Wales, amongst whom the vast majority died of disease or were killed in the century that it took these states to reach a stable and more humane outlook.

The Western Desert natives in the area of the gold-strikes were also fortunate in that it was soon realized that the country was suitable for cattle, and the early 20th century saw the establishment of large pastoral properties in the western parts of this region, which extended as far to the east as Cosmo-Newberry and attracted some of the town
aborigines back to the country. The cattle stations were a boon to these aborigines, most of whom became good stock hands, being used to track and round up cattle, and becoming excellent horsemen. They were ideal for this type of work, as they rapidly learned the routine and quickly understood the basic mechanics in repairing and maintaining windmills and fences. Most important, they were happy to stay for prolonged periods in isolated localities. In return, these stations, which tend to be run on autocratic lines, the manager being frequently the owner or partner, looked after his aborigine employees fairly well as they represented his major work force. Although they were poorly paid monetarily, the manager did house and feed them, the housing frequently being poor but the rations adequate or better, while he also felt responsibility for the employees dependants whom he clothed and also gave medical attention when necessary. The aborigines work was not arduous, and it was accepted practice for them to go walkabout when they felt like it, the manager maintaining a larger work force than necessary to cover these frequent wanderings. The aborigines of the western part of the Western Desert region benefited quite considerably from these cattle stations, maintaining to a large extent their tribal structure with it's ritual, lore and corroborees. They still used and maintained their native skills on walkabout and to a limited extent at work, while at the same time enjoying a stable economy on which they could rely.
The change over from cattle sheep rearing on these properties in the 1920's only increased the work available to the aborigines of the region, and to this day it still represents the major employment available to these people.
THE MISSIONS

The earliest missionary ventures in the Western Desert region were those of individual religious workers, either attached to an established church or functioning on their own as evangelical and social workers in the mining settlements. Most of their efforts were directed towards the white settlers; but a few did realise the plight of the aborigines and did try to help them.

The first true native mission was formed by Mr. Schenk at Mount Margaret in 1922 (65 miles from Leonora). Mr. Schenk was a man of both religious zeal and practical energy and, while much of the mission's efforts were expended on preaching, he did insist that the aborigines in the mission were gainfully employed, repaying them in kind, and serious attempts were made to educate the children by establishing a school, and at a later date a small hospital was added. He also established a trading agency with the aborigines purchasing their artifacts. Some of the success of this mission during that period is shown by the standard of its pupils, one of whom, Miss Sadie Corner, is a triple-certificated matron and was recently awarded an M.B.E. for her services to nursing; another, Miss Rae Miller is a qualified schoolteacher; while several others hold clerical jobs of responsibility. It must be noted however, that all the successful ones are only part-native, and none of the full-blood aborigines have attained a similar
Schenk's success with Mt. Margaret Mission was an inspiration to Wade, who decided to extend the work and, with considerable courage, set off with a few helpers by camel train to found the Warburton Ranges mission in 1934. This mission is one of the most isolated in Australia, situated as it is 380 miles east of Laverton towards the eastern part of the Western Desert. Wade also was a man of both vision and drive and rapidly established his permanent mission, being lucky enough to find an area with easily accessible water and rock suitable for building. He therefore, with his staff, became the first permanent white contact with the majority of the Western Desert natives.

Cundelee Mission, 30 miles north of Zanthus, was established by Carlisle with the intention of restricting the begging activities of the aborigines along the trans-Australian railway line, and acted as a ration depot for the indigent natives of this area.

More recently, Kurrawang Mission (1949) at Kalgoorlie, Norseman Mission (1942) and Cosmo Newberry Mission (1951), have been established, the former two situated in towns to look after the urban aborigines, while the latter was originally a penal settlement 80 miles north-east of Laverton which was given to the United Aborigines Mission to run as a
cattle station for the training of aborigines from the interior. None of these missions are run by any of the established churches, and they draw their staff from the more evangelical groups. Kurrawang and Norseman missions are controlled by the Church of Christ, while Mount Margaret, Warburton Ranges and Cosmo Newberry are controlled by the United Aborigines Mission which consists of a mixture of the Church of Christ, Seventh Day Adventists and the more extreme Methodists.

All the missions are in charge of a superintendent appointed by the controlling order and are staffed by volunteers, the majority of whom are married and have children.

Although the missions did, in many cases, accomplish a non-traumatic initial contact between the Western Desert aborigines and western civilization, their prolonged influence has not necessarily been for the good of the aborigine.

The major defect stems from their staff selection in my opinion. Since their foundation, they have attracted the more fanatical and idealistic type of missionary, with little practical experience of dealing with people, and the missionaries themselves lead a life which is abnormal to that generally accepted by the standards of western civilization. Most of the missionaries avowed purpose is to improve the spiritual welfare of the aborigines (that is, to convert them to the particular group to which the mission-
aries belong) and all other aspects of their welfare are considered to be of relatively minor importance. The missions main line of attack in converting the aborigines has been to break down their tribal structure and in this they have been partially successful by opposing the marriage laws, and decreasing the respect for both the elders of the tribe and the tribal laws. By preaching and emphasising that each man is entitled to only one wife they have fermented unrest amongst the younger males who normally would not marry until their late twenties or early thirties, and made them dissatisfied with the elders taking several wives. In their attempts at education in the more outlying regions, such as Warburton Ranges, they have had several deleterious effects. The first is that, by retaining the children at the mission, they tend to make the adults remain in the vicinity of the mission, thus forming large and permanent camps with poor sanitation and exposure to infection, while the adults in turn become indigent, depending on rations to maintain themselves. Secondly, by keeping the children at school they prevent them from being taught the bushcraft that is so necessary if they are to maintain themselves in this environment and they in turn become indigent. The standard to which these children are educated is invariably extremely low, and there is no satisfactory employment available for them either locally or in the more distant towns. Thirdly, as the parents initially remain in the vicinity of the
missions because of the children being educated there, then in turn when the children eventually grow up they won't leave to try and seek employment elsewhere because their parents are now settled in this vicinity, and thus a vicious circle of untrained aborigines with no tribal structure are gathered into permanent camps in relation to the missions. Although Cosmo Newbury in theory will help to correct this deficiency by both training and providing employment for aborigines as stockmen, this comparatively small work force will have little effect on the whole population. Mount Margaret Mission is in a slightly better situation as it does teach them elementary manual skills and is closer to the pastoral and goldmining industry of the goldfields where some employment can be obtained.

At the present time it would appear that the missions in their initial concept were good and served a useful purpose, but since that time they have accomplished almost nothing and, unless they are controlled more vigorously by government administration, their continued influence will be harmful.

In defence of the missions, it must be stated they do not have much choice in their staff. Few people volunteer for this work, and each of the staff is dependent on charity for supporting himself or herself and their dependents, and much of their energy is channelled into raising finance for themselves.
The major problem, therefore, of the aborigines associated with the missions are that, due to the influence of the missionaries, they are unsuited to continue their normal, nomadic life and at the same time are not given either a suitable training or sufficient contact with normal western civilization to be able to live and compete in the towns of the region.

It is certain in my opinion that assimilation of the aborigines of the Western Desert will not be achieved under the present system of mission training, except for the possibility of Cosmo Newbury Mission and, to a much more limited extent, Mount Margaret Mission. Cundelee Mission and Warburton Ranges Mission, which deal with the majority of the aborigines, are both impracticable and have few facilities, while Kurrawang and Norseman Missions are associated with aborigines already used to town life.
Although the Constitutional Act of the Western Australian Parliament of 1889 theoretically included the part-blood and full-blood aborigines, there was little done to control their exploitation before the Aborigine Act of 1905 was passed. This Act not only defined legally the term "Aborigine" but was specifically passed to protect and improve their welfare, being granted an annual treasury fund of 10,000 per annum and appointing a Department of Aborigines with the necessary power to provide food, clothing and medicine for those in need, controlling areas of land set aside as reserves for the natives, to which access by white people without special permission was an offence; and also preventing co-habitation between the aborigines and white persons. Included in this Act was legislation making it an offence to employ aborigines under a certain age; to supply alcohol and opium to natives; and making it mandatory for employers to provide medical aid to sick aboriginal employees.

This Act, following relatively closely on the foundation of Western Australia and particularly soon after the white settlement of the interior, did much to alleviate the hardships of the aborigines, saving them from the long period of exploitation which marked the colonisation of the eastern States. The Aborigines Act Amendment Act of 1911 increased the fine for supplying liquor and also included the recipient of the liquor as being guilty of an offence. Included in this
Act was legislation preventing a plea of guilty being submitted by an aborigine to a Court except by permission of the local Protector of Natives appointed by the Department of Aborigines, these Protectors usually being the managers of the native reserves. In the subsequent Aborigines Act Amendment Act (1936 and 1940), only minor modifications occurred.

The former increased the range of part-blood people to be classified as natives and made the medical examination of diseased aborigines mandatory. It also legislated that sickness and accident funds include aborigines; and established "native courts" under magistrates with wide discretionary powers to conduct trials against aborigines who were thought to have committed offences against other natives within their tribal structure.

The latter Act did little apart from altering the fines against the sale of liquor and to alter the title of the Acts to "The Native Administration Act",

The Amendment Act of 1941 was passed with the intention of preventing the spread of leprosy by restricting the passage of aborigine across the 20° south parallel except by the express permission of the Administrator.

In 1944 a new era for the aborigine started with the enactment of the natives' "Citizenship Rights" Act allowing
aborigines who fulfilled certain qualifications to assume full Australian citizenship, with both its benefits and responsibilities. The Amendment Act of 1947 altered only minor details of preceding Acts, but further progress was made by the appointment of Magistrate F.E.A. Bateman to conduct a survey of native affairs. His report (1968) was tabled in both Houses of Parliament in Western Australia in 1948. This report had far reaching effects, and was the main instrument in establishing the Native Welfare Department in 1954, following the passage of the Native Welfare Act in 1954.

The Native Welfare Department extended rapidly and, by its activity and control, brought all aborigines within the scope of all social benefits, normally only available to Australian citizens, by amending and improving much of the legislation in the Native Welfare Act of 1960.

More recently, all aborigines have been granted full citizenship status under the Citizenship Rights Act (1964), removing the ban on alcohol, and giving all aborigines the right to vote, as well as full participation in local State and Federal Government affairs, while still protecting them from possible exploitation by the Native Welfare Act of 1963.

The present major effects on the Western Desert aborigines of Government legislation are through the Native
Welfare Department; the social benefits; and the medical services provided by the Government through the Native Welfare Department.
THE NATIVE WELFARE DEPARTMENT, which was established in 1954, as a result of the Bateman Report (reference 21) replaced the Department of Aborigines which had suffered from the grave defect that both the members and the Administrator were appointed from other branches of the Civil Service and frequently had no experience of aborigines. An additional defect was the centralized position of this department in Perth (where the number of aborigines are minimal) with only a few Protectors appointed on the various reserves.

The Native Welfare Department was founded on the lines of the New Guinea administration and, in its initial stages, all the senior officials, including the Commissioner, were recruited from this service, thus ensuring that persons with practical native administrative ability were appointed. With this nucleus, the Department commenced to recruit and train its own field officers, coincidentally establishing district officers in all the principle aboriginal territories, with largely autonomous power in their own region. The Native Welfare Department also returned all the hospitals and nursing establishments which had been established and controlled by the Department of Aborigines to the Public Health Department, thus ensuring that an even standard of treatment and medical facilities were available to both white and aboriginal population.

As the Department grew, sub-offices with a Local
Welfare Officer in charge, were established in nearly all towns in the State of any size. These officers were particularly concerned with local conditions and are responsible to their District Officer who, in turn, follows the broad policy planned by the Central Office, in Perth.

The duties of the local Welfare Officer are numerous and, as their title suggests, their sole concern is the welfare of the aborigines in their district. This consists of supervising the local native reserves; ensuring adequate toilet and washing facilities; the general hygiene of the area; supplying adequate food and clothing, blankets, and medical attention for all indigent aborigines; and attempting to find suitable employment for them. For those employed, they check that wages, living conditions and workers' compensation are satisfactory, and that arrangements are made to transport the employee to and from their place of work at the start and termination of employment. For pensioners, they generally supervise them, and for the less sophisticated aborigines they supervise their pension money, saving some and ensuring that adequate rations are provided with the rest, as well as providing some pocket-money.

The Department also provides legal representation for any aborigine, both in civil and criminal actions and legal advice for contracts and business ventures. Within the Department itself, there is an attempt to employ as many
aborigines as possible and to give both financial and material help to those who wish to purchase houses, and to provide training in the use of these facilities by attempting to graduate them through a range of dwellings of increasing complexity.

In the Western Desert region this training at Cosmo Newbury consists of passing from the 'humpy' to a simple, circular mid-brick dwelling with an iron roof and a concrete floor with a central fire. At Biddies Patch on the Leonora reserves, a simple one roomed corrugated iron dwelling on a concrete base is used. Both these designs allow, in the case of death of an occupant, for the structure to be moved from it's original platform and to be re-used with minimal waste. Thereafter, two or three roomed dwellings on the reserve, and then normal houses in the towns are used, and this education scheme at present is working well. Apart from the houses supplied by the Native Welfare Department, pensioners are encouraged to buy their own simple dwellings, the Department organising their erection and providing the labour.

In other regions, the Native Welfare Department has provided manual training schools and all aboriginal children of any district are guaranteed school and university or technical college education (or, in fact, any type of training for which they show the necessary ability) although few have reached this stage.
The Western Desert aborigines are controlled through the District Office in Kalgoorlie, which has a staff of a District Officer and two Field Officers with the necessary clerical staff, Sub-offices with permanent Local Field Officers are situated at Leonora and Laverton, the latter having an attached Projects Officer who supervises building, production of artifacts and the development of the reserves. All these officers spend as much time on tour as in their respective towns, and the clerical staff cope with the minor problems during their absence.

The Native Welfare Department is thus attempting to assimilate the aborigines without destroying their cultural characteristics, and are trying to preserve the tribal structure as a basis of security. It is yet too early to predict the eventual outcome, but the method appears to be correct, with the emphasis on a satisfactory training fitting them for a reasonable position in western society and educating them in the use of western amenities, while leaving the social organisation intact. It is inevitable that in this early stage of flux the critics claim the Department is too lax in its central control and, by providing the services it does, is making the aborigines lazy and removing their self-reliance. Although there may be some truth in these claims it is impossible to visualize any other method that does not possess greater limitations. While the attempt is to preserve their
cultural characteristics it is inevitable that assimilation must eventually submerge them. Some of the effects of social service benefits and medical services provided by or through the Native Welfare Department are already capable of assessment in the Western Desert region.

**SOCIAL SERVICE BENEFITS**

By representation through the Native Welfare Department, all aborigines became entitled to the benefits enacted by the Social Services Act 1960. This gave them the right to apply for maternity benefits, child endowment benefits, and for both age and invalid pensions. All these entitlements have had a considerable influence on the social structure of the Western Desert natives.

The maternity and child endowment benefits can be studied together. The present benefits of $30 for the birth of each child and child endowment $2, $6, $12 per month for 1, 2 or 3 children until 16 years of age represents to the aborigine with their low cost of living a comparatively high income. It is, in fact, an income on which they can live well within their own society, and this income increases rapidly with the birth of each subsequent child. It is my opinion that this is one of the most important factors in the apparent rise in birth rate among the Western Desert natives. This is particularly apparent amongst the urban population, amongst
whom families of eight and nine children are not uncommon, and who exist almost entirely on the income from maternity and child endowment. This effect has also spread to the more nomadic tribes, where again a general increase in the size of families is occurring and this is apparently due to the decrease in infanticide. Despite the relative isolation of the nomadic groups, they are all quite knowledgable of their entitlements under this Act and waste no time in applying for them at the nearest mission station towards the end of pregnancy. The more nomadic groups are also quite well acquainted with the buying power of this money and use it at the missions for purchasing food and utensils. An interesting example of the effect of these benefits occurred while I was in practice at Leonora. Twins were born to an aboriginal couple at Warburton and, following custom, they wished to dispose of the younger. In this case they did not wish to practise infanticide but just leave it in the hospital and not take any further interest in it. When, however, it was pointed out to them what the twins would be worth in the way of additional maternity and endowment benefits, they were rapidly persuaded to look after both of them, thus demonstrating how financial inducements can overcome deeply ingrained traditions and tribal culture. There is no reason for believing that similar conditions do not exist in other native groups, and it is likely that a major rise in the aboriginal population will occur as a direct result of this social welfare legislation.
Age and Invalid Pension Benefits have also had other effects on the social structure of the Western Desert aborigines, apart from the direct medical benefits. At present, all female persons over the age of 60 years and males over 65 years receive a pension of $10. each per week, and all persons medically examined and certified to be 85% incapacitated for employment receive the same. In addition, their medical treatment is free and concession fares for travel are also granted. Both white and native pensioners receive the same grants. The results is that, what is a poor income for the average white person with their higher standards of living and their increased basic costs, represents comparative luxury to the aborigine. Due to their social structure, this added income goes to the general support of the whole tribe and forms a substantial amount due to the relatively greater number of aboriginal than white pensioners. The greater number of natives receiving pension benefits results from both their longevity (a minor factor) and their almost universal lack of western skills, the latter insuring that even comparatively minor inflictions make them more than 85% incapacitated for employment, as the only employment obtainable is stock work and labouring.

This increase of wealth, although it is dependent on the older tribal members, is distributed throughout the tribe and has affected either directly or indirectly the lives of
most of the members. The effects on the elderly people, both male and female, are the direct ones of improving their standard of nutrition, as they are now able to purchase food to augment that foraged and hunted; and generally, the Native Welfare Officers and missionaries exercise some control to ensure that a reasonable variety of food is supplied, in many cases the Protectors acting as trustees of the pension, giving the natives an amount of pocket-money to spend as they like and arranging for the rest to be supplied as goods.

Another effect on the elderly people is that they now represent an active source of the tribe's food-providing potential and are carefully looked after by the other members, their illnesses being reported early and if at all infirm they are left at the missions, in the towns, or in the settlements while the tribe go walkabout.

A further effect is a direct consequence of the last as, due to their new importance, much more respect is given to the elders in matters of tribal affairs. This is a recurrence of the respect that was the normal state before the effect of western civilization and the ill-directed efforts of many of the missionaries who undermined it during the past century.

However, the effects of Social Benefits on the younger members of the tribe are often detrimental, the tendency being for the younger men to forgo their hunting as food supplies,
although often of poor quality, are now relatively assured, which has led to many of them settling on a fairly permanent basis close to the towns, stations and missions where stores are present. The combination of large groups and young adults with too little to occupy both their time and energy has led to increasing inter-tribal and intra-tribal friction.

In my opinion, Social Benefits are a major bar to assimilation of the aborigines as they can live comparatively well within their own experience and social structure, without the necessity of working either in western civilization, or, hunting within their aboriginal social system. These factors, combine with their lack of envy, their method of sharing all belongings, and their lack of desire to acquire material effects, gives them an outlook for which the western way of life presents few attractions.
MEDICAL PROBLEMS DUE TO THE CULTURAL

TRADITIONS OF THE WESTERN DESERT

ABORIGINES
The cultural traditions of the Western Desert aborigines present a variety of problems in providing adequate medical treatment, and the following methods of circumventing them are based on personal experience.

A major problem is the retention of aboriginal in-patients after a death occurs. Due to their fear of the dead persons spirit it is customary for all patients who are capable of walking to leave the hospital immediately a death has occurred, and they will not return for a variable period of several days to several weeks, depending on the "potency" of the spirit. To a large extent this can be avoided if the person in the terminal stages of illness is transferred from the general wards to a more isolated and private room. If the patients do leave the hospital it is necessary to establish a treatment centre outside the immediate area of the hospital to continue patient care.

Correct dosage of medication is also difficult. The major problems are of three types. The first is due to the usual tradition of sharing all goods and the aborigine automatically shares his medicine with all his relatives. The second is that despite instructions he will tend to take the total medication in a single dose
believing this to be more effective, and the third is that all treatment tends to be discontinued when the patient feels better. The only method of satisfactorily overcoming these problems is by more frequent in-patient care and supervised dispensing of medication, and in many cases parenteral therapy is preferable to oral as a means of ensuring the correct dosage is received by the patient.

Dressings can also become a positive hazard as they will be left untended unless active steps are taken to supervise their regular changing and eventual removal.

The closely-knit structures of the family group is traditional with the aborigine and it is essential to try to maintain it throughout medical treatment. Children especially become fretful if separated from their mother and we have found our best results are obtained if the mother and her other children are also admitted with the sick child and live in the same ward. This calls for reorganisation of the normal ward structures but is well worth the additional effort. It is of course, absolutely mandatory in the case of breast fed infants and children that the mother accompanies the child and in the case of premature infants, lactation is continued.
The attempt to maintain a form of tribal life within the confines of hospital care also necessitates the provision of one of the aborigines major security symbols - namely a fire. Aborigines are unhappy if they cannot congregate around a fire and we found a greater acceptance of hospital care when we had an open fire installed in the general ward. Apart from the obvious happiness it provides it is much less hazardous than the fires which the aborigines will light for themselves if they are not provided. I have seen a fire in an old four gallon tin, perched precariously on two stones, burning on a wooden floored room at Warburton Range hospital. This had been lit without the nurse's knowledge.

Feeding also presents problems, not in the actual provision of food, but the traditional habit of not wasting anything edible, makes in-patients save any food which they have not eaten, storing it in their beds or lockers and this poses one of the major problems of outback medicine, which is the control of flies, and disease spreading insects.

Despite the aborigines' lack of hygiene, as we understand the term, this presents few problems in the hospitalized patient, as the use of toilet facilities,
sputum mugs and bathing are readily accepted. It is however, necessary to emphasize that toilets do not function as general garbage disposal units, and it is essential to supervise baths and showers if patients are not going to scald themselves. The lack of toilet training in the child can stretch the resources of the hospital laundry.

There are only two medical procedures which run counter to Western Desert tradition and these are circumcision before puberty and the intra-venous collection of blood. With the former it is important that circumcision is not done in the nomadic child else he will never be considered properly initiated, and with the latter the adult males regard it as a form of blood letting and while they have no personal objection to the procedure it must not be performed in the view of any female.

These relatively simple medical problems which arise as a direct extension of the Western Desert aboriginal culture are easily overcome, when one is aware that they exist, but they have not been previously presented in any medical literature, and this knowledge is necessary if optimum treatment is to be achieved.
THE PHYSICAL CHARACTERISTICS

OF THE

WESTERN DESERT ABORIGINES.
It is generally believed that all Australian aborigines are derived from a common stock, the major proponents of this homogenous and unitarian theory of origin being Wood Jones (1934), Campbell, Gray and Hackett (1936), Howells (1937), and Abbie (1951). There have, however been a number of authorities who have disagreed with this view, or, in the study of particular physical characteristics have demonstrated significant variations in these features between various aboriginal groups. As early as 1881, Foelsche commented on the different physical appearances of the aborigines of the Northern Territory and those situated in the south. Mourant (1927), Hadlicka (1928), Wagner (1937), and Fenner (1939) all emphasised the varying skull dimension in different groups of aborigines.

In 1941 Birdsell published his tri-hybrid theory of the origin of the Australian aborigines, on the basis of investigation into the physical appearance and the distribution of some of the blood groups in aborigines in various parts of Australia. Though his theory has not generally been accepted, his publication did highlight the physical differences that exist between various groups of aborigines. In brief, he postulated the immigration of three, successive, racial groups from Asia across the Surat shelf to Australia and he names these racial groups The Barreneans, Murrayians and Carpenterians after the localities in Australia where he found what he considered to be the most typical types of each group, and
showing a specific physical characteristics. He postulated that the Barreneans were the first migrant wave and he considered them to be of negrito type with affinities to the present Andamanese and he believed that they had been pushed south by successive immigrant waves and had been largely eliminated except for the Tasmanian aborigines, (who are now extinct), and in some isolated groups of aborigines on the Atherton Tableland around the Barren Lakes, described by Tindale (1939). The Murrayians, the next invasive wave, he believed to be a primitive Caucasian group best typified by the aborigines in the Murray River area and having affinities with the Ainu of Rikkoda and Sokaler. The final migrant wave he named the Carpenterianians who pushed the Murrayians before them and intermingled with them in the more southern regions, although the majority probably settled in the north around the Gulf of Carpentaria, and it is in this region, that he believes the physical characteristics of this group are best seen. This final group he considered to represent a fourth major racial type, of equivalent status to the Caucasians, Negroid and Mongoloid races and these are in fact the typical Australoids of most anthropological classifications, showing affinities with the Viedad elements of India and they are the type most widely described in the literature as the typical Australian aborigine.

However, during the time I spent in the Western Desert area I was impressed by the varying physical features of this
group of aborigines, many of whom had obvious Caucasian features despite the fact they were full-blood, although the majority fitted into the pattern of the typical Australoid. I was also surprised at the relative lack of knowledge of basic parameters of the physical characteristics of the Western Desert aborigines and the variations between this and other groups of Australian aborigines.

In this section I will detail my studies of the sumatographical features of the Western Desert aborigines considering in order the adult male, the adult female and the child.

I will next present my studies of the formed elements of the blood in the Western Desert aborigines and present, where relevant, such studies as have been conducted either in this or other groups of aborigines and which are concerned with these factors.

The third section will present the known distribution of blood groups and other genetic markers in both the Western Desert and other groups of aborigines. It is in this section that one may well be able to substantiate or disprove, a unitarian, or a multi-focal origin of the Australian aborigine. Most of the gene distributions in this section have of course been done by other authors and due reference to these authorities are made. Most of the gene frequencies presented for
the Western Desert area were performed by Gerard Vos and I was privileged to work with him during most of his collecting expeditions and I am grateful to him for the knowledge which he imparted to me.

The fourth and fifth parts of this section consist of the presentation of some basic biochemical blood estimations and physiological parameters in both the Western Desert and other groups of aborigines.

The discussion following this section is based on the comparison of the physical features of the Western Desert aborigines as compared to other groups of Australian aborigines, and also the comparison between the Western Desert aboriginal and normal Caucasian parameters.
THE PHYSICAL FEATURES OF THE
WESTERN DESERT ABORIGINAL
THE ADULT MALE

The Western Desert adult male aborigine is of average height, the mean being 5'7" with a range of 5'2" to 6'1-1/4" and with an average weight of 143 lb., ranging from 95 lb. to 176 lb. These figures are derived from a study of 140 adult men in whom I considered there was no apparent disease and are therefore representative, as far as can be judged, of the normal range of height and weight. The details of these figures in relation to age are shown in Tables 1 and 2 respectively. Analysis of these figures, by dividing the aborigines into urban and nomadic, i.e. those who have been brought up either in a town area or with long contact with western civilisation and the more nomadic group at Warburton Ranges shows that while all aborigines in this area have a mean height and weight below that of the Caucasian norm stated by Geigy 1960, the urban aborigines tend more closely towards the Caucasian norm than do the nomadic aborigines in this area. The apparent diminution in height and weight that occurs in the elderly nomadic aborigines in the Western Desert region, may be either real or only apparent, as there may be a true diminution in height and weight in older life, or it may be related to poorer nutrition in childhood compared with the younger adult aborigines.

The Western Desert adult male is generally thin in build but muscular, the most notable feature being long,
thin upper and lower limbs, due to a relative increase in the length of both these limbs (Table 3), compared to Europeans.

The upper limb is thin with particularly a relative and actual increase in the length of the forearm which terminates in a small slender hand. The usual digital formula of the hand is $3 > 4 > 2 > 5 > 1$, this occurring in 66% while 31% have the formula $3 > 2 > 4 > 5 > 1$, and only 3% have $3 > 2 = 4 > 5 > 1$ (Table 4). The fingerprint patterns are predominantly the whorl type, these constituting 68%, the remainder being arch type (Table 5).

The lower limb is not only thin, with decreased thigh and calf girths, but also absolute increase in length due principally to both a relative and an absolute increase in the length of the lower leg (Table 3). The feet are slender with a prominent calcaneum, thick cornified soles of rubbery consistency, and the usual digital formula of the feet is $1 > 2 > 3 > 4 > 5$, although the remaining 22% show the formula $2 > 1 > 3 > 4 > 5$. The long, narrow feet are normally held parallel with each other while walking.

The trunk is relatively well developed, the absolute measurements showing narrow shoulders and a narrow
pelvis with a short trunk (Table 3). The abdomen is flat although frequently distended after eating, and both the buttocks and the lumbar curvature are less developed than in Europeans.

In general, all the body dimensions are reduced in the elderly except for the antero-posterior diameter of the chest which is slightly increased. This increase, I believe is due to emphysema and not a racial characteristic.

The head is normally dolichocephalic, hypsicephalic and acrocephalic, although in many individual, any or all of these measurements may fall within the normal Caucasian range. The skull, in both anatomical specimens and on x-ray, is noted for the tendency to develop Wormian bones in the occipital region and also for the poor development of both the mastoid and sternomastoid processes. The nasal cavity is usually chaemirhinic and the orbital cavities are microseme with sharp, clearly defined borders. The supra-orbital development of the nasion is normally well marked but may vary from very prominent to minimal, although the degree of nasal depression is more apparent than real due to the large supra-orbital development. In the aboriginal skulls of the Western Desert region which I have x-rayed, there has been markedly poor development of the frontal and the maxillary air sinuses, and a marked,
generalised increase in the thickness of the calvarium. I have confirmed these clinical observations on the aboriginal skulls belonging to the Western Australian University Department of Anatomy.

The superior and inferior alveolar margins normally project and vary between a marked to moderate prognathism, but the mandible is relatively light with a receding synthesis.

The teeth are invariably large and closely compacted, with long roots, and they show considerable attrition with age. Non-eruption of the third molar is not uncommon and is present in 15% of the adult male aborigines I have examined. I have not seen any examples of the so-called "Murrayian" genetic tendency to form fourth molars which was referred to by Birdsell (1941).

The facial features show considerable variation, the forehead usually slopes backwards slightly, although it is occasionally vertical or laterally recessive.

The supra-orbital ridges are usually moderate but may be markedly prominent or less frequently vertical and rarely non-existent. However this feature tends to increase with age and develops as a secondary sexual characteristic in the male.
The nose varies in shape, and in the majority it is moderately flattened while a minority are either markedly flattened with wide flaring alae, a few are prominent and thin. The former two types are more frequently associated with depression of the nasal root than the latter.

The ears are usually well formed, of moderate size and width, with more than half having free lobes, while approximately 50% have small Darwinian tubercles. Large Darwinian tubercles are however very rare.

The orbital cavities are wider than they are high, and the eyeballs may apparently be inophthalmic due to the supra-orbital ridge development, but they are never actually recessive. The iris is always darkly pigmented, usually a deep chocolate brown, although lighter or darker shades may be seen. The conjunctiva frequently show pigmented patches, particularly in the line of the orbital fissure, and a circum-corneal ring of pigmentation may occasionally be present, while in the older people arcus senilis is not uncommon. The conjunctival pigmentation varies between yellow and brown, and it generally increases both in quantity and the depth of colour with age. The fundi are an even slatey-pink, quite unlike the tigroid fundus of the mongoloid race. Variations in the depth of pigmentation occur, but it tends to increase with age.
On account of the deeper fundal pigmentation, the optic disc appears to be lighter than that of the European.

The Buccal mucosa also frequently shows patches of slatey-pink pigmentation, which again increases with age, but the normal mucosa is generally similar to that of Europeans, and is the most useful area in which to detect cyanosis during

Skin pigmentation is extremely variable in both distribution and colour. In the Western Desert region the majority of males are a deep chestnut brown, but they range from deep snuff brown to a very dark brown and brownish-black. Pigmentation is darkest in the linea alba, areola and genital areas, becoming successively lighter in the deltoïd and shoulder region, the biceps, the ventral aspects of the forearm and the body, while the palms of the hands and the soles of the feet are always pale, and the nail beds and the lips are a slatey-pink colour. Again depth of skin pigmentation increases with age.

The hair is variable in quantity, colour and distribution. That of the head is generally brownish-black in colour although light brown is quite common in the younger adults, but true black is very uncommon, while grey hair and baldness become increasingly common after
middle age. Some of the elderly men have pure white hair. The hair length is never more than moderate, although some is removed to make artefacts, and is usually of a moderately wavey form, although straight hair is not uncommon and is more frequent than deep waves. I have never seen helically spiralled hair in this region. The eyebrows are usually dark, frequently black, and they are of an even width, tapering slightly laterally. Again grey is commonly present after middle age and in the elderly, white is not uncommon. In quantity the eyebrows are almost invariably moderate, both abundant and scanty distribution of hair being uncommon, but the quantity often diminishes after middle age. The hair of the beard and the moustache is usually moderate though it is frequently cut or plucked, but it is occasionally abundant, and much less often scanty. The beard hair is often darker than that of the head, coarser, and of a much deeper wave form. After middle age, grey and white interspersions are common, and the quantity often decreases in the elderly, having initially increased from puberty to middle age. Chest hair is usually dark brown but is often light brown in the younger age groups and grey in the elderly. The quantity is usually moderate, but when not, it is more frequently scanty than abundant. The pubic hair is usually dark brown or black, moderate in quantity and of similar distribution to that of the European Male. In the
elderly it diminishes and is interspersed with grey and greyish-white. The hair of both the nasal orifice and the external auditory meatus are much thicker, closer, darker and more abundant than in the European adult male.

THE ADULT FEMALE

The adult female is of average stature in my series of 123 examinations, with a mean height of 5'1-1/2", ranging from 4'6" to 5'10", and with a mean weight of 118 lb. ranging from 84 lb. to 130 lb. This group was selected as being free of apparent disease, and there is a general decrease in height in the elderly, as shown in Tables 6 and 7. The urban group of adult females more closely approximate to the Caucasian norms while the nomadic groups are both well below this in height and weight.

The Western Desert female is generally of slim build, frequently thin and is never muscular. The proportional increase in length of the forearm and lower leg and of the total length of the upper and lower limbs is similar to the male, though the actual supra-sternal-supra pubic length is almost the same in both the male and female (Table 8). The Bi-acromial diameter is much less in the female than in the male, but the pelvic diameters are practically identical.
The woman of the Western Desert region have shallow lumbar curves, and flat buttocks compared with the average Caucasian female. The general skeleton of the female is lighter and more slender than the male and the skull usually has much less defined markings, with the individual diameters and indices lying more frequently within the normal, Caucasian range. Chaemo-rhinna is much less common and the supra-orbital ridge development is less marked in the female.

The average cranial capacity is only slightly less than that of the male and the skull is generally thinner and lighter in build, although still much thicker than in the Caucasian. Wormian bones are common and poor development of the maxillary and mastoid air sinuses occur in the female, although less frequently than in the male.

The facial features tend more to a Caucasian form than in the male, and apart from much smaller proportion having a broad flattened chaemorhinmic nose, there is much less apparent nasal root depression, due to the decreased development of the supra-orbital ridges. The ears are also smaller than in the male, but are well formed, and a similar proportion of females as males have free lobes and Darwinian tubercles (Table 5).

The hands and feet are particularly slender
although the dermatolytic pattern (Table 10) and the
digital formula (Table 9) distribution again are similar
to that of the male.

Pigmentation of skin, hair, conjunctive and
buccal mucosa are lighter in the female, the hair being
commonly a light brown with bleached tips in the younger
women and it is usually of moderate length and deeply
waved. The skin pigmentation generally increases with
age, and the pigmentation of the iris and fundus are
identical to that in the male.

In the elderly grey hair is common, and occasion­
ally pure white occurs. The pubic hair is most commonly
dark brown and like the hair of the beard and moustache
is less in quantity than that of the average European
female.

The breasts are always pendulous after child­
bearing and are small with little subcutaneous fat,
especially in the nomadic group. After middle age strophy
occurs and the lederly breasts are often virtually
non-existent. The nipple is always everted, of moderate
size, and usually developed at the level of the fourth
intercostal space. It is deeply pigmented and occasionally
considerable asymmetry between the two breasts is present,
which I believe may be due to previous infection, and in
several women the history supports this.

THE CHILD

At birth the average weight of the child is 6.1 lb. with an average length of 20.5 inches. The range in weights in my series varied from 4 lb. 10 oz. to 8 lb. 5 oz. and the length from 18.4 to 22 inches. The generally thin limbs and trunk make the babies appear much lighter and thinner than the average Caucasian child, and there is a decrease in subcutaneous fat.

Babies are always pink at birth after respiration is established, although occasionally they show a slightly dusky hue to the skin. The iris is unpigmented and the hair which is commonly thick on both the body and the head is generally black, that of the head often being thick, coarse and straight. Pigmentation of the body becomes apparent during the first few weeks of life commencing with the genital region.

During the early months considerable subcutaneous fat is deposited and the child becomes chubby, the birthweight usually being doubled and trebled before the sixth and twelfth month respectively, in the absence of disease (Table...). Pigmentation of the skin is usually well established by three months and is generally a
cafe au lait colour, slowly darkening over the following years. Permanent hair is frequently very fair and this colour often continues into late teenage. The air is also more often straight in the younger children and increases in waviness with age, and as a general rule, the fairer the hair the less wavey it is. The neurological development of the child is similar as far as can be assessed, as that of the white children. Sucking, moro and grasp reflexes are well established at birth or shortly after, but the Babinski reflex is variable in the age at which it appears. Observation of fixed and moving objects, hearing reflexes, lifting of the head and crawling, all develop at an age comparable to that of the white child. However walking, (as already described) and speech are usually relatively late in developing, averaging three years for the former and four for a very limited vocabulary in the latter. It is apparent that the late development of speech and walking is due to environmental rather than neurological factors, as the development of these milestones are closely related to the degree of assimilation of the aborigine.

The rate of dentition corresponds with that of a Caucasian child.

The growth pattern of both the male and female child are similar to that of white children, except that
until eleven years of age the girls are taller and usually heavier than the boys at which age the males surpass the females, as shown in Tables 10, 11, 12 and 13. There is often a period of stasis in both height and weight in the 3-5 year age group which is associated in my experience with feeding problems and the high incidence of infectious diseases, particularly gastro-enteritis which occurs at this time. Similar restrictions in height and weight in other groups of aboriginal children have been noted by Kirke (1959) and Barrett (1971). Growth in the males continues until about 25 years of age, puberty generally appearing at 12-14 years. The girls generally pass the menarche at 12 years of age and growth continues until late teens or early twenties. In the series of heights and weights I studied in the Western Desert aboriginal children there is a significant difference in these factors in children who attend missions around town as compared to those in more nomadic circumstances, and again these factors seem to be more environmentally than genetically controlled.

As puberty is reached both sexes tend to lose the deep lumbar lordosis and protuberant abdomen which are a frequent characteristic of the children. Both the lordosis and protuberant abdomen have been stated to be due to malnutrition, although it is difficult to find
any obvious clinical evidence of this and it is not associated with hypersplenism or hepatomegaly. It is however frequently related to the size of the meal the child has just eaten, and is extremely variable in degree.

Children also show similar proportional characteristics and a slim body as seen in the adult. The supra-orbital ridges are a secondary sexual characteristic in the male, and not usually developed in the young child, but start to enlarge in the male after puberty.
### Table 1.

**Western Desert Aborigines - Adult Males.**

**Mean Height in Relation to Age.**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>20-24</td>
<td>26 67.3</td>
<td>14 68.3</td>
<td>12 65.9</td>
</tr>
<tr>
<td>25-29</td>
<td>18 67.0</td>
<td>10 67.8</td>
<td>8 66.1</td>
</tr>
<tr>
<td>30-39</td>
<td>32 67.6</td>
<td>19 68.3</td>
<td>13 66.6</td>
</tr>
<tr>
<td>40-49</td>
<td>25 66.9</td>
<td>16 67.6</td>
<td>9 65.7</td>
</tr>
<tr>
<td>50-59</td>
<td>16 67.8</td>
<td>9 69.0</td>
<td>7 66.2</td>
</tr>
<tr>
<td>60-69</td>
<td>14 64.9</td>
<td>7 65.8</td>
<td>7 64.0</td>
</tr>
<tr>
<td>70 +</td>
<td>9 65.0</td>
<td>5 65.5</td>
<td>4 64.2</td>
</tr>
<tr>
<td>Total</td>
<td>140 66.9</td>
<td>80 67.8</td>
<td>60 65.8</td>
</tr>
<tr>
<td>Range</td>
<td>62.0 - 73.2</td>
<td>64.5 - 73.2</td>
<td>62.0 - 70.5</td>
</tr>
<tr>
<td>S.D.</td>
<td>2.58</td>
<td>2.43</td>
<td>2.01</td>
</tr>
</tbody>
</table>

*Age in years, height in inches.

Personal series.
### Table 2.

**Western Desert Aborigines + Adult Males.**

**Mean Weight in Relation to Age.**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>20-24</td>
<td>26</td>
<td>142.5</td>
<td>14</td>
</tr>
<tr>
<td>25-29</td>
<td>18</td>
<td>138.6</td>
<td>10</td>
</tr>
<tr>
<td>30-39</td>
<td>32</td>
<td>146.1</td>
<td>19</td>
</tr>
<tr>
<td>40-49</td>
<td>25</td>
<td>148.4</td>
<td>16</td>
</tr>
<tr>
<td>50-59</td>
<td>16</td>
<td>149.4</td>
<td>9</td>
</tr>
<tr>
<td>60-69</td>
<td>14</td>
<td>131.6</td>
<td>7</td>
</tr>
<tr>
<td>70+</td>
<td>9</td>
<td>128.3</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>140</td>
<td>142.6</td>
<td>80</td>
</tr>
<tr>
<td>Range</td>
<td>95.0 - 176.0</td>
<td></td>
<td>103.3 - 176.0</td>
</tr>
<tr>
<td>S.D.</td>
<td>14.82</td>
<td></td>
<td>12.43</td>
</tr>
</tbody>
</table>

Age in years, weight in pounds.

Personal series.
### Table 3.

**Western Desert Aborigines — Adult Males.**

**Average Body Proportions.**

(36 subjects)

<table>
<thead>
<tr>
<th>Body Dimension</th>
<th>Mean Measurement</th>
<th>% of Height</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Height</td>
<td>66.8</td>
<td>100.0</td>
</tr>
<tr>
<td>Trunk Length</td>
<td>28.4</td>
<td>42.5</td>
</tr>
<tr>
<td>Upper Arm Length</td>
<td>13.2</td>
<td>19.8</td>
</tr>
<tr>
<td>Lower Arm Length</td>
<td>10.4</td>
<td>15.6</td>
</tr>
<tr>
<td>Hand Length</td>
<td>7.9</td>
<td>11.8</td>
</tr>
<tr>
<td>Upper Limb Length</td>
<td>31.5</td>
<td>47.2</td>
</tr>
<tr>
<td>Upper Leg Length</td>
<td>18.2</td>
<td>27.2</td>
</tr>
<tr>
<td>Lower Leg Length</td>
<td>16.0</td>
<td>24.0</td>
</tr>
<tr>
<td>Lower Limb Length</td>
<td>41.0</td>
<td>61.4</td>
</tr>
<tr>
<td>Bi-acromial Diameter</td>
<td>14.2</td>
<td>21.3</td>
</tr>
<tr>
<td>Transverse Chest Diameter</td>
<td>10.4</td>
<td>15.6</td>
</tr>
<tr>
<td>A. — P. Chest Diameter</td>
<td>8.3</td>
<td>12.4</td>
</tr>
<tr>
<td>Intertuberous Diameter</td>
<td>10.3</td>
<td>15.4</td>
</tr>
</tbody>
</table>

All measurements in inches.

Personal series.
Table 4.

Western Desert Aborigines - Adult Males.

Digital Formulae.
(179 subjects)

<table>
<thead>
<tr>
<th>Hand</th>
<th>Pattern</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>3 &gt; 4 &gt; 2 &gt; 5 &gt; 1</td>
<td>119</td>
<td>66.5</td>
</tr>
<tr>
<td></td>
<td>3 &gt; 2 &gt; 4 &gt; 5 &gt; 1</td>
<td>55</td>
<td>30.7</td>
</tr>
<tr>
<td></td>
<td>3 &gt; 2 = 4 &gt; 5 &gt; 1</td>
<td>5</td>
<td>2.8</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>179</td>
<td>100.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Foot</th>
<th>Pattern</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 &gt; 2 &gt; 3 &gt; 4 &gt; 5</td>
<td>139</td>
<td>77.7</td>
</tr>
<tr>
<td></td>
<td>2 &gt; 1 &gt; 3 &gt; 4 &gt; 5</td>
<td>40</td>
<td>22.3</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>179</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Personal series.
### Table 5.

**Western Desert Aborigines - Adult Males and Females.**

#### Dermatoglyphic Patterns.

<table>
<thead>
<tr>
<th>Type</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whorls</td>
<td>122 (68.2%)</td>
<td>134 (63.8%)</td>
</tr>
<tr>
<td>Arches</td>
<td>57  (31.8%)</td>
<td>76  (36.2%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>179 (100.0%)</td>
<td>210 (100.0%)</td>
</tr>
</tbody>
</table>

#### Darwinian Tubercles.

<table>
<thead>
<tr>
<th>Type</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>86  (48.0%)</td>
<td>104 (49.5%)</td>
</tr>
<tr>
<td>Absent</td>
<td>93  (52.0%)</td>
<td>106 (50.5%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>179 (100.0%)</td>
<td>210 (100.0%)</td>
</tr>
</tbody>
</table>

#### Ear Lobe Pattern.

<table>
<thead>
<tr>
<th>Type</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free</td>
<td>106 (59.2%)</td>
<td>115 (54.8%)</td>
</tr>
<tr>
<td>Adherent</td>
<td>73  (40.8%)</td>
<td>95  (45.2%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>179 (100.0%)</td>
<td>210 (100.0%)</td>
</tr>
</tbody>
</table>

*Personal series.*
Table 6.

Western Desert Aborigines - Adult Females.

Mean Height in Relation to Age.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>20-24</td>
<td>33</td>
<td>60.7</td>
<td>20</td>
</tr>
<tr>
<td>25-29</td>
<td>24</td>
<td>61.9</td>
<td>16</td>
</tr>
<tr>
<td>30-39</td>
<td>16</td>
<td>62.6</td>
<td>7</td>
</tr>
<tr>
<td>40-49</td>
<td>28</td>
<td>61.5</td>
<td>16</td>
</tr>
<tr>
<td>50-59</td>
<td>15</td>
<td>62.3</td>
<td>8</td>
</tr>
<tr>
<td>60-69</td>
<td>4</td>
<td>59.4</td>
<td>1</td>
</tr>
<tr>
<td>70+</td>
<td>3</td>
<td>59.6</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>123</td>
<td>61.5</td>
<td>69</td>
</tr>
<tr>
<td>Range</td>
<td>54.0-70.0</td>
<td>56.5-70.0</td>
<td>54.0-67.5</td>
</tr>
<tr>
<td>S.D.</td>
<td>2.01</td>
<td>3.21</td>
<td>1.53</td>
</tr>
</tbody>
</table>

Age in years, height in inches.

Personal series.
Table 7.

Western Desert Aborigines - Adult Females.

Mean Weight in Relation to Age.

Aboriginal Group.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>20-24</td>
<td>33</td>
<td>121.5</td>
<td>20</td>
</tr>
<tr>
<td>25-29</td>
<td>24</td>
<td>122.7</td>
<td>16</td>
</tr>
<tr>
<td>30-39</td>
<td>16</td>
<td>115.6</td>
<td>7</td>
</tr>
<tr>
<td>40-49</td>
<td>28</td>
<td>114.6</td>
<td>16</td>
</tr>
<tr>
<td>50-59</td>
<td>15</td>
<td>117.7</td>
<td>8</td>
</tr>
<tr>
<td>60-69</td>
<td>4</td>
<td>103.7</td>
<td>1</td>
</tr>
<tr>
<td>70+</td>
<td>3</td>
<td>102.3</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>123</td>
<td>117.9</td>
<td>69</td>
</tr>
<tr>
<td>Range</td>
<td>84.0-160.0</td>
<td>93.5-160.0</td>
<td>84.0-127.5</td>
</tr>
<tr>
<td>S.D.</td>
<td>13.26</td>
<td>15.35</td>
<td>8.27</td>
</tr>
</tbody>
</table>

Age in years, weight in pounds.

Personal series.
Table 8.

**Western Desert Aborigines - Adult Females.**

**Average Body Proportions.**

( 31 subjects )

<table>
<thead>
<tr>
<th>Body Dimension</th>
<th>Mean Measurement</th>
<th>% of Height</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Height</td>
<td>61.5</td>
<td>100.0</td>
</tr>
<tr>
<td>Trunk Length</td>
<td>28.4</td>
<td>46.2</td>
</tr>
<tr>
<td>Upper Arm Length</td>
<td>12.4</td>
<td>20.2</td>
</tr>
<tr>
<td>Lower Arm Length</td>
<td>9.3</td>
<td>15.1</td>
</tr>
<tr>
<td>Hand Length</td>
<td>5.9</td>
<td>9.6</td>
</tr>
<tr>
<td>Upper Limb Length</td>
<td>27.6</td>
<td>44.9</td>
</tr>
<tr>
<td>Upper Leg Length</td>
<td>16.3</td>
<td>26.5</td>
</tr>
<tr>
<td>Lower Leg Length</td>
<td>14.4</td>
<td>23.4</td>
</tr>
<tr>
<td>Lower Limb Length</td>
<td>34.9</td>
<td>56.7</td>
</tr>
<tr>
<td>Bi-acromial Diameter</td>
<td>12.4</td>
<td>20.2</td>
</tr>
<tr>
<td>Transverse Chest Diameter</td>
<td>9.3</td>
<td>15.1</td>
</tr>
<tr>
<td>A. -P. Chest Diameter</td>
<td>7.1</td>
<td>11.5</td>
</tr>
<tr>
<td>Intertuberous Diameter</td>
<td>10.2</td>
<td>16.6</td>
</tr>
</tbody>
</table>

All measurements in inches.

Personal series.
Table 9.
Western Desert Aborigines – Adult Females.

Digital Formulae.
(210 subjects)

<table>
<thead>
<tr>
<th>Pattern</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 &gt; 4 &gt; 2 &gt; 5 &gt; 1</td>
<td>148</td>
<td>70.5</td>
</tr>
<tr>
<td>3 &gt; 2 &gt; 4 &gt; 5 &gt; 1</td>
<td>51</td>
<td>24.3</td>
</tr>
<tr>
<td>3 &gt; 2 = 4 &gt; 5 &gt; 1</td>
<td>11</td>
<td>5.2</td>
</tr>
<tr>
<td>Total</td>
<td>210</td>
<td>100.0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pattern</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 &gt; 2 &gt; 3 &gt; 4 &gt; 5</td>
<td>169</td>
<td>80.5</td>
</tr>
<tr>
<td>2 &gt; 1 &gt; 3 &gt; 4 &gt; 5</td>
<td>41</td>
<td>19.5</td>
</tr>
<tr>
<td>Total</td>
<td>210</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Personal series.
Table 10.

Western Desert Aborigines - Male Children.

Mean Height in Relation to Age.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>No.</th>
<th>Mean Age</th>
<th>Mean Ht.</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>35</td>
<td>0.0</td>
<td>20.5</td>
<td>1.52</td>
</tr>
<tr>
<td>3 mth.</td>
<td>37</td>
<td>0.25</td>
<td>23.3</td>
<td>0.98</td>
</tr>
<tr>
<td>6 mth.</td>
<td>26</td>
<td>0.5</td>
<td>25.4</td>
<td>1.10</td>
</tr>
<tr>
<td>9 mth.</td>
<td>24</td>
<td>0.75</td>
<td>27.1</td>
<td>1.06</td>
</tr>
<tr>
<td>1 yr.</td>
<td>16</td>
<td>1.0</td>
<td>28.6</td>
<td>1.09</td>
</tr>
<tr>
<td>1 +</td>
<td>29</td>
<td>1.6</td>
<td>30.6</td>
<td>1.34</td>
</tr>
<tr>
<td>2 +</td>
<td>31</td>
<td>2.5</td>
<td>33.1</td>
<td>1.57</td>
</tr>
<tr>
<td>3 +</td>
<td>45</td>
<td>3.4</td>
<td>36.2</td>
<td>1.68</td>
</tr>
<tr>
<td>4 +</td>
<td>28</td>
<td>4.5</td>
<td>40.1</td>
<td>1.87</td>
</tr>
<tr>
<td>5 +</td>
<td>36</td>
<td>5.5</td>
<td>42.2</td>
<td>1.85</td>
</tr>
<tr>
<td>6 +</td>
<td>28</td>
<td>6.6</td>
<td>44.8</td>
<td>1.75</td>
</tr>
<tr>
<td>7 +</td>
<td>29</td>
<td>7.6</td>
<td>46.3</td>
<td>1.89</td>
</tr>
<tr>
<td>8 +</td>
<td>35</td>
<td>8.5</td>
<td>48.7</td>
<td>2.01</td>
</tr>
<tr>
<td>9 +</td>
<td>42</td>
<td>9.4</td>
<td>50.3</td>
<td>1.97</td>
</tr>
<tr>
<td>10 +</td>
<td>36</td>
<td>10.5</td>
<td>52.5</td>
<td>2.15</td>
</tr>
<tr>
<td>11 +</td>
<td>41</td>
<td>11.6</td>
<td>55.6</td>
<td>2.32</td>
</tr>
<tr>
<td>12 +</td>
<td>37</td>
<td>12.4</td>
<td>56.5</td>
<td>2.33</td>
</tr>
<tr>
<td>13 +</td>
<td>28</td>
<td>13.5</td>
<td>59.2</td>
<td>2.42</td>
</tr>
<tr>
<td>14 +</td>
<td>26</td>
<td>14.5</td>
<td>61.8</td>
<td>2.57</td>
</tr>
<tr>
<td>15 +</td>
<td>31</td>
<td>15.5</td>
<td>65.1</td>
<td>2.56</td>
</tr>
<tr>
<td>16 +</td>
<td>29</td>
<td>16.4</td>
<td>65.8</td>
<td>2.67</td>
</tr>
<tr>
<td>17 +</td>
<td>17</td>
<td>17.7</td>
<td>66.8</td>
<td>2.35</td>
</tr>
<tr>
<td>18 +</td>
<td>15</td>
<td>18.5</td>
<td>67.2</td>
<td>2.69</td>
</tr>
<tr>
<td>19 +</td>
<td>12</td>
<td>19.5</td>
<td>67.5</td>
<td>2.25</td>
</tr>
</tbody>
</table>

Age in years, height in inches.

Personal series.
Table 11.

Western Desert Aborigines - Male Children.

Mean Weight in Relation to Age.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>No.</th>
<th>Mean Age</th>
<th>Mean Wt.</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>35</td>
<td>0.0</td>
<td>6.2</td>
<td>0.85</td>
</tr>
<tr>
<td>3 mth.</td>
<td>37</td>
<td>0.25</td>
<td>12.5</td>
<td>1.82</td>
</tr>
<tr>
<td>6 mth.</td>
<td>26</td>
<td>0.5</td>
<td>15.8</td>
<td>2.10</td>
</tr>
<tr>
<td>9 mth.</td>
<td>24</td>
<td>0.75</td>
<td>17.2</td>
<td>2.16</td>
</tr>
<tr>
<td>1 yr.</td>
<td>16</td>
<td>1.0</td>
<td>18.9</td>
<td>1.89</td>
</tr>
<tr>
<td>1 +</td>
<td>29</td>
<td>1.6</td>
<td>20.7</td>
<td>2.02</td>
</tr>
<tr>
<td>2 +</td>
<td>31</td>
<td>2.5</td>
<td>25.3</td>
<td>2.46</td>
</tr>
<tr>
<td>3 +</td>
<td>45</td>
<td>3.4</td>
<td>28.5</td>
<td>2.38</td>
</tr>
<tr>
<td>4 +</td>
<td>28</td>
<td>4.5</td>
<td>32.8</td>
<td>2.51</td>
</tr>
<tr>
<td>5 +</td>
<td>36</td>
<td>5.5</td>
<td>34.7</td>
<td>2.60</td>
</tr>
<tr>
<td>6 +</td>
<td>28</td>
<td>6.6</td>
<td>38.5</td>
<td>3.12</td>
</tr>
<tr>
<td>7 +</td>
<td>29</td>
<td>7.6</td>
<td>42.8</td>
<td>3.69</td>
</tr>
<tr>
<td>8 +</td>
<td>35</td>
<td>8.5</td>
<td>48.3</td>
<td>4.33</td>
</tr>
<tr>
<td>9 +</td>
<td>42</td>
<td>9.4</td>
<td>56.6</td>
<td>5.21</td>
</tr>
<tr>
<td>10 +</td>
<td>36</td>
<td>10.5</td>
<td>63.6</td>
<td>5.72</td>
</tr>
<tr>
<td>11 +</td>
<td>41</td>
<td>11.6</td>
<td>65.2</td>
<td>7.21</td>
</tr>
<tr>
<td>12 +</td>
<td>37</td>
<td>12.4</td>
<td>70.7</td>
<td>6.82</td>
</tr>
<tr>
<td>13 +</td>
<td>28</td>
<td>13.5</td>
<td>79.2</td>
<td>7.46</td>
</tr>
<tr>
<td>14 +</td>
<td>26</td>
<td>14.5</td>
<td>82.8</td>
<td>8.01</td>
</tr>
<tr>
<td>15 +</td>
<td>31</td>
<td>15.5</td>
<td>93.7</td>
<td>8.65</td>
</tr>
<tr>
<td>16 +</td>
<td>29</td>
<td>16.4</td>
<td>109.2</td>
<td>8.55</td>
</tr>
<tr>
<td>17 +</td>
<td>17</td>
<td>17.7</td>
<td>125.4</td>
<td>9.93</td>
</tr>
<tr>
<td>18 +</td>
<td>15</td>
<td>18.5</td>
<td>131.8</td>
<td>10.76</td>
</tr>
<tr>
<td>19 +</td>
<td>12</td>
<td>19.5</td>
<td>129.6</td>
<td>10.35</td>
</tr>
</tbody>
</table>

Age in years, weight in pounds.

Personal series.
**Table 12.**

Western Desert Aborigines - Female Children.

Mean Height in Relation to Age.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>No.</th>
<th>Mean Age</th>
<th>Mean Ht</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>31</td>
<td>0.0</td>
<td>20.5</td>
<td>0.98</td>
</tr>
<tr>
<td>3 mth.</td>
<td>35</td>
<td>0.25</td>
<td>22.6</td>
<td>1.03</td>
</tr>
<tr>
<td>6 mth.</td>
<td>30</td>
<td>0.5</td>
<td>24.8</td>
<td>1.09</td>
</tr>
<tr>
<td>9 mth.</td>
<td>29</td>
<td>0.75</td>
<td>26.7</td>
<td>1.13</td>
</tr>
<tr>
<td>1 yr.</td>
<td>21</td>
<td>1.0</td>
<td>28.0</td>
<td>1.42</td>
</tr>
<tr>
<td>1 +</td>
<td>26</td>
<td>1.4</td>
<td>29.1</td>
<td>1.37</td>
</tr>
<tr>
<td>2 +</td>
<td>31</td>
<td>2.4</td>
<td>32.6</td>
<td>1.41</td>
</tr>
<tr>
<td>3 +</td>
<td>33</td>
<td>3.5</td>
<td>35.3</td>
<td>1.57</td>
</tr>
<tr>
<td>4 +</td>
<td>40</td>
<td>4.4</td>
<td>39.6</td>
<td>1.68</td>
</tr>
<tr>
<td>5 +</td>
<td>36</td>
<td>5.6</td>
<td>41.8</td>
<td>1.71</td>
</tr>
<tr>
<td>6 +</td>
<td>39</td>
<td>6.6</td>
<td>44.2</td>
<td>2.16</td>
</tr>
<tr>
<td>7 +</td>
<td>31</td>
<td>7.5</td>
<td>47.3</td>
<td>1.96</td>
</tr>
<tr>
<td>8 +</td>
<td>27</td>
<td>8.5</td>
<td>50.4</td>
<td>2.15</td>
</tr>
<tr>
<td>9 +</td>
<td>26</td>
<td>9.4</td>
<td>51.9</td>
<td>2.73</td>
</tr>
<tr>
<td>10 +</td>
<td>31</td>
<td>10.4</td>
<td>53.6</td>
<td>2.48</td>
</tr>
<tr>
<td>11 +</td>
<td>42</td>
<td>11.5</td>
<td>55.1</td>
<td>2.56</td>
</tr>
<tr>
<td>12 +</td>
<td>29</td>
<td>12.5</td>
<td>56.8</td>
<td>2.65</td>
</tr>
<tr>
<td>13 +</td>
<td>25</td>
<td>13.5</td>
<td>58.3</td>
<td>2.32</td>
</tr>
<tr>
<td>14 +</td>
<td>21</td>
<td>14.7</td>
<td>60.7</td>
<td>2.49</td>
</tr>
<tr>
<td>15 +</td>
<td>28</td>
<td>15.5</td>
<td>60.9</td>
<td>2.56</td>
</tr>
<tr>
<td>16 +</td>
<td>32</td>
<td>16.6</td>
<td>61.5</td>
<td>2.82</td>
</tr>
<tr>
<td>17 +</td>
<td>22</td>
<td>17.6</td>
<td>61.8</td>
<td>2.69</td>
</tr>
<tr>
<td>18 +</td>
<td>19</td>
<td>18.5</td>
<td>61.9</td>
<td>2.57</td>
</tr>
<tr>
<td>19 +</td>
<td>17</td>
<td>19.5</td>
<td>61.3</td>
<td>2.63</td>
</tr>
</tbody>
</table>

Age in years, height in inches.  

Personal series.
### Table 13.

Western Desert Aborigines - Female Children.

#### Mean Weight in Relation to Age.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>No.</th>
<th>Mean Age</th>
<th>Mean Wt.</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>31</td>
<td>0.0</td>
<td>6.0</td>
<td>0.79</td>
</tr>
<tr>
<td>3 mth.</td>
<td>35</td>
<td>0.25</td>
<td>11.9</td>
<td>1.53</td>
</tr>
<tr>
<td>6 mth.</td>
<td>30</td>
<td>0.5</td>
<td>14.7</td>
<td>1.36</td>
</tr>
<tr>
<td>9 mth.</td>
<td>29</td>
<td>0.75</td>
<td>16.3</td>
<td>1.55</td>
</tr>
<tr>
<td>1 yr</td>
<td>21</td>
<td>1.0</td>
<td>13.0</td>
<td>2.03</td>
</tr>
<tr>
<td>1 +</td>
<td>26</td>
<td>1.4</td>
<td>20.5</td>
<td>2.12</td>
</tr>
<tr>
<td>2 +</td>
<td>31</td>
<td>2.4</td>
<td>24.6</td>
<td>2.32</td>
</tr>
<tr>
<td>3 +</td>
<td>33</td>
<td>3.5</td>
<td>27.2</td>
<td>2.17</td>
</tr>
<tr>
<td>4 +</td>
<td>40</td>
<td>4.4</td>
<td>31.1</td>
<td>2.01</td>
</tr>
<tr>
<td>5 +</td>
<td>36</td>
<td>5.6</td>
<td>33.2</td>
<td>2.54</td>
</tr>
<tr>
<td>6 +</td>
<td>39</td>
<td>6.6</td>
<td>35.8</td>
<td>2.78</td>
</tr>
<tr>
<td>7 +</td>
<td>31</td>
<td>7.5</td>
<td>40.9</td>
<td>3.12</td>
</tr>
<tr>
<td>8 +</td>
<td>27</td>
<td>8.5</td>
<td>46.7</td>
<td>3.55</td>
</tr>
<tr>
<td>9 +</td>
<td>26</td>
<td>9.4</td>
<td>55.3</td>
<td>4.52</td>
</tr>
<tr>
<td>10 +</td>
<td>31</td>
<td>10.4</td>
<td>64.5</td>
<td>4.37</td>
</tr>
<tr>
<td>11 +</td>
<td>42</td>
<td>11.5</td>
<td>66.7</td>
<td>4.59</td>
</tr>
<tr>
<td>12 +</td>
<td>29</td>
<td>12.5</td>
<td>71.3</td>
<td>4.91</td>
</tr>
<tr>
<td>13 +</td>
<td>25</td>
<td>13.5</td>
<td>80.6</td>
<td>5.48</td>
</tr>
<tr>
<td>14 +</td>
<td>21</td>
<td>14.7</td>
<td>31.4</td>
<td>5.35</td>
</tr>
<tr>
<td>15 +</td>
<td>28</td>
<td>15.5</td>
<td>93.5</td>
<td>6.21</td>
</tr>
<tr>
<td>16 +</td>
<td>32</td>
<td>16.6</td>
<td>106.7</td>
<td>6.12</td>
</tr>
<tr>
<td>17 +</td>
<td>22</td>
<td>17.6</td>
<td>119.3</td>
<td>6.56</td>
</tr>
<tr>
<td>18 +</td>
<td>19</td>
<td>18.5</td>
<td>120.7</td>
<td>7.32</td>
</tr>
<tr>
<td>19 +</td>
<td>17</td>
<td>19.5</td>
<td>118.7</td>
<td>7.35</td>
</tr>
</tbody>
</table>

*Age in years, weight in pounds.

*Personal series.*
THE BLOOD CELLS OF THE WESTERN DESERT ABORIGINES
The characteristics of the formed elements of blood of the aborigines in general and the Western Desert natives in particular have been examined in a variety of respects, and they demonstrate deviations from the normal Caucasian pattern both in form and distribution.

The red blood cells in the Western Desert natives are normal in morphology and are the same size and shape as the corresponding Caucasian population, in the films I have studied. The total red cell count lies within the normal Caucasian range, with a mean of $5.65 \times 10^6$ R.B1/Gen in adult males, and $4.89 \times 10^6$ in adult females. These means and the range are shown in Table 14. These figures were reached with randomly selected healthy aborigines and the tests were performed with the aborigines in a good state of hydration. These figures agree closely with a study conducted by Pitney and Davis (1957) in the same region. The total red cell counts in children in my experience are within the normal European range, but in these I have eliminated any child from 3 to 5 years of age who I considered had a possible nutritional iron deficiency.

Caseley-Smith (1958) in his investigations of the aboriginals of Central Australia (with close affinities to the Western Desert natives) found a significant decrease in the diameter of the red blood cells in the aborigines, the
mean being 7.59 µ in males as opposed to 7.91 µ in Europeans. This difference has not yet been verified by other workers and it is possible that some dehydration may have been present in his series.

The Reticulocytes Count in the Western Desert aborigines are shown in Table 14, and these again lie within the normal range for Europeans, averaging 7.3 in the males and 9.6 in the females per thousand red blood cells, and these again are in fairly close agreement with the Central Australia aboriginal series of Caseley-Smith (1958). There are other reports from the Western Desert region.

Haemoglobin estimations in the Western Desert regions have been previously detailed by Davidson (1957) and Pitney and Davis (1957) who demonstrated that in the small group of aborigines examined there was no evidence of anaemia, and that the normal haemoglobin range for these people lay within the Caucasian range. Caseley-Smith (1958) showed, however, a significantly higher haemoglobin concentration in the aboriginal male than in white males (with a ratio of 16.47 to 15.91 G. per 100 mls), and an even more significant increase in aboriginal females (16.21 to 14.1 G. per 100 mls) in the Central Australian region.

In the Central Desert aborigines I have examined, the estimation being performed with a Sahli haemoglobin-
meter and being checked frequently against standards provided by the Commonwealth Health Laboratories in Kalgoorlie, I can confirm that this area has no evidence of anaemia amongst the aborigines and that high normal values are the rule (Table 15). In this study, the adult males had an average of 14.91 G. per 100 mls ranging from 11.0 to 19.8 G per 100 mls and with elderly males having a mean of 16.15 G per 100 mls and a range of 12.3 to 17.9 G per 100 mls. Amongst the females, the adults have a mean of 14.29 G per 100 mls with a range of 12.1 to 16.5 G per 100 mls, and in elderly, the mean is 15.98 G per 100 mls with a range from 13.9 to 17.1 G per 100 mls. In the pregnant female, the average is 13.11 G per 100 mls and the lactating female has an average of 13.41 G per 100 mls. These figures and their means and range are given in Table 15.

These haemoglobin levels were consistently higher than amongst the corresponding white population of this area. The blood sampling was conducted under similar conditions and dehydration was not present.

These levels agree with Horsfall (1964) who also demonstrated a significant difference between white and aboriginal females during the reproductive age. However, I could not demonstrate a fall during pregnancy compared
with the non-gravid state as Horsfall showed in the aborigines in Queensland. Wardlaw (1935), in the Northern Territory, demonstrated no significant difference in the haemoglobin levels of aboriginal and white populations, but this was a small, restricted series conducted under many difficulties.

Butz-Olsen (1958) working in Central Australia, investigated the presence of abnormal haemoglobins, but could not find any of the common haemoglobinopathies and there have been no references to any abnormal haemoglobins having been detected subsequently.

The white blood cells have also been studied by myself in a series of healthy aborigines of the Western Desert.

The total white cell counts show little difference between the male and the female aboriginal population, the mean being 756 for the former and 7428 per c.mm. for the latter. These mean levels and their standard deviations are shown in Table 16. These figures were obtained in aborigines who had no evidence of dehydration, and with no evidence of infection.

Pitney and Davis (1957) studying a similar group of Central Australian aborigines in an earlier series found no significant difference between the normal white and
native figures, but Caseley-Smith (1958) at Haast's Bluff found significantly higher total white cell counts in the aboriginal than in a white control population, although he stated that some lysis may have occurred in both groups due to temperature conditions and delay in counting. This factor may also have accounted for the lower figures presented by Maegreith (1938) in a similar type of study. Both of these factors I avoided.

The polymorphonuclear leucocytes form the major portion of the white cell count, and in the Western Desert aborigines, in my series, showed no significant sexual difference, forming $60.82 \pm 11.86\%$ in the adult male and $61.93 \pm 37\%$ of the total male and female white cell counts respectively, the actual mean values being 4,534 and 4,599 cells per c.mm.

These results closely correspond to those of Caseley-Smith (1958) in the Central Desert aborigines. I could not, however, demonstrate in the above group any significant shift to the left in the morphology, or a significantly greater number of circulating young polymorphonuclear leucocytes in the peripheral blood, such as was stated by Maigreith (1938) in the Northern Territory aborigines.
The Basophil count closely corresponds to the normal white range, as shown in Table 16, forming 0.35% and 0.46% respectively of the total male and female white cell counts, averaging 26 and 24 cells per c.mm. respectively. These again correspond closely to the figures found by Caseley-Smith for Central Australian aborigines.

Eosinophils, however, show a marked difference in the Western Desert natives that I have studied; the mean eosinophil count was 396 in the male and 414 in the female per c.mm. forming 5.31 \( \pm \) 1.57% and 5.58 \( \pm \) 2.46% of the total male and female count respectively (Table 16). Caseley-Smith (1959), Wardlaw (1935) and Pitney et al. (1957) also found similar variations in their series.

The lymphocytes in the Western Desert natives (Table 16) showed no significant difference between sexes or with the normal white values. The group I studied had a mean of 2,160 and 1,967 cells per c.mm. for the adult males and females respectively, forming 28.96% and 26.48% of the total white cell counts.

Caseley-Smith (1959) found similar counts in the Central Desert natives and using lymphocyte haemograms he demonstrated that there was a significant increase in the number of young lymphocytes in the aborigines of the Central Desert as compared with his white controls. Neither
Pitney at al. (1957) or Maigreith (1938) commented on these features.

The monocytes appear to have a normal morphology, and the mean count in the Western Desert natives in my series shows no significant difference on the mean value for Europeans. Amongst the Western Desert natives I found the mean monocyte count was 340 and 412 cells per c.mm. for adult males and females forming 4.56% and 5.55% of the total white cell count respectively.

Caseley-Smith (1958, 1959) did, however, find a significant difference between the aboriginal males and females, and a significant difference between aboriginal male and white male controls, but no difference between his aboriginal females and white female controls when studying the Central Desert aborigines.

The platelets counts were estimated by myself in a small series of Western Desert natives (Table 17), and these lay within the normal range of 100,000 to 250,000 with a mean of 250,000 per c.mm. The mean adult male count was 330,000, and the mean adult female count was 290,000 per c.mm; with an overall range from 160,000 to 750,000 per c.mm. No other references to thrombocyte counts have appeared in the literature on aborigines.
Blood film examination performed in this series of Western Desert aborigines revealed no abnormalities apart from those detailed above, and there were no abnormalities of morphology on staining; no megalocytes; anisocytosis or poikilocytosis in the films studied.

Other blood characteristics I estimated in the Western Desert aborigines were the bleeding time, the coagulation time, and the clot retraction. No comparative figures are available on other groups of aborigines, and no previous examinations have been made in the Western Desert area.

The bleeding time was estimated in a series of 24 adult aborigines, and as seen from Table 17, all were within the normal Caucasian range and with a mean of 166 seconds. No significant differences were present in the mean bleeding time of the two sexes (180 seconds for male, 151 seconds for female) and the overall range was 70 to 265 seconds.

The coagulation time was estimated using venous blood, and again values were within the normal Caucasian range, with a mean value of 553 seconds ranging from 360 to 725 seconds with a mean of 557 seconds for males and 550 seconds for females (Table 17).
The clot retraction was also performed in the same individuals that were used for bleeding time and coagulation time, and the mean of the total series was 50.3% with a mean of 48.7% for males and 52% for females, the range being from 44% to 64%.

Neither the bleeding time nor clot retraction in aborigines have been reported in the literature.
### Western Desert Aborigines.

#### Mean Red Cell and Reticulocyte Counts.

<table>
<thead>
<tr>
<th>Age gp.</th>
<th>No.</th>
<th>Mean age</th>
<th>Mean R.B.C.</th>
<th>Range</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>15</td>
<td>0.0</td>
<td>5.8</td>
<td>4.9 - 7.2</td>
<td>0.53</td>
</tr>
<tr>
<td>1 -</td>
<td>10</td>
<td>0.7</td>
<td>4.5</td>
<td>3.5 - 5.8</td>
<td>0.48</td>
</tr>
<tr>
<td>1 +</td>
<td>16</td>
<td>1.6</td>
<td>4.3</td>
<td>3.0 - 5.9</td>
<td>0.61</td>
</tr>
<tr>
<td>2 +</td>
<td>19</td>
<td>2.5</td>
<td>3.7</td>
<td>3.1 - 5.7</td>
<td>0.43</td>
</tr>
<tr>
<td>3 +</td>
<td>22</td>
<td>3.5</td>
<td>3.8</td>
<td>2.9 - 5.3</td>
<td>0.55</td>
</tr>
<tr>
<td>4 +</td>
<td>14</td>
<td>4.6</td>
<td>4.1</td>
<td>3.2 - 6.1</td>
<td>0.63</td>
</tr>
<tr>
<td>5-9</td>
<td>26</td>
<td>7.3</td>
<td>4.6</td>
<td>3.4 - 6.6</td>
<td>0.72</td>
</tr>
<tr>
<td>10-14</td>
<td>32</td>
<td>12.8</td>
<td>5.1</td>
<td>3.1 - 6.8</td>
<td>0.57</td>
</tr>
</tbody>
</table>

**Males**

<table>
<thead>
<tr>
<th>Age gp.</th>
<th>No.</th>
<th>Mean age</th>
<th>Mean R.B.C.</th>
<th>Range</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-19</td>
<td>17</td>
<td>17.3</td>
<td>5.6</td>
<td>4.1 - 7.2</td>
<td>0.58</td>
</tr>
<tr>
<td>20-45</td>
<td>26</td>
<td>33.4</td>
<td>5.7</td>
<td>3.9 - 8.1</td>
<td>0.53</td>
</tr>
<tr>
<td>45 +</td>
<td>21</td>
<td>51.2</td>
<td>5.6</td>
<td>4.3 - 6.5</td>
<td>0.46</td>
</tr>
</tbody>
</table>

**Females**

<table>
<thead>
<tr>
<th>Age gp.</th>
<th>No.</th>
<th>Mean age</th>
<th>Mean R.B.C.</th>
<th>Range</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-19</td>
<td>29</td>
<td>17.1</td>
<td>4.9</td>
<td>3.8 - 5.9</td>
<td>0.62</td>
</tr>
<tr>
<td>20-45</td>
<td>34</td>
<td>30.6</td>
<td>4.8</td>
<td>3.7 - 6.1</td>
<td>0.53</td>
</tr>
<tr>
<td>45 +</td>
<td>19</td>
<td>54.5</td>
<td>5.3</td>
<td>4.7 - 6.2</td>
<td>0.47</td>
</tr>
<tr>
<td>Pregnant</td>
<td>79</td>
<td>23.6</td>
<td>3.8</td>
<td>3.2 - 5.6</td>
<td>0.46</td>
</tr>
<tr>
<td>Lactating</td>
<td>32</td>
<td>24.3</td>
<td>4.4</td>
<td>3.3 - 6.1</td>
<td>0.62</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sex</th>
<th>No.</th>
<th>Mean retic./1000R.B.C.</th>
<th>Range</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>13</td>
<td>7.33</td>
<td>4-20</td>
<td>4.42</td>
</tr>
<tr>
<td>Females</td>
<td>22</td>
<td>9.18</td>
<td>3-31</td>
<td>6.75</td>
</tr>
</tbody>
</table>

Age in years, R.B.C. counts x 10^6/cc.

**Personal series.**
Table 15.

**Western Desert Aborigines.**

Mean Haemoglobin Values in Relation to Age.

<table>
<thead>
<tr>
<th>Age gp.</th>
<th>No.</th>
<th>Mean age</th>
<th>Mean Hb.</th>
<th>Range</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>15</td>
<td>0.0</td>
<td>20.4</td>
<td>17.1  - 23.2</td>
<td>1.53</td>
</tr>
<tr>
<td>1 -</td>
<td>10</td>
<td>0.7</td>
<td>13.7</td>
<td>10.3  - 16.9</td>
<td>1.32</td>
</tr>
<tr>
<td>1 +</td>
<td>16</td>
<td>1.6</td>
<td>11.3</td>
<td>10.6  - 14.4</td>
<td>1.06</td>
</tr>
<tr>
<td>2 +</td>
<td>19</td>
<td>2.5</td>
<td>10.2</td>
<td>8.3   - 13.6</td>
<td>1.52</td>
</tr>
<tr>
<td>3 +</td>
<td>22</td>
<td>3.5</td>
<td>10.7</td>
<td>8.5   - 13.8</td>
<td>1.32</td>
</tr>
<tr>
<td>4 +</td>
<td>14</td>
<td>4.6</td>
<td>11.2</td>
<td>9.6   - 14.5</td>
<td>1.21</td>
</tr>
<tr>
<td>5-9</td>
<td>26</td>
<td>7.3</td>
<td>12.5</td>
<td>10.1  - 15.9</td>
<td>1.56</td>
</tr>
<tr>
<td>10-14</td>
<td>32</td>
<td>12.8</td>
<td>13.6</td>
<td>10.1  - 16.7</td>
<td>1.78</td>
</tr>
</tbody>
</table>

**Males**

<table>
<thead>
<tr>
<th>Age gp.</th>
<th>No.</th>
<th>Mean age</th>
<th>Mean Hb.</th>
<th>Range</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-19</td>
<td>17</td>
<td>17.3</td>
<td>14.8</td>
<td>11.3  - 19.8</td>
<td>1.86</td>
</tr>
<tr>
<td>20-45</td>
<td>26</td>
<td>33.4</td>
<td>14.9</td>
<td>11.0  - 18.5</td>
<td>2.14</td>
</tr>
<tr>
<td>45 +</td>
<td>21</td>
<td>51.2</td>
<td>16.1</td>
<td>12.3  - 17.9</td>
<td>1.87</td>
</tr>
</tbody>
</table>

**Females**

<table>
<thead>
<tr>
<th>Age gp.</th>
<th>No.</th>
<th>Mean age</th>
<th>Mean Hb.</th>
<th>Range</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-19</td>
<td>29</td>
<td>17.1</td>
<td>14.1</td>
<td>10.6  - 16.9</td>
<td>1.62</td>
</tr>
<tr>
<td>20-45</td>
<td>34</td>
<td>30.6</td>
<td>14.3</td>
<td>12.1  - 16.5</td>
<td>1.53</td>
</tr>
<tr>
<td>45 +</td>
<td>19</td>
<td>54.5</td>
<td>15.8</td>
<td>13.9  - 17.1</td>
<td>1.28</td>
</tr>
<tr>
<td>Pregnant</td>
<td>79</td>
<td>23.6</td>
<td>13.1</td>
<td>11.5  - 15.1</td>
<td>1.21</td>
</tr>
<tr>
<td>Lactating</td>
<td>32</td>
<td>24.3</td>
<td>13.4</td>
<td>11.1  - 14.8</td>
<td>1.64</td>
</tr>
</tbody>
</table>

Age in years, Hb. in gm./100 ml.

Personal series.
### Table 16.

**Western Desert Aborigines — Adult Males and Females.**

**Total and Differential White Cell Counts.**

<table>
<thead>
<tr>
<th>Cell type</th>
<th>Males</th>
<th>S.D.</th>
<th>Females</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polymorphs.</td>
<td>4534</td>
<td>884</td>
<td>4600</td>
<td>696</td>
</tr>
<tr>
<td>Lymphocytes.</td>
<td>2160</td>
<td>570</td>
<td>1967</td>
<td>547</td>
</tr>
<tr>
<td>Monocytes.</td>
<td>340</td>
<td>120</td>
<td>412</td>
<td>212</td>
</tr>
<tr>
<td>Basophils.</td>
<td>26</td>
<td>16</td>
<td>34</td>
<td>23</td>
</tr>
<tr>
<td>Eosinophils.</td>
<td>396</td>
<td>117</td>
<td>414</td>
<td>183</td>
</tr>
<tr>
<td>Total W.B.C.</td>
<td>7456</td>
<td>1013</td>
<td>7428</td>
<td>1043</td>
</tr>
</tbody>
</table>

All means and standard deviations to nearest whole number.

Personal series.
Table 17.

Western Desert Aborigines - Adult Males and Females.

**Blood Clotting Characteristics.**

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No. of subjects</strong></td>
<td>48</td>
<td>55</td>
</tr>
<tr>
<td><strong>Platelet count</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(cells/c.mm.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean.</td>
<td>330,000</td>
<td>290,000</td>
</tr>
<tr>
<td>Range.</td>
<td>210,000 - 760,000</td>
<td>160,000 - 680,000</td>
</tr>
<tr>
<td>S.D.</td>
<td>43,000</td>
<td>37,000</td>
</tr>
<tr>
<td><strong>Bleeding time</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(seconds)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean.</td>
<td>180</td>
<td>151</td>
</tr>
<tr>
<td>Range.</td>
<td>70 - 265</td>
<td>83 - 267</td>
</tr>
<tr>
<td>S.D.</td>
<td>23</td>
<td>31</td>
</tr>
<tr>
<td><strong>Coagulation time</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(seconds)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean.</td>
<td>557</td>
<td>549</td>
</tr>
<tr>
<td>Range.</td>
<td>360 - 725</td>
<td>370 - 715</td>
</tr>
<tr>
<td>S.D.</td>
<td>63</td>
<td>72</td>
</tr>
<tr>
<td><strong>Clot retraction</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean.</td>
<td>48.7</td>
<td>52.3</td>
</tr>
<tr>
<td>Range.</td>
<td>44 - 62</td>
<td>46 - 61</td>
</tr>
<tr>
<td>S.D.</td>
<td>1.2</td>
<td>1.5</td>
</tr>
</tbody>
</table>
THE BLOOD GROUPS OF THE WESTERN DESERT ABORIGINES
THE ABO GROUP

The first group of aborigines to be investigated were from Queensland, and they were studied by Tebbutt (1922) and this was later extended by Tebbutt and McConnell (1923). These original investigators showed a surprisingly high proportion of O (55.9%) and a low proportion of B (6.2%) with intermediate A (37.3%) groupings. The comprehensive studies of Clelland (1926, 1927, 1928, 1930, 1931, 1932, 1936 and 1938), Lee (1926), Hackett et al. (1936), Birdsell and Boyd (1940) and Simmons et al. (1922, 1954, 1957, 1958) demonstrated that the original assessments were erroneous, Tebbutt having included a number of mixed-blood natives in his series. These authors showed that all Australian aborigines belong to Group A or Group O, and that the B factor is lacking throughout the Australian continent except in Cape York peninsula, which Wilson et al. (1924) considered it to be due to recent admixture with Malaysian and New Guinea natives. Moreover, it was demonstrated by Birdsell (1940) that all aborigines of Group A belong to the sub-group $A_1$. Birdsell, in his isogenic distribution tables of Australia, later expanded by Wilson et al. (1944) also demonstrated considerable variation in the distribution of the $O$ and $A_1$ genes, ranging from a preponderance of $O$ in the north to the highest concentration of the $A_1$ in the centre of Australia.
The Western Desert aborigines, therefore, have a distribution of Group O of 43.1% and Group A₃ of 56.9%, with a genetic distribution of P (factor A₃): 0.343, Q (factor B) = 0, and R (factor 0) = 0.567. These figures of Birdsell (1940) are in close accord with the more recent, but as yet unpublished, investigations of Vos et al. (1969) which show the highest concentration of Group A₃ to be in the centre of the continent, in the Warburton Ranges area.

THE M, N, S, GROUP

Amongst Australian aborigines this group was first extensively investigated by Birdsell and Boyd (1940), although Clelland (1938) had made some earlier contributions. Again, Wilson et al. (1944) extended Birdsell's series and produced a modified isogone distribution, showing the variations of the incidence of the M and N factors in the Australian continent. He demonstrated that, while all groups of aborigines have a high incidence of N, this incidence increases towards the centre of Australia, the highest incidence occurring in the Western Desert aborigines (in the Laverton-Warburton Range area). An isolated pocket of extremely high N (n = 0.94) at Ernabella Mission in South Australia was discovered by Simmons, Graydon and Semple (1954). Sanger (1950), and Sanger and Walsh and Kay (1951) investigated the distribution of the S factor in
Queensland, and this was later extended by Simmons et al. (1954, 1957, 1958) for the Central Australian region, while Vos (1962) has investigated the Western Desert natives. All these authors have found a complete absence of S in the Australian aborigine, and the distribution of the M,N,S system in the Western Desert is \( M = 4\% \), \( MN = 39\% \), \( N = 57\% \), \( S = 0\% \).

**THE RHESUS GROUP**

This was first investigated in Australian aborigines by Simmons, Graydon and Sample (1944) following the work of Landsteiner and Weiner (1940, 1941) on negroes, Weiner and Matson (1942) on North American Indians, and Levine and Wong (1943) on Chinese, all of whom showed racial variations in this system. Simmons et al. (1944) in their initial series of 281 full blood aborigines, found that all were Rhesus positive, and confirmed this in the 1948 series of Simmons and Graydon.

Further investigations by Simmons et al. (1954) 1957, 1958a and 1958b) allowed them to state the probable Rhesus genetic frequencies as \( R_1 \) (CDe) = 0.6530; \( R_2 \) (cDE) = 0.1945; \( R_0 \) (cDe) = 0.1037; \( R_2 \) (CDE) = 0.0477; \( \lambda \) (Cde) = 0.001, and proved conclusively that all Australian aborigines were Rhesus positive. They also showed a gradation of gene
frequencies from north to south, the highest incidence of $R_1 (0.727)$ occurring in the Cape York peninsula, where Simmons (1957) considered it to be due to Melanesian influence, as New Guinea natives show an $R_1$ incidence of 0.945. Correspondingly, in the Cape York area, $R_2$ is lower.

The Western Desert aborigines closely correspond with other Western Australian natives, having a Rhesus group gene distribution according to Simmons (1957) of $R_1 = 0.6558$, $R_2 = 0.2073$, $R_0 = 0.0849$, $R_z = 0.0461$, $r = 0.0060$, also aborigines of the Western Desert showing phenotypes $R_z/R_z$ were not uncommon.

Many variants of the Rhesus group have been reported in the Western Desert region, and an incidence of 0.41% high grade and 0.18% low grade $D^u$ ($Rh O$) variants were noted by Simmons (1958). No $C^W$ variants have been noted within Australia, although reports of high and low grade $D^u$ variants in the other parts of Australia have been made by Sanger (1950, 1951). An important variant of the $E$-antigen is discussed by Vos and Kirk (1962) who discovered a naturally occurring anti-$E$ antibody in a Western Desert aborigine and demonstrated the antigen as a complex similar to the $C + C^W$ complex postulated by Race and Sanger (1956), the $E$ complex consisting of $E + E^t$. They also demonstrated
that, while both the homozygous and heterozygous modes of inheritance are widespread in all E positive Australian aborigines, (shown by the varying degree of reaction between the anti E\textsuperscript{t} and aborigines E positive cells), no such variation can be shown in the E positive Caucasian blood, all of these reacting vigorously with the anti E\textsuperscript{t}. Although no E\textsuperscript{u} variants have been reported in the Western Desert region, Sanger, Walsh and Kay (1951) found a few cases in the Northern Territory and Queensland. Vos (1960) has also tested for E\textsuperscript{u} variants in the Western Desert region and in other groups of Western Australian natives without finding any positive cases.

Partial deletion of some of the gene factors is not uncommon amongst aborigines, but to date only one blood showing complete deletion with the phenotype \texttt{---/-----} has been recorded. This was discovered in a healthy Western Desert aboriginal woman by Vos, Vos and Kirk (1961) and a complete absence of the d-like antigen confirmed by Levine et al. (1962). Although a similar case has more recently been reported in America, this blood does not fulfil the criteria of complete deletion, and is considered by Vos (1963) to show only partial suppression of the d-like antigen.

**THE P GROUP**

This system shows considerable local variation
between different groups of aborigines. Sanger et al. (1951), in a study of Bathurst Island natives, noted 34.7% were P positive, while Simmons et al. (1954) in a scattered series recorded 65% as being P positive. In a further series in Central Australia (1959) showed only 23% positive. Vos (1962) states that this is the average for the Western Desert natives. There is no literature recording any attempt to break down the P blood group system into further component parts in the aboriginal population.

THE LEWIS GROUP

The Lewis blood group system shows a reasonably even genetic distribution throughout all groups of Australian aborigines. Simmons (1958) estimated the variation at 6.9% to 7.3%, Le⁺ and that the mean incidence for 1836 Western Australian aborigines tested was 6.9% Le⁺.

THE LUTHERAN GROUP

The Lutheran blood group system distribution in Australian aborigines has been variously reported on by Simmons, Graydon and Semple (1954), Simmons (1958) and Vox (1960), the latter referring in particular to the Western Desert natives. All these authors state that all aborigines tested so far lacked the Lu(+) antigen.
THE KELL GROUP

This system has also been reported by Simmons (1958) and Vos (1960) and is identical to the Lutheran group in that all aborigines lack the K+ antigen.

THE DUFFY GROUP

The Duffy blood group system is the opposite of the preceding two in distribution according to Simmons (1958) and Vos (1960), all aborigines being Duffy positive.

THE DIEGO GROUP

The Diego blood group system has only been briefly investigated in aborigines of the Cape York peninsula and of Central Australia by Simmons (1957, 1962), while Vos (1967) has investigated some of the Western Desert aborigines. In none of these series has any evidence of the Di(+) antigen been found in full blood aboriginals.

THE KIDD GROUP

This system has a distribution in Australian aborigines which has been briefly investigated by Simmons (1962) when he cited a 61-69% distribution of the jk (A+) antigen. Vos (1964) is in agreement with this figure for the Western Desert aborigines. He also noted in his investigations that, of the Western Desert natives who are
Kidd negative, a large percentage have demonstrable anti-jka antibodies, and that the distribution of this is very much higher than is found in the Caucasian population.
Table 18.

Western Desert Aborigines.

Distribution of Blood Groups.

<table>
<thead>
<tr>
<th>System</th>
<th>Group</th>
<th>W.D. Aborigines</th>
<th>European</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABO.</td>
<td>A₁</td>
<td>56.9%</td>
<td>33.4%</td>
</tr>
<tr>
<td></td>
<td>A₂</td>
<td>0.0%</td>
<td>10.0%</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>0.0%</td>
<td>7.2%</td>
</tr>
<tr>
<td></td>
<td>AB</td>
<td>0.0%</td>
<td>3.0%</td>
</tr>
<tr>
<td></td>
<td>O</td>
<td>43.1%</td>
<td>46.4%</td>
</tr>
<tr>
<td>Rh.</td>
<td>Positive</td>
<td>100.0%</td>
<td>85.0%</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>0.0%</td>
<td>15.0%</td>
</tr>
<tr>
<td>MNS.</td>
<td>M</td>
<td>2.4%</td>
<td>28.7%</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>67.2%</td>
<td>23.9%</td>
</tr>
<tr>
<td></td>
<td>MN</td>
<td>30.4%</td>
<td>47.4%</td>
</tr>
<tr>
<td></td>
<td>S</td>
<td>0.0%</td>
<td>55.0%</td>
</tr>
<tr>
<td>Kell</td>
<td>Positive</td>
<td>0.0%</td>
<td>10.0%</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>100.0%</td>
<td>90.0%</td>
</tr>
<tr>
<td>Lewis</td>
<td>Positive</td>
<td>7.0%</td>
<td>20.5%</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>93.0%</td>
<td>79.5%</td>
</tr>
<tr>
<td>Lutheran</td>
<td>Positive</td>
<td>0.0%</td>
<td>8.0%</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>100.0%</td>
<td>92.0%</td>
</tr>
<tr>
<td>P</td>
<td>Positive</td>
<td>32.0%</td>
<td>71.4%</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>68.0%</td>
<td>28.6%</td>
</tr>
<tr>
<td>Duffy</td>
<td>Positive</td>
<td>100.0%</td>
<td>66.0%</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>0.0%</td>
<td>34.0%</td>
</tr>
<tr>
<td>Kidd</td>
<td>Positive</td>
<td>65.0%</td>
<td>77.0%</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>35.0%</td>
<td>23.0%</td>
</tr>
</tbody>
</table>

After various authors — see text.
OTHER GENETICALLY INHERITED CHARACTERISTICS
OF THE
WESTERN DESERT ABORIGINES
Other genetically determined characteristics which have been studied in aborigines are the distribution of secretors and tasters (P.T.C. tests), and the distribution and variations of the serum factors, the Gc and Gm protein factors, the Haptoglobins and the Transferrins.

**SECRETORS**

Secretors among aborigines, show, as do Caucasians, a close correlation between secretion and Lewis negative blood groups, a fact reported by Simmons, Semple and Gradton (1954). The percentage of secretors is similar to that of the European races, forming 75% of the total population, with a general aboriginal distribution.

A marked difference has been demonstrated between the Western Desert aborigines and Caucasian secretors by Vos (1960), who has stated that the aborigines, as opposed to the white population, secrete high titres of H-substance in relation to the A1 group, and Vos and Steinberg (1963) consider that a different genetic locus of A1 is present in the aborigine.

**TASTERS OF P.T.C.**

In the various groups of aborigines tested by Simmons et al (1954 and 1957), Tasters of P.T.C. form 50% of the population, showing an even distribution throughout the tested area. The Western Desert natives were not specifically
examined, but closely related tribes are included in this series, and it is probable that a similar distribution occurs in the Western Desert area.

**THE Gc FACTORS**

The Groups Specific Component of the alpha-globulins were investigated in Australian aborigines by Clive, et al (1962), who reported that apart from the major variants of Gc\(^1\) and Gc\(^2\) a completely new variant, Gc\(^{ab}\), was present in the aboriginal population. Further investigations by Kirk, Clive and Bearn (1963) demonstrated that, while the average distribution in aborigines of the various factors is Gc\(^1\) = 0.767, Gc\(^2\) = 0.194, Gc\(^{ab}\) = 0.038, these factors are not evenly distributed in the population. Gc\(^2\) has its highest incidence (19.4%) in the Cape Yorke area, and decreases towards the centre of Australia, to reach its lowest incidence of 4% in the Western Desert aborigines. Gc\(^{ab}\) shows a similar distribution, with a gene frequency of 0.067 in the Cape Yorke area, decreasing to 0.004 in the Western Desert area. As yet, none of the Gc variants "xy" or "Chippewa" have been noted in Australian aborigines.

**THE Gm FACTORS**

These have been studied in South-East Asia and in two groups of aborigines in Australia (Kimberley and the Western Desert groups) by Vos et al, (1963). Although the
three major variants $Gm^a$, $Gm^b$ and $Gm^x$ are present in the Kimberley group, $Gm^b$ is absent in the Western Desert group, and this group show a gene frequency of $Gm^a = 0.730$, $Gm^x = 0.270$, $Gm^b = 0.000$, $Gm^{ab} = 0.000$.

THE HAPTOGLOBIN SYSTEM

This system consists of two genetically determined variants $Hp^1$ and $Hp^2$, giving rise to homozygous ($Hp^1 Hp^1$, $Hp^2 Hp^2$) and heterozygous genotypes ($Hp^1 Hp^2$) were initially studied in Australian aborigines by Butz-Olsen (1958) who demonstrated that the gene frequency of $Hp^1$ increased from the coastal to the central regions, the $Hp^1$ rising from 0.46 in Cape Yorke to 0.63 in Central Australia. The Central Australian natives who adjoin the Western Desert natives, have a distribution of $Hp^1 Hp^1 = 40\%$, $Hp^1 Hp^2 = 47\%$ $Hp^2 Hp^2 = 13\%$. It is probable that a similar distribution exists amongst the Western Desert natives, though no figures have yet been obtained from this region. No Ahaptoglobinemic aborigines have been described, by either Butz-Olsen (1958) or by Horsfall and Smithies (1958) who confirmed the gene frequency rates.

THE TRANSFERRINS

This fraction of the beta-globulin component of the plasma proteins, was first realized to be genetically inherited by Smithies (1957), in his work on aboriginal
sera, in which he detected two major variants $Tf^c$ and $Tf^d$. These showed simple inheritance through two Alleomorphic genes and was further proven by the extended series of 120 Queensland aborigines examined by Horsfall and Smithies (1958). Smithies and Hillier (1959) considered the $Tf^d$ gene to be present in 10% of the Australian aboriginal population, but gave no regional distribution figures, and their results are only applicable to Queensland aborigines. Although eight variations of the Transferrins have been noted by Giblett et al (1959), only the original two, $Tf^C$ and $Tf^D$, (now known as $Tf^C$ and $Tf^D$), have been noted in aborigines, $Tf^d$ being restricted to negroes, and $Tf^b$ present in the Canadian white population. Kirk, Clive and Bearns, (1963) do however, state that there is a genetic gradation within the transferrin system, although they do not give any details.

SICKLE CELL TRAIT

Sickle Cell Trait in aborigines blood has been looked for by Horsfall and Lehmans (1953) and Simmons (1958), but to date no evidence has been found of the presence of this factor in any Australian aborigines, including a small group of Western Desert aborigines studied by Vos (1966).
THE BIOCHEMISTRY
OF THE
WESTERN DESERT ABORIGINES
Several aspects of the biochemistry of the blood of apparently normal aborigines have been investigated by various authorities, and some of these show significant variation from the normal range found in Europeans.

**THE PLASMA PROTEINS**

The plasma proteins were originally studied in the Western Desert aborigine by Curnow (1957) in an attempt to discover evidence of malnutrition in this group of aborigines. He observed, however, that there was a significant rise in the total protein level, mainly due to a rise in their gamma-globulin fraction with a slight rise in the albumen level. Later Beveridge (1967) in an extended survey of Western Australian aborigines confirmed these results, and gave the mean levels of the various plasma protein fractions in the aborigines as albumen = 3.6 (4.09), alpha-1 globulin = 0.934 (0.29), alpha-2 globulin = 0.91 (0.75), beta-globulin = 1.23 (0.93), gamma-globulin = 2.33 (1.15), total globulin = 4.82 (3.11), and total protein level = 8.43 (7.20) - the figures in brackets referring to European controls, and all measurements being in grams per 100 mls.

There is, therefore, a significant decrease in the albumen level, a significant increase in the alpha-1, alpha-2, and beta-globulin fractions, and a markedly significant increase in the gamma-globulin fraction. These result
in significant increases in both total globulin and total plasma protein levels. Wilkinson et al (1958) confirmed these observations in the Central and South Australian aborigines.

SERUM VITAMIN B\textsubscript{12}

Serum vitamin B\textsubscript{12} levels were first noted by Pitney and Davis (1957) to be significantly and consistently raised in the Western Desert aborigines. These aborigines show a mean serum level of vitamin B\textsubscript{12} of 751 ug. per ml. compared with the mean level in European controls of 456 ug. per ml. Pitney (1962) in a further series including other groups of aborigines in Western Australians showed that this is a constant feature, the mean levels of various groups ranging from 738 ug. per ml. in the northern Kimberley aborigines to 1032 ug. per ml. in the Hall's Creek region.

SERUM FOLATE

The only levels of folate described is in a small study of aborigines in the Western Desert area by Davis, Kelly and Byrne (1965) in which 54% of the males and 62% of the females had values below the accepted lower limit of normal for Europeans. The range in this series was 2.7 - 18.3 ug./ml.
SERUM CHOLESTEROL

Serum cholesterol is significantly lower in aborigines than in the European population. Charnock et al. (1959) compared tribal aborigines, detribalised aborigines, and European controls, and they demonstrated respective means of 212, 234 and 286 mg. per cc., using full blood Central Australian aborigines as the tribal group.

Riseborough et al. (1961) in a very small group (6) of Western Desert aborigines and who do not form an accurate comparison, as all had yaws and were probably showing some degree of malnutrition, had serum cholesterol levels of 95 to 140 mg. per cc.

SERUM CAROTENE

Serum carotene has only been recorded in the same group as above by Riseborough (1961), and the recorded range was 2-20 ug. per 100 ml., in these Western Desert aborigines, which is considerably lower than the average European range of 200-300 ug. per 100 ml. No other investigators have apparently studied these levels.

SERUM MAGNESIUM

Serum magnesium estimates were performed by Charnock et al. (1959) in Central Australia and these showed a mean level of 2.50 ± 0.43 mg./cc. with no significant variation
from European controls, although both groups varied with the locality. His European range was 1.34 - 3.82 mg. per 100 cc.

**SERUM CALCIUM**

Serum calcium estimations in a small group of Western Desert natives by Riseborough (1961) showed a range of 8.0 - 8.8 mg. per cc. The normal Europeans range in this area is 8.8 - 10.4 mg. per 100 ml.

**SERUM PHOSPHORUS**

Serum phosphorus levels estimated in the same group by the same author showed a range of 3.7 to 5.9 mg. per 100 cc. compared with the normal European range of 10 to 14.1 mg. per 100 cc.

**SERUM ALKALINE PHOSPHATASE**

Serum alkaline phosphatase was also estimated in the above group by Riseborough (1961), the range was 5.5 to 16.9 K.A. units, while the normal European range is 3 to 13 K.A. units.

**SERUM AMYLASE**

Serum amylase levels, again assayed in the same group of aborigines, range from 116 to 200 units per 100 cc., all lying within the accepted European range of 95 to 250 units.
Blood urea estimates in the same group of Western Desert natives by Riseborough (1961) show a range of 22 to 50 mg. per 100 cc. In the series (15 patients) I have examined in the Western Desert area and who were apparently healthy showed a range from 25 to 36 mg. per 100 cc., and it would appear that the aboriginal values should lie within the normal European range of 14.2 to 39.4 mg. per 100 cc.

Blood glucose estimations have not been previously reported in the full-blood Australian aborigines, but in a series of glucose/tolerance/tests I have performed with Western Desert aborigines (Table ), all were within the normal European range with no evidence of a low renal threshold, the mean fasting value being 84.15 mg. per 100 ml. and respective values of 133.33, 150.00, 127.50 and 90.83 mg. per 100 ml. for the half, one, one and a-half, and two hour values.
### Table 19.

**Western Desert Aborigines - Adults.**

**Glucose Tolerance Test.**

(6 clinically normal subjects)

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age</th>
<th>Fasting</th>
<th>30 min.</th>
<th>1 hr.</th>
<th>1 1/2 hr.</th>
<th>2 hr.</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td>23</td>
<td>85</td>
<td>125</td>
<td>140</td>
<td>130</td>
<td>85</td>
</tr>
<tr>
<td>M</td>
<td>31</td>
<td>80</td>
<td>140</td>
<td>165</td>
<td>130</td>
<td>95</td>
</tr>
<tr>
<td>M</td>
<td>34</td>
<td>85</td>
<td>130</td>
<td>160</td>
<td>125</td>
<td>90</td>
</tr>
<tr>
<td>M</td>
<td>28</td>
<td>80</td>
<td>125</td>
<td>145</td>
<td>135</td>
<td>90</td>
</tr>
<tr>
<td>F</td>
<td>33</td>
<td>90</td>
<td>140</td>
<td>155</td>
<td>135</td>
<td>100</td>
</tr>
<tr>
<td>F</td>
<td>37</td>
<td>85</td>
<td>140</td>
<td>145</td>
<td>110</td>
<td>85</td>
</tr>
</tbody>
</table>

Mean value 84.15 133.33 150.00 127.5 90.83

Test performed using "Dextrotest".

Personal series.
PHYSIOLOGICAL OBSERVATIONS

IN THE

WESTERN DESERT ABORIGINES
Physiological observations have been made on Australian aborigines by various researchers.

**PULSE RATE**

Pulse rate investigations by Ray (1927) in Central Australian aborigines showed a mean pulse rate in males of 94 beats per minute and 88 beats per minute in females.

In my five years clinical experience with the Western Desert aborigines, I have never noticed any significant difference in pulse rate between the aboriginal and the European populations of this area, either in health or disease.

**BLOOD PRESSURE**

Blood pressure estimations in various groups of aborigines have been conducted by Ray (1927) and Caseley Smith (1959) in Central Australia, and Abbie and Schroder (1960) and van Dongen et al (1962) in Arnhem Land. These authors note a significantly lower systolic and diastolic pressure in the female when compared with the male aborigine, and that both sexes have significantly lower systolic and diastolic pressures than the normal Caucasian levels.

The Western Desert natives correspond closely to this description, showing, in a series I have studied a mean adult average pressures of 112/72 mm. of Hg. in 20-29 year old male
Table 20.

Western Desert Aborigines.

Mean Blood Pressure in Relation to Age.

<table>
<thead>
<tr>
<th>Sex</th>
<th>No.</th>
<th>Age group</th>
<th>Mean age</th>
<th>Mean systolic</th>
<th>Mean diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>16</td>
<td>0-4</td>
<td>3.5</td>
<td>87.3</td>
<td>54.1</td>
</tr>
<tr>
<td></td>
<td>29</td>
<td>5-9</td>
<td>7.4</td>
<td>98.0</td>
<td>58.2</td>
</tr>
<tr>
<td></td>
<td>34</td>
<td>10-14</td>
<td>12.8</td>
<td>105.2</td>
<td>64.9</td>
</tr>
<tr>
<td></td>
<td>23</td>
<td>15-19</td>
<td>16.4</td>
<td>110.4</td>
<td>70.1</td>
</tr>
<tr>
<td></td>
<td>35</td>
<td>20-29</td>
<td>25 app.</td>
<td>112.6</td>
<td>72.4</td>
</tr>
<tr>
<td></td>
<td>38</td>
<td>30-39</td>
<td>35 &quot;</td>
<td>115.3</td>
<td>74.3</td>
</tr>
<tr>
<td></td>
<td>36</td>
<td>40-49</td>
<td>45 &quot;</td>
<td>121.5</td>
<td>80.8</td>
</tr>
<tr>
<td></td>
<td>28</td>
<td>50-59</td>
<td>55 &quot;</td>
<td>125.8</td>
<td>82.5</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>60-69</td>
<td>65 &quot;</td>
<td>128.0</td>
<td>86.8</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>70 +</td>
<td>75 &quot;</td>
<td>135.6</td>
<td>99.2</td>
</tr>
<tr>
<td>Female</td>
<td>25</td>
<td>0-4</td>
<td>3.2</td>
<td>86.9</td>
<td>53.2</td>
</tr>
<tr>
<td></td>
<td>33</td>
<td>5-9</td>
<td>7.8</td>
<td>96.6</td>
<td>56.4</td>
</tr>
<tr>
<td></td>
<td>26</td>
<td>10-14</td>
<td>12.7</td>
<td>99.6</td>
<td>63.8</td>
</tr>
<tr>
<td></td>
<td>27</td>
<td>15-19</td>
<td>17.1</td>
<td>103.5</td>
<td>69.7</td>
</tr>
<tr>
<td></td>
<td>23</td>
<td>20-29</td>
<td>25 app.</td>
<td>108.1</td>
<td>71.8</td>
</tr>
<tr>
<td></td>
<td>31</td>
<td>30-39</td>
<td>35 app.</td>
<td>109.4</td>
<td>72.1</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>40-49</td>
<td>45 &quot;</td>
<td>114.6</td>
<td>76.9</td>
</tr>
<tr>
<td></td>
<td>17</td>
<td>50-59</td>
<td>55 &quot;</td>
<td>118.3</td>
<td>78.3</td>
</tr>
<tr>
<td></td>
<td>22</td>
<td>60-69</td>
<td>65 &quot;</td>
<td>121.7</td>
<td>85.4</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>70 +</td>
<td>74 &quot;</td>
<td>129.6</td>
<td>89.5</td>
</tr>
</tbody>
</table>

Age in years, B.P. in mm.Hg.

Personal series.
age group, and 108/72 mm. of Hg. in the 20-29 year old female age group. These rise progressively to 135/92 and 129/85 mm. of Hg. in the 70 year old male and female groups respectively. The extended details of the systolic and diastolic mean blood pressures in males and female aborigines in various age groups in the Western Desert are presented in Table 19.

**VITAL CAPACITY**

Vital capacity of Central Australian aborigines was studied by Hay (1927) who found a range in adult males of 2,562 to 3,425 cc., but due to the difficulty occurred, in obtaining proper experimental conditions and co-operation, little significance can be placed on these results. No other results are available.

**BODY TEMPERATURE**

Body temperature estimated by Hay (1927) in the Central Australian aborigines was found to be 0.6°F higher than European controls, averaging 99.4°F in female subjects. However, Hicks et al (1938) found, in the same region, that aboriginal oral temperatures were lower than white controls, although not significantly so.

I observed no difference in oral temperatures in the Western Desert natives and the white population.
BASAL METABOLIC RATE AND OXYGEN CONSUMPTION TESTS

These were estimated by Hicks and O'Connor (1938) and they revealed no significant difference between aborigines and white controls. The same authors could find no significant changes in the skin circulation of the Central Australian aborigines with changes of environmental temperature.

There are no comparable reports in regard to the Western Desert aborigines.

CLINICAL TESTS

There are remarkably few reviews of the results of clinical tests conducted within the aboriginal population, and it is difficult to assess the range of response to these tests, and to define the parameters of normality.

GLUCOSE TOLERANCE TEST

As detailed previously, in a small series I studied in the Western Desert area, in apparently healthy aborigines, the response to 50 Gm. of oral glucose was within the normal European range, (Table 18) although both the rise and fall in blood sugar levels was relatively rapid.

These tests were performed on urbanised aborigines and it is not known what results would occur in the nomadic group, exposed to the same glucose stress. There was no
indication that the renal tolerance to glucose is any less in the Western Desert aborigine than in the Caucasian.

**CASONI TEST**

The Casoni test has been reported by Maigreith (1938) and Caseley Smith (1959) as having doubtful value in Central Australian aborigines, there being no correlation between hydatid disease, eosinophilia, and positive Casoni tests. They showed a higher proportion (40%) of positive reactors in central areas, despite a low incidence of hydatid disease. Warner et al (1957) in a similar area, using an elevated eosinophilia as a screening test, also conducted a series of Casoni tests and found that of 3 positive results none gave a positive complement fixation test.

In a small series in the Western Desert region of 16 aborigines tested, which were selected at random, 7 gave positive reactions to the Casoni test. In none of these patients could hydatid disease be confirmed either at that time or later. It appears that a large percentage of Western and Central Desert aborigines show positive Casoni reactions when no disease is present. A possible theory to explain this is advanced later in the discussion.

**MANTOUX TEST**

King et al (1951) in an extensive survey of Kimberley aborigines showed no difference in reaction to tuberchun testing between aborigines and white people.
DISCUSSION

OF THE

PHYSICAL CHARACTERISTICS

OF THE

WESTERN DESERT ABORIGINALS
INTRODUCTION

The physical characteristics of the Western Desert aborigines may be discussed from two separate aspects.

Firstly, they may be considered in relation to those of other groups of Australian aborigines, from which it may be possible to form some conclusions as to the racial origin of these people.

Secondly, they may be considered in relation to the accepted normal standards of Europeans in Australia, and from the various similarities or differences present it may be possible to determine whether which characteristics are environmentally or genetically determined, thus giving some insight into the possible changes which will occur in these factors with aboriginal assimilation into western civilization.
THE COMPARISON OF WESTERN DESERT AND OTHER ABORIGINAL GROUPS

From the physical description given of the Western Desert aborigines, it is apparent that they show a close resemblance to the Central Australian group which has been described by Campbell et al (1936), but that, in many ways, they differ from the aborigines of the Northern Territories described by Foelsche (1881) and Howells (1937); of the coastal areas of Western Australia described by Love (1936); and those of Queensland, described by MacIntosh (1951). In general, the latter three groups (Northern Territory, Queensland and coastal areas of Western Australia) form a very similar and homogeneous group whose major features have been summarised by Birdsell (1949) under his Carpenterian type, which corresponds to the Australoid race of most anthropologists.

From personal observation of the Western Desert natives, and from Campbell's (1936) description of the aborigines of the Central Desert, these two groups also show close similarities to each other, but they exhibit marked disparities from the peripheral groups.

Close examination of the physical features of the aborigines of the Central parts of Australia show three arbitrary but reasonably distinct groups:—
Western Desert Aborigines - Pigmentation.

Fair Hair and Light Skin.

Plate 6.
Firstly, there is a relatively large group forming approximately 33% of the total population who correspond closely with the peripheral aborigines, and who largely fulfil the criteria of Birdsell's Carpentaria type.

Secondly, there is a minor group of some 15% who are remarkable for their light pigmentation, less prominent brows, less developed supra-orbital ridges and less marked nasal root depression, with thin, straight noses and less obvious cephalic and limb-body indices when compared to the Carpenterian type. This group, in fact, shows body affinities with a primitive Caucasian type and closely corresponds to Birdsell's Murrayian group except that the nose, which has a high relief, is not as broadened as he described, and I have found no increased incidence of eruption of a fourth molar tooth in this group. The hair is also wavy or straight, and there is a tendency to baldness.

Thirdly, there is the major group of an intermediate type, showing some characteristics of both the so-called Carpenterian and Murrayian types to a greater or lesser degree, and who also show a smooth pattern of continuity between the two preceding extreme groups.

Further evidence of the physical differences between the coastal and the central aborigines is provided by the craniometric studies of Berry (1910), Hadlicka (1928), Wood-Jones (1929), Krogman (1933), and Wagner (1937) all of whom
emphasise the homogeneity of the peripheral aborigines, while Fenner (1939) demonstrated variations in the skull parameters between the coastal and central tribes in his particularly extensive analysis throughout the Australian continent.

These differences, in combination with the more recently discovered variations in gene frequencies of the blood groups and serum factors again suggest the possibility that the Australian aborigine may not be of uni-racial origin and it is from this aspect that we will compare the physical characteristics of the Western Desert and other aborigines.

In considering the possible origins of the Western Desert and therefore all the Australian aborigines, the following points must be born in mind:

(1) If the origin is uni-racial, then any differences between groups of aborigines must be explainable on the grounds of environment and/or random genetic drift;

(2) If the origin is multi-racial then any differences between groups, in order to substantiate such a hypothesis, must be greater than can be explained by environmental factors in conjunction with random genetic drift;

As there has never been any evidence of marked
Melanesian influence in the Australian aborigine, it can be assumed that the aborigine must have immigrated from Asia, across the Surat Straits in one or more waves, and as such would have inhabited initially the Northern Territory and then spread southwards along the eastern and western seabords to the southern part of the continent.

Expansion to the interior would have come later, either in the search for new hunting grounds or due to successive waves of immigrants and it is therefore in the Central Australian groups of aborigines, in comparison with the peripheral aborigines, that we would expect to see any evidence of a multiracial origin if such has occurred, provided that successive immigrant waves had been physically and genetically dissimilar. Nevertheless, we would not expect a sharp and distinctive difference to exist between the two groups, even in the circumstances of a bi-racial origin on the following three counts:-

(1) Traditionally the majority of aborigines, have occupied the coastal regions and hinterland of the Northern Territory, Queensland, Western Australia, New South Wales and South Australia, forming on the available evidence a homogenous group.

(2) If the above is accepted, then it must be assumed that if successive immigrant waves occupied the northern and peripheral parts of Australia any preceding race has been
submerged and evidence of it's presence will only be found, if any traces remain, in the more isolated areas.

(3) Among the most isolated areas is the Western Desert, and it is here that evidence would most likely be apparent. However, it is known from both the transmission of disease (see later) and the fact that such articles as pearl shell ornaments which were worn by these people, that they were not completely isolated from the peripheral aborigines and that some degree of social contact in the two groups must have existed. It therefore follows that the Western Desert aborigines would, if they are of the same origin as the peripheral aborigines, either be identical to them in characteristics or show only changes due to environmental factors, and that any changes due to random genetic drift would be decreased by reason of the continuing social contacts. If, however they are of a multiracial origin they would show differences from the more homogeneous peripheral groups, probably maintaining in some of its members the physical characteristics of the various racial groups. It is unlikely that all the pure racial characteristics would be present in the one individual, but that the group itself, would represent a range consisting of the typical features of successive immigrant races, which in the case of the last race must be the same as the peripheral group of aborigines.
In consideration of the external features alone, there is in the Western Desert aborigines some substantiation of a possible multi-racial origin of the Australian aborigines, and it was on this range of physical features in conjunction with the distribution of the ABO and A.M. blood groups that Birdsell (1941) based his hypothesis. However, such features as shape of nose and distribution of body hair, baldness, somato-typing craniometry and osteometry are of relatively little value in defining the origin of the Australian aborigines, as Abbie (1951 and 1969) noted that any differences that have been demonstrated are comparatively minor and such features are notably affected by environment and unknown genetical factors. Both of these authorities however, did not consider four clearly marked, genetically inherited, characteristics that are present in the external features of the Western Desert aborigines.

These factors are fair hair, straight hair, light body pigmentation and the incidence of arches in the fingerprints of this group, and it appears from the literature that these characteristics are confined to the Western Desert and Central Australian aborigines. From my own observation, it is only two of these four features are independent, as both straight and fair hair are frequently associated, although fair and wavy hair is not uncommon, but dark and straight hair is rare. Also, light hair and light body pigmentation are more commonly associated, than dark hair and light body.
pigmentation, while light hair and dark pigmentation is very rare. Despite the linkage between these three factors, all of which have virtually the same expression, in being neither dominant nor recessive, these traits could only be present in the Central Australian aborigines due to one or more of four mechanisms.

(a) **Environmental Factors.**
These people inhabit the hottest and most constantly exposed area in the continent and it is most unlikely that these conditions would either favour or induce light pigmentation of either the body or the hair. It is almost certain from experience of other racial groups that the reverse would be the more likely, and on other racial experience we can exclude the cause.

(b) **Random Genetic Drift.**
The concept would lead one to expect at least some expression of the trait to be seen in the peripheral aboriginal tribes, but there is no evidence that this is so. Furthermore, the incidence of these features is at its peak both north and south of the Great Victoria Desert. It diminishes towards the eastern and western boundaries through which contact between the north and south groups of the Western Desert aborigines is maintained and this, feature also opposes the concept of random genetic drift.

(c) **Mutation.**
Several factors negate this hypothesis. These are,
as a non-dominant factor it would be most unlikely to have attained the frequency which is present, and also as the frequency is at its highest in both the Warburton and Cundalee groups, between which there is no direct social contact, one would then have to postulate separate areas of identical mutation. McLarty (1957) has confirmed the lack of contact between these two groups and, furthermore, due to geographical reasons, any contacts would have to occur via the Kalgoorlie and Leonora groups in which the trait is less prevalent.

(d) **A Multi-Racial Origin.**

This concept proposes that the above features are inherited from a preceding race. By exclusion of other mechanisms this would appear to be the most likely, but this in itself does not give exact proof of this mechanism. Equally importantly the distribution of these features do not exclude this mechanism.

The surface indications of the Western Desert aborigines do therefore lend some support to Birdsell's theory, and more exact proof or disproof must be looked for in those factors which fulfil the postulates of Boyd (1940), as specific genetic markers and are known to be inherited in a simple mendelian manner. These markers may be found in the blood groups, the serum factors and in both secretor and
P.T.C. taster status.

Within the blood groups which have been studied, the Rhesus and Duffy groups are apparently homogeneous for all aborigines being 100% positive, in all groups. Similarly, the Kell, Lutheran, and Diego groups are also homogeneous, all three groups being 100% negative amongst the Australian aborigines. The homogeneity of these groups is however of little significance, as it has been estimated by Wiener (1942) that the Rh negative antigen has probably arisen in the European races within the past 2000 years and to date the Diego group has not been found outside negro populations.

Within the ABO, Rh, MNS, Lewis, P and Kidd groups, however, there are marked differences in the frequency of the gene distributions amongst the aborigines.

Most extensively, and also the first studied of these, is the ABO group. As previously stated, the B factor is only found in a limited area of Northern Queensland and is almost certainly of recent introduction. Therefore, from our present viewpoint, we need only consider the A and O factors, and of the A group only A1 is present. Birdsell and Boyd (1940) and later Wilson (1944) constructed isogene maps of the distribution of these two factors and showed that there is a marked increase in the frequency of the A1 towards the interior of Australia, attaining it's most consistently high levels, in the eastern part of the Western Desert and Central Australian
aborigines. The frequency of group A₁ increases from 0.084 in the Cape York peninsula to 0.343 in the Western Desert. In association with the ABO gene distribution in aborigines Vos (1962) in his secretor-status studies, has observed that, while the secretor-status is dependent on the Lewis factor (as in the white population) the titratable activity is related to the A₁ group (unlike the Caucasian where the association is with A₂). Moreover, as the secretor-status is followed from the coastal to the inland areas, a relatively abrupt change occurs between the Leonora and the Warburton groups, those of Leonora showing a frequency similar to the coastal aborigines of Western Australia, while those of the Warburton area, despite an increase in the genetic frequency of A₁ and a similar Lewis state to the other Australian aborigines, have a marked decrease in their secretor-status. A marked genetical difference is present therefore, between some of the Western Desert aborigines and the coastal group. In other groups tested by Vos, this same difference exists between the Northern Territory and Queensland aborigines, (who resemble the coastal aborigines of Western Australia) and the Central Australian aborigines (who are similar to the Warburton natives) in their secretor-status.

Although all aborigines are Rh positive, the phenotype distribution shows clinal variations. Of these, the most obvious is that of the R⁰ group which is maximal in the Northern Territory, (0.232) and minimal in the Western Desert
(0.084). \( R^r \) by comparison is of a more even distribution, while \( R^2 \) again shows an increase in the Western Desert and Central Australian groups with a frequency of 0.207. \( R^2 \) also reaches a high frequency of 0.461 in the Western Desert aborigines, a figure that is only exceeded by a localised group of aborigines. In this locality it is probably due to recent immigrant influence (corresponding to the B factor), from New Guinea natives.

The aboriginal genotype frequencies within the RL system may be more complex than previously realised. Vos and Kirk (1962), in a most elegant series of experiments, proved that the \( E \) factor is in fact complex of several separate factors, designated \( E, E^T, E^t \), which are inherited in a simple mendelian manner and occur in either the homozygous form \((E^T + E^T)\) or \((E^t + E^t)\) or the heterozygous states \((E^T + E^t)\). Using their specific and naturally occurring antibody anti-\( E^T \), they have estimated the gene frequency of \( E^T \) and \( E^t \) in various parts of Australia, showing that from it's highest frequency in the Western Desert (0.47) there is a rapid diminution of frequency (0.34) towards the Central Western region, and a further decrease northwards becoming non-existent (0.00) in the most northern coastal areas. Although Vos and Kirk discussed their results within the present concepts of the Rh system, they have ignored the anthropological significance of those findings which list for the first time a clearly identifiable, mendelian-inherited,
characteristic which is present in a restricted geographical area its related population of aborigines in Australia. This particular aspect will be discussed later.

Within the MNS system, no aborigine possess the S factor, and again a gradient of M and N gene frequency exists between the peripheral and central groups, M decreasing from 0.312 in the Northern Territory to 0.325 in the Western Desert; becoming lower in the more eastern part of this area and, in fact, reaching it's nadir in the Ernabella region of South Australia where the frequency is 0.060, this area being closely related to the Western Desert.

The Lewis system, from the few areas examined, is apparently evenly distributed throughout Australian aborigines, of whom approximately 7% are Lewis positive.

The P group is also evenly distributed, but again only few and scattered series are available.

Within the Kidd group there are no marked variations in the distribution factors, although evidence is given by Vos (1964) that within the Western Desert complex a large proportion of the Kidd negative population demonstrate anti-Jk(a) antibodies. Further investigations are required to assess the significance of this factor.

The serum factors show a considerable range in their
gene frequency. Within the Gc group, Gc$^1$ is highest in the Warburton Range area, (0.922) and Cundulee (0.977), and diminishes rapidly at the periphery to 0.717 at Edward River Mission. Reciprocal to Gc$^1$ is Gc$^2$ which has its highest value, (25%) in the Cape Yorke area and decreases to 4% in the Western Desert area, following a clinal distribution similar to the 0 factor in terms of spacial distribution, and this is paralleled by the Gc$^{ab}$ factor with a distribution from 5% at Cape Yorke to under 1% in the Western Desert, (0% at Warburton and 1% at Cundulee).

A smaller series of Gm factors studied, also shows a similar clinal distribution, Gm$^2$ decreasing from 0.730 in the Western Desert to 0.577 in the Kimberleys, the respective figures for Gm$^{ax}$ being 0.270 and 0.256. However, Gm$^{ab}$ shows the reverse, diminishing from 0.167 in the Kimberley area to 0.000 in the Western Desert area, while the Gm$^b$ factor is present in neither district.

The haptoglobin similarly show clinal decrease from the north to the south, and more noticeably in the interior. Hp$^2$ decreases from 0.54 in Cape Yorke to 0.37 in Central Australia, and Kirk and Lei (1961) confirm that in the transferrin system the Tf$^d$ gene decreases markedly as one moves peripherally from the high frequency present in the Western Desert area.
The gene frequencies of the known blood and serum factors therefore resolve themselves into two distinct patterns:

1. Those that are evenly spread throughout the entire population of aborigines, which in turn can be divided into sub-groups:

   (a) Where only one factor is present with a frequency of 1,000 as in the Rh (d) and Duffy factors; or Kell, 0.000 as in the Luther, and Diego factors, and also in the S factor of the MNS system.

   (b) Where two factors are present, with a relatively constant ratio in all aboriginal groups as in the Lewis and P groups.

2. Where frequency variations exist in the genotypes between relatively large groups of the aboriginal population. These include the remaining blood groups and also sub-divisions of the Rh groups, serum factors and the secretor mechanism. All these genes demonstrate a virtually identical pattern, although there are variations in degree. This pattern consists of homogeneous distribution of the individual gene in the aborigines of the Northern Territory, the north of Queensland and the north of Western Australia, with only minor frequency changes down the eastern and western seabords. This particular group I will refer to as the peripheral aborigines and they numerically form the majority of Australian aborigines. Within this group itself, some
aberrations are seen, particularly in the B factor of the ABO system and the R¹ of the Rh system in the Cape Yorke population. These local variations are undoubtedly due to recent contacts, and this has been substantiated by Simmons et al (1944 and 1954). When these particular gene frequencies are further plotted it is found that as one moves to the interior of the continent a gradient of change occurs which reaches it's acme or nadir in one of three groups, either the Western Desert aborigines, the Central Australian aborigines, or the northern tribes of South Australia, and these three groups form, on physical, cultural and genetical grounds, a closely related group which I will refer to as the Central aborigines. Within this general area, there are some minor groups situated in the north-easterly part (the Aranda and Luritga tribes) who genetically bear a close resemblance to the peripheral group and of whom Birdsell (1950) on the basis of cultural and physical characteristics, and more recently Kirk, Cleave and Bearn (1963) on genetical features, have provided reasonable proof that they are a recently immigrant group from the peripheral region of Australia, and on this evidence will be excluded from the Central Australian group.

These genes which show a gradient of frequency between the peripheral and central populations, can also be divided into two sub-patterns:-
(a) Those that are present in both populations, and include the ABO, the Rh sub-groups except for Et, the MN of the MNS system, the Gc and Gm serum factors, the haptoglobinins and transferrins, and also the secretor-status.

(b) Those factors which are present in only the central or the peripheral groups, that is the E^t, the Gc^ab, the Gm^ab factors.

If the factors in 2(a) are considered, there is an apparent wide range of variations in gene frequency, but if we plot the gradient of increase in the frequency from the peripheral to the central areas, then these systems in which we have statistically significant figures, that is those applying to relatively large population of each group, show a close correlation in scope of the gradient. This method is statistically applicable as the ABO and MNS systems each resolve themselves into two gene systems, B and S being inapplicable respectively in each system. Similarly, the haptoglobin group is limited to two genes, while within the GC system the Gc^1 and Gc^2 genes are the prominent ones, Gc^ab being aberrant, and it can be ignored at present. The GM system can be resolved into Gm^a and Gm^ax types, as Gm^ab is limited to the peripheral group and will also be considered later.

Only one gene of each of these systems needs to be
compared, as in most cases the other gene forms a reciprocal and is dependent in it's genetic expression on the frequency of it's co-gene. Moreover, the frequency gradient of R\textsuperscript{2} and R\textsuperscript{1} in the Rhesus system is closely related to the gradients of A, N, Gc\textsuperscript{1}, Gm\textsuperscript{a} and Hp\textsuperscript{1}, of which the latter five are virtually identical, and shown in Figure (4).

The science of genetical serology is based on the presumption that separate blood groups are independently inherited and are subject to a low rate of mutation and are not influenced by environment. In fact, a mild interdependence between the ABO and Rh systems has been postulated by Kirk (1961) and Mourant (1958) has also drawn attention to the role of blood groups in natural selection, although the particular aspects he discussed do not apply to the aborigines of Australia, as they are all rhesus positive.

If we accept that the ABO, Rh, MNS, Gc, Gm and Hp factors are independently inherited in the Australian aborigine and also obey the laws of Mendelian inheritance, then the almost identical changes in the genetic gradient in these factors from the peripheral to the central regions must be due to a single factor, which has influenced each of the separate frequencies to the same relative extent. The only possible mechanisms concerned may be that of isolation of a small population and resultant genetic drift, or alternatively that the Australian native population is not homogeneous in
origin, and that we are sampling a mixture of dissimilar ethnic groups.

Isolation of a small group has been constantly quoted by such authorities as Abbie (1951) and Simmons (1962) to account for the variations found in individual gene frequencies. The concept of genetic drift in a limited population has become a convenient receptacle for depositing a large number of unexplained genetic patterns and is certainly used loosely without sufficient thought being given to what is entailed by the term and without being sure that the term is applicable to the population to which it is applied. The concept itself entails such variable factors as the degree of inter-marriage and the deletion of unfavourable, or the increase of particularly favourable gene complexes, the size of the population. It also includes isolation from outside contacts, apart from isolation in a purely geographical sense. If the Western Desert aborigines in particular, and the Central Australian natives in general, are considered from these separate aspects, then it is true to say that they form the most isolated and least accessible group. The term is, however, relative, and there is abundant evidence from the spread of diseases such as small-pox (subsequently), and the acquisition of artifacts that can only be obtained from the coastal regions of Australia (see previously) that the isolation is much less than is generally considered, and that reasonably frequent social contacts have existed between both the coastal and
peripheral groups. In addition, corroborres are attended by widely separated groups, and, within the Warburton Ranges area contacts are frequent with aborigines from the more coastal regions of Western Australia and there is also a constant flux of movement between Warburton Range aborigines and those of the Eastern Central groups. Moreover, these social contacts are not necessarily transient meetings but due to the complex hospitality laws of the tribes frequently result in genetic interchange. Furthermore, the marriage laws and their strict application to tribal (totemic) rather than blood relationships also tend to prevent marked inbreeding, and within the genetic groups already considered, there is no evidence of gene-complex incompatibility. Therefore, none of these factors, either singly or in combination, are sufficient to account for the variation in gene frequency distribution between the peripheral and central groups of aborigines.

Neither can isolation be implicated as examination of the Western Desert group reveals from the aspect of social contact, considerably less movement between the Warburton-Laverton-Leonora complex and the Zanthus-Cundulee complex than exists between these individual complexes and their peripheral neighbours. However, despite the relative isolation between these sub-groups of the Western Desert natives, there is no marked difference in their genetic constitution. Further proof of the lack of true isolation of the central from the peripheral groups lies in the observation that the genetic
gradients in the factors that have been extensively studied, are generally smooth, while the genetic variation due to isolation should show an abrupt change and not the gently changing isogenic pattern at present seen.

The only possible explanation to satisfy the above facts are that two distinct populations are being sampled, of which the peripheral group is relatively homogeneous, but is extending in a decreasing frequency into the central area, whose original population were of a different genetic constitution quantitatively rather than qualitatively.

This interpretation of the heterogeneous origin of the Australian aborigines is further confirmed by the factors in 2(b), that is, those genetically inherited characteristics which are largely or entirely restricted to either the peripheral or central groups. These include the $E^t$, $Gc^{ab}$, and $Gm^{ab}$ factors.

The $E^t$ factor is at it's highest frequency in the Western Desert area, and the frequency decreases towards the eastern areas. The high frequency therefore observed in the widely separated Warburton and Cundulee areas, and the low frequency in the Leonora-Kalgoorlie region argues against the possibility this factor has arisen by within the time that these regions have been inhabited, unless one accepts the coincidence of spontaneous mutation in two separate areas.

If this factor has been introduced from another region, then
we must account for the identical and marked increase in the frequency of the factor in both the Warburton and Cundulee areas with their lack of contact with each other, compared with the more peripheral regions. The only explanation is that the $E^t$ factor was introduced to both the Warburton and Cundulee aboriginal groups at the same time and in the same frequency. This would indicate a common parent stock which has later emmigrated to these separate areas in which the expression of the gene has remained unchanged, and that this parent stock is different from the parent stock of the coastal aborigines in which this factor has not been yet discovered, although there is some expression of the trait in the intermediate groups between the coastal and central regions.

The argument that the factor $E^t$ may have been initially present in the parent stock of the peripheral groups of aborigines and has subsequently been eliminated is unlikely, as the much more frequent social contacts and genetic mixing make both the mechanisms of random genetic drift and group isolation untenable. Similarly deletion due to an "anti-$E^t$" cannot be upheld as such an antibody has not been demonstrated, while the only naturally occurring "anti-$E^t$" has been found in an aborigine of the Western Desert region and this appears to have no "in vivo" activity, according to Vos (1962).
The only explanation which fits the distribution of the $E^t$ factor is that it was present in the parent stock of the Central Australian aborigines, and not in the parent stock of the peripheral aborigines, who are therefore genetically dissimilar.

Further evidence of the dissimilarity of the central and peripheral aborigines is provided by the distribution of the $G^a_{ab}$ and the $G^a_{ab}$ serum factors, both of which show decreasing frequency from the coast to the interior. While $G^a_{ab}$ has failed to penetrate the central regions, $G^a_{ab}$ has, but only in the Cundulee area where the frequency is 1.8%, while it is absent from the Warburton area. This further emphasises the lack of contact between these two groups of Western Desert aborigines.

As the $G^a_{ab}$ factor is not present outside Australia it must have arisen either in the coastal aborigines (where it has its highest incidence) by mutation or been introduced in a relatively low frequency in one of the immigrant waves. The relatively high frequency of $G^a_{ab}$ in South-east Asia could account for the introduction of this gene at a later date into the aboriginal population from outside contacts, but the relatively high overall frequency in coastal aborigines would more likely support its introduction with the parent stock, rather than a later innovation.

The only explanation which accounts for the varying
gene frequencies between the central and peripheral groups of aborigines, is that peripheral group are a homogeneous immigrant race which have invaded the north of Australia across the Surat Straits, have expanded down the coast-lines and penetrated to the interior, and that a preceding and genetically dissimilar race have been pushed progressively into the interior and been largely assimilated by the more recent race. However, traces of this preceding race can be identified in the most isolated communities in the Western Desert and Central Desert region.

CONCLUSIONS

The preceding analysis of the physical features and genetic characteristics of the blood groups and serum factors of the Australian aborigines prove their hybrid origin, and for the first time gives confirmation to the general outline of Birdsell's hypothesis of their origin, and that hybrid origin can be adequately explained on the basis of two successive immigrant waves who correspond closely with Birdsell's Carpenterian and Murrayan types. Within the areas considered, there is no evidence of an earlier wave that would agree with his Barrenian type.

It is also possible to postulate the probable genetic characteristics of these races, both of whom lack the B factor of the ABO system, the Rh negative factor, the S factor of the MNS system, and the Kell, Lutheran and Diego
antigens.

The Carpenterian group are the equivalent of the previously described peripheral group, and are seen in their purest form in the natives of Arnhem Land, whose physical characteristics have been well described by Howells (1937) and Birdsell (1949) and their genetic constitution has already been noted. They have also extended into the interior in decreasing numbers, where they are mixed with the Murrayian type, which is characterised by light pigmentation of the body; fair, straight or mildly waved, hair; with a higher incidence of arches in their dermatoglyphics; with a tendency to baldness, and a high relief and relatively broad nose (but not as broad as Birdsell suggests); with a stocky build and less emphasised cranial indices than the Carpenterians.

The genetic characteristics of the blood of the initial race was probably homozygous A¹, and homozygous N in the MNS system, the acquisition of other factors in these groups resulting from subsequent peripheral admixture. In the Rhesus group, R¹ and R² were significantly more frequent, and all the E group were probably also homozygous, E+Et/E+Et. However, the P and Lewis factors were approximately the same as the later immigrants, while the Gmab and Gcab factors were completely lacking, but they were also probably homozygous for both the Gmα and Gcα factors, while the Hp¹ frequency was significantly higher than in the succeeding
There is also evidence that the secretor-status was markedly different, and that transferrin "d" was dominant.

In general the present peripheral groups of Australian aborigines correspond closely to Birdsell's Carpenterian group which are a separate racial entity and analagons to the "Australoid Race." The evidence available supports that a preceding racial group, of a primitive "Caucasoid" type was present prior to the immigration of the Australoid Race, and that traces this separate racial group can be seen in the Western Desert aborigines.
THE COMPARISON OF THE WESTERN DESERT ABORIGINES AND THE WHITE POPULATION OF AUSTRALIA

The physical characteristics of the Western Desert aborigines, as detailed previously differ markedly in many respects from the accepted standards of the white population of Australia, and are worthy of individual discussion.

THE PHYSICAL FEATURES

The body measurements, so exactly calculated by anthropologists, are probably of little significance. The average height and weight of both sexes and at all ages are significantly lower than those found in the white population, but these are probably due to nutritional and environmental factors.

This is shown by the increased mean heights and weights which occur in the more settled groups of aborigines. Therefore, while it is useful to know the average at present, it must be realized that this is changing and will continue to change with increasing assimilation.

What is unknown at present, is what the optimum heights and weights for this group of people are, and these facts can only be discovered by constantly observing the effects of assimilation and the changes that will occur. At present, there is no evidence to suggest that the optimum of these measurements in the Western Desert aborigine should
not be in the vicinity of the optimum for the white population.

Likewise, the various body and cranial indices can be expected to undergo changes with assimilation. There is no statistical proof at present, but I have the impression and there is a trend that with the better standards of nutrition supplied to the children at certain missions, they are displaying a heavier musculature; they are generally taller than their more nomadic contemporaries; and they more closely approximate to the European body form, with an increased breadth of shoulder and less marked differences in the trunk, limb, and girth indices.

It has already been proven that the platycnemia and platymeria demonstrated by the aborigines is environmental in origin, while the anterior bowing of the tibia is pathogenic. Similarly, cranial dimensions have been shown in other primitive regions to be considerably influenced by nutrition, and there is no reason for assuming that the same may not occur in both the Western Desert and other aborigines.

Therefore, while much emphasis is laid upon the normal values of body measurement in the aborigine, and the degrees by which they differ from the normal Caucasian standards, it is important to remember that most of these differences are due to environmental, (nutritional, and pathogenic) influences, and will not be constant. Although the differences will probably decrease with assimilation and environmental changes,
as yet we have no knowledge of what the eventual norm will be, or more importantly, what will constitute the optimum standard for these people.

CHARACTERISTICS OF THE BLOOD

There are well documented differences in haematology between the aborigines of the Western Desert and the white population. In the former, the total red cell count and the total haemoglobin are remarkable for their consistently high level, both in the Western Desert and Central Desert aborigines. The findings of Caseley Smith (1958) that the cells of the Central Australian aborigines are significantly more spherical than those of white controls has not yet been confirmed in other regions, and we are not as yet in a position to evaluate this result.

The high haemoglobin levels of the Western Desert and Central Australian aborigines have been confirmed by Caseley Smith (1958), Davidson (1957), and Pitney (1957), and my findings are similar. These have been used by the above authors as an index of nutritional status of the population and, from the results evaluated, there is no evidence of malnutrition in this area. I am of the opinion that the problem is more complex. These high levels obtain in all groups of the Western Desert, both male and female, in all adult age groups, regardless of whether the females are pregnant, lactating or post-menopausal. There is also no significant
alteration in the haemoglobin values between the predominantly nomadic and the partially settled groups, despite the fact that the former probably have an adequate iron intake in their normal diet, while the latter who exist largely on tea, damper and other carbohydrates, with only occasional protein in the form of meat and almost never any vegetables, have a diet which by western standards is very poor. In addition, both adult series suffer from chronic blood loss; the females from the physiological losses due to menstruation, pregnancy and prolonged lactation; and the males from frequent blood letting ceremonies when considerable quantities may be lost.

The white population on a similarly poor diet to many of the settled aborigines and with the same degree of chronic iron loss, would almost certainly become anaemic. Therefore, a normal haemoglobin, for the Western Desert aborigines, is only indicative of a satisfactory iron absorption and that this does not necessarily mean, in my opinion, that their general state of nutrition or diet is satisfactory. It also indicates that the aborigine is capable of maintaining an adequate iron balance in circumstances impossible for persons of European extraction. As the balance can obviously be maintained on a lower iron intake, then they must possess a more efficient method of absorbing it than the white population, either due to more dietary iron being made available; by absorption; or, alternatively, more efficient duodenal and upper small bowel mucosa iron transfer mechanism.
There is no evidence on clinical grounds of increased iron stores in the body, and recovery from acute blood loss is the same as in the white population, while short term acute illnesses, as are seen particularly in children, can rapidly precipitate anaemia. Moreover, the chronic iron deficient anaemia which follows acute enteritis is usually associated with duodenal mucosal changes. It would appear likely from this evidence that increased iron absorption in the Western Desert aborigines is associated with a more effective mucosal transfer system, but further study is required to determine the exact mechanism.

In the white cell series, there are two marked differences between the Western Desert aborigine and the white population. These are the increased number of neutrophils and eosinophils, both of which are reflected in a higher total white cell count.

The rise in the neutrophil fraction is also accompanied by a shift to the left in the Arnot count and it is probable, as both Pitney (1957) and Caseley Smith (1957) suggested, that the principle cause of the leukocytosis is infection, as chronic respiratory tract disease is extremely common in the aborigine.

The eosinophilia, is more difficult to assess. It has generally been regarded as due to parasitic infestation, particularly due to hydatid disease, trichiniasis and
intestinal worms, and is commonly seen in primitive communities as noted by Pitney (1957). This is true of the peripheral group of aborigines, where all these diseases are relatively common. However, in the Western Desert area, in my experience, parasitic infestation is extremely rare, and Davidson (1960) has agreed with this view. Furthermore, Allergic states are unknown in the Western Desert aborigine, and on this evidence it would appear that some more subtle and unexplained factor is the cause of the eosinophilia.

The possibilities could be an, as yet unidentified, parasite which is endemic, although this is unlikely as several medical surveys have been conducted in this area; an unrecognized state of hypersensitivity; or it may be a genetically determined characteristic. Whatever the cause, it is worthy of further investigation, as the facts at present suggest that there is a fundamental difference in the cause of eosinophilia in the Western Desert aborigines as compared to the white population.

BIOCHEMICAL DIFFERENCES

Biochemical tests show significant variation between the aboriginal and the white population, especially in the plasma protein, the serum vitamin B₁₂, and the plasma cholesterol levels.

The total plasma protein level is significantly
increased in the aborigine, due to an increase in the gamma-globulin fraction, and a lesser increase in the alpha- and beta-globulin fractions. Curnow (1957) in his investigations of the Western Desert area, and Beveridge (1963) in the north-west of Western Australia concluded that the rise was due to chronic infection, but it must be noted, that in other primitive communities, similar initial levels have not been reduced to completely normal white population equivalents merely by improving the environment, as Long et al (1956) have shown in relation to the assimilation of negroes in America. It is probable that factors other that the nutritional state and chronic infection may determine the increased plasma protein levels, and that some, as yet undetermined, racial factor also exerites a controlling influence.

The serum vitamin B\textsuperscript{12} levels have been extensively studied by Pitney (1957, 1962), who concluded that the high serum levels were due to increased binding of the vitamin to plasma proteins, and that the high levels therefore reflect the state of the plasma protein fraction to which the binding occurred, rather than the total body content of the vitamins. Pitney correlated the serum vitamin B\textsuperscript{12} level with the alpha-2 globulin fraction, and noted this was also high in the aboriginals of the Blackstone area in the Western Desert who had a marked eosinophilia. Molliin and Ross (1955) studying serum vitamin B\textsubscript{12} levels in the European race, found a close correlation between high levels and the myelo-proli-
ferative diseases. More recently, Scopa (1964 - personal comm.) has noted a significant increase in the plasma cells in bone marrow studies in aborigines. The correlation between these factors is at present impossible to assess, but it is possible that a racial characteristic determines both the eosinophilia and the plasma-cytosis in aborigines, these in turn being related to the production of a particular fraction of the alpha-globulin complex which binds the serum vitamin B\textsuperscript{12}. According to Pitney (1957), free vitamin B\textsuperscript{12} is not significantly raised in the Western Desert population.

Further research on this aspect is necessary for the final elucidation of the problem, and at present it cannot be assumed that a high vitamin B\textsuperscript{12} serum assay is necessarily indicative of good vitamin B\textsuperscript{12} body reserves, in the Western Desert aborigine.

The serum cholesterol level is significantly lower in the aboriginal population than in white controls, and again the difference cannot be explained on a nutritional basis only, as assimilation studies by Charnock et al (1959) have shown that, although the average level rises, it still remains significantly lower than in the white controls. What other factors are involved are unknown at present, but there may be some alteration in cholesterol metabolism in the aborigine which makes him particularly liable to
develop cholithiasis (see later) when on a western diet.

ANTIBODY REACTIONS

The Casoni test still presents an enigma in the Western Desert and Central Australian aborigines, as Caseley-Smith (1959) has demonstrated there is no correlation in the latter region between a high eosinophilic count and a positive Casoni test. Further, Warner et al (1957), showed, that, in a series of aborigines with positive Casoni tests, none demonstrated a positive complement fixation test. I have also shown that a high percentage of the aborigines of the Western Desert area are positive reactors to this test, and there is close correlation between by series and that of Caseley-Smith. I have, moreover, never seen any evidence of hydatid disease in this region, and Davidson (1960) confirms this. It therefore follows that a high percentage of aborigines in the Central Australian area demonstrate a hyper-sensitivity to hydatid fluid, which is known to contain a "P-like" antigen, according to Race and Sanger (1962). These aborigines who are Casoni positive must therefore possess an anti-"P-like" antibody which reacts with the "P-like" antigen of hydatid fluid under conditions of skin testing, but not in the complement fixation test, and this antibody must therefore lack the specificity of a direct antibody to the antigen present in hydatid fluid, but be related to the "P-like antibody". It would therefore appear that the Western
Desert and Central Australian aborigines possess a "P-like antibody" which is not present in the white population. The most probably source of the antibody would be in the P blood group system, and it is possible that the Western Desert aborigines possess a complex P group, similar to the E complex previously described within the Rhesus system. Further studies of the P blood group system in the aboriginal population is necessary to prove or disprove this theory. If such a complex were to be substantiated, then further evidence of a multi-racial origin of the Australian aborigines might be discovered. At present it appears that this clinical, and presumably genetic, reaction to the Casoni Test occurs only in the Central group of aborigines.
INTRODUCTION

While working among the Western Desert aborigines, I was impressed by the different pattern of disease which affects this group of people in comparison with the local Caucasian population and while many of the differences can be explained on the basis of different environmental conditions, lack of sanitation and poor hygiene, there also appeared to be a distinct racial difference.

I have based the format of this section on Cecil and Loeb's textbook of Medicine, (1951), which was my standard textbook during the time the field work was carried out. This format may appear to give undue length to this part of the dissertation but it has the advantage of allowing the recording of both positive and negative observations on the frequency of disease in the Western Desert aborigines. I have also included under each individual disease, such references as are available regarding the frequency of the disease not only in Western Desert aborigines but also in other groups of Australian aborigines, in order to establish whether or not significant variations occur between different groups of Australian aborigines, and also between Australian aborigines in general and the Caucasian population.

One of the problems encountered in this section is the poor annotation of disease among Australian aborigines and in
order to gain a general picture, many old and possibly unreliable references must be considered. Part of this problem was the difficulty of establishing an accurate diagnosis under the conditions in which most aborigines were investigated and treated, as few ancillary aids were available to establish the diagnosis.

A particular problem in attempting to analyse the available literature on aboriginal disease is trying to establish whether or not the references referred to full-blood aboriginals only or include part-castes. My own observations have been restricted to the full-blood aborigines of the Western Desert region and as far as possible I have tried to exclude references which include part-caste data.

The difficulty in gaining reliable information is likely to continue as long as there is no necessity in Australia to denote the race of the person in either notifiable or non-notifiable diseases, and as long as different States have different definitions of what constitutes an aborigine. e.g. The Native Welfare Department of Western Australia includes all persons of more than one-eighth part aboriginal blood. In the series I present here on the Western Desert aborigines, the judgment as to whether the individual is full caste or not is a combination of my own, Matron Corner's (who knows all and is related to many of the aborigines) and to the genealogical surveys conducted by Gerrard and Del Ves during
their serological studies in the Western Desert area.
CONGENITAL ABNORMALITIES
During the five years in the Western Desert region I observed 4 congenital anomalies. These were, a young adult male with a congenital dislocation of the left hip; a 3 year old child with cleft palate; a 5 year old child with conical cornea, and a 17 year old male with congenital heart disease, which was diagnosed as an atrial septal defect.

There are many reports in the literature of congenital anomalies in other groups of Australian aborigines, although their apparent frequency has almost certainly been exaggerated due to the ease with which they are diagnosed, as most of these are superficial, skeletal abnormalities. Various deformities recorded are those of a boy in South Australia with webbed and deformed fingers, noted by Ramsay Smith (1906); a case of bi-lateral webbed hands and one of pseudo-hermaphrodisism in a 16 year old person, both of these recorded by Brianl and Holmes (1915) in the Bathurst Island region. An aboriginal man in South Australia with polydactyly of both the upper and lower limbs. His two brothers and his sister were also stated to have the same deformity by Helms (1896) during the Elder exploring expedition. Cleland (1928) noted a specimen of what he considered to be congenital diverticulum of the stomach of an aboriginal in the Adelaide pathology museum and also cites Leighton Jones of Darwin as having seen several pseudo-hermaphrodites at Goulburn Island, and Illingworth of Taroom, Queensland, as having observed a 7 year old hermaphrodite; an
year old girl with polydactyly; and an adult male with congenital dislocation of the hip.

Basedow (1932) noted a case of cleft palate without hare lip among the natives of South Australia and also several infants with umbilical hernia and an adult whom he described as a congenital eunuch which from his description, is probably a case of bilateral undescended testicles.

Love (1936) observed a native adult with a cleft palate and hare lip in the north of Western Australia, while Gray and Cleland (1934) recorded what they described as a man with undeveloped genitalia in Central Australia. Black and Cleland (1938) make note of a male eunuch of 23 years of age, who they say may have bilateral undescended testicles or possibly had them taumatically avulsed. More recently Foro (1942) recorded three cases of pseudo-hermaphrodites and two others with congenital sexual abnormalities of which he does not give any specific description, but all of whom he says were related and lived on Bathurst Island. Crotty and Webb (1960) in their study of mortality amongst aborigines in the Northern Territory observed two cases of congenital heart disease, two of hydrocephalus and one of imperforate anus. More recently Buntine (1969) has described what appears to be true albinism in a full blood Australian aborigine and Malker (1969) has commented on the difficulties of someone with this abnormality
surviving in the natural Australian environment.

It is probable that both major and minor congenital abnormalities are not uncommon in the Australian aboriginal. However, those with major developmental defects are unlikely to survive under the normal nomadic conditions. It is also apparent that children with malformations are neither shunned nor destroyed because of them, and that in fact considerable tribal help is given to these unfortunate individuals.
TRAUMATIC DISEASE
Disease due to trauma accounts for approximately 5% of all patients treated in the Western Desert area. The types of trauma seen fall into three major groups - which are lacerations, burns and fractures.

**Lacerations.** In the Western Desert area, all types of lacerations are common. They normally heal quickly and only infrequently develop secondary infection if suturing is not required. Sepsis is common in those wounds sutured and dressed, unless they are closely supervised by medical staff. I disagree with Cleland (1928) who stated that aborigines heal more quickly and sepsis is less common than in the European race. I have noticed no difference when both races are treated under identical conditions, but have noted that secondary infection is more common in aborigines when the wounds are sutured and are not frequently redressed. However, I must agree that aborigines with minor lacerations which they themselves have treated initially with wood ash and earth, if left undisturbed and if they do not require initial suturing, then fairly rapid healing with a low frequency of secondary infection occurs.

Lacerations in the Western Desert result from three major causes.

The first of these is due to mulga wood splinters which are common in the bush. These are particularly sharp
and brittle branches of the mulga tree which may be still attached to the tree or fallen to the ground. The common sites of penetration are the foot, the leg and more rarely, the upper limbs, the torso, or the eye. These sharp dry sticks penetrate extremely deeply, they invariably break and shatter, and the wound cannot be adequately cleaned unless it is widely opened to its full depth and all splinters removed. Secondary infection is common and deeply placed splinters are the most frequent causes of chronically discharging wounds. Although the dorsum of the foot is frequently lacerated it is relatively seldom that the sole is penetrated due to the thick rubbery corium developed by the aborigines.

The second type of injury is that due to spear wounds which as previously described are normally restricted to the upper and outer aspects of the thigh and are usually clean. The aborigines themselves are experts at removing the spear without shattering it and relatively seldom are foreign bodies left in situ. In most cases the type of spear used for these affrays are the non-barbed variety, so that they can if necessary be broken off and pushed straight through the skin. Of 12 patients I have treated in this category, all have healed rapidly and without infection, and unless wide lacerations have occurred it is my opinion that these wounds are better left unsutured. Problems do however, arise with spear wounds that have missed their appointed mark, when generally some more vulnerable part of the body is hit. The
most dangerous and common error is for the spear to strike the inside of the thigh severing the femoral artery. I have seen one death result from this and to my knowledge a total of three deaths have occurred from this cause during the past ten years in the Western Desert area. I have never seen a spear strike above the pelvis, but I have seen two cases in which the spear struck bone in the lower limb and shattered. In one, the fragments were wedged between the left tibia and fibula and had to be surgically removed, and in another, the spear entered the right ankle joint and shattered. This also required exploration and removal of fragments. Both cases healed satisfactorily with no resultant disability.

The third major type of laceration encountered in the Western Desert aborigines are scalp lacerations generally due to domestic arguments, being inflicted either by nulla nullas between the women as described earlier or by either the men or the women hitting each other either with nulla nullas or stones, or due to laceration of the scalp by stones in displays of grief. These lacerations heal with remarkable rapidity. Infection of the scalp is uncommon in my experience.

**Burns.** Burns are extremely common in the Western Desert aborigines and these occur principally amongst children in the 3 to 6 year old age group and in the elderly. In the former group they are usually due to the child falling in the
fire while in the older adult group they are due to rolling into the fire at night while asleep or drunk. Burns most frequently occur in the lower limbs, but are not uncommonly encountered in both the upper limbs and on the trunk, particularly the abdomen. All degrees of burns have been seen in this area, and they invariably become secondarily infected unless early hospital treatment is instituted. Later contracture scars, particularly of the hands and knees are not uncommonly seen in those burns which have not had adequate treatment. Corneal burns are also relatively frequent but will be discussed under diseases of the eye.

The relatively high incidence of burns also occurs in other aboriginal groups as noticed by Cleland (1928), Black and Cleland (1938) and Basedow (1919) and (1932) among the South Australian aborigines.

Fractures. In the Western Desert region, fractures are remarkably uncommon amongst the nomadic natives, but are much more frequent among the station employees, where their work of stock herding and horse breaking increases the incidence. In the nomadic aborigine, I have never seen a fracture of the lower limb, although fractures of the upper limb are not infrequent but are usually restricted to women as a result of being hit by a nulla nulla when usually both radius and ulna are fractured, although occasionally only one of these bones, and much less frequently the humerus is
broken. In aborigines employed in stock work, both upper and lower limb fractures have been noted. They do not seem to appear as frequently as amongst the Caucasian population.

Fractures of other sites e.g. phalanges, seem to appear with the same incidence as the white population. Fractures of the skull are extremely rare in my experience amongst the Western Desert aborigines despite many obvious severe contusions to the skull. Among the older inhabitants of the Western Desert region the results of previous mal-union are not uncommon, as a number of women have been seen with obvious bony deformity and angulation at the site of an old fracture, and one elderly aboriginal woman from the Warburton Ranges has a false joint in the middle of the left fore-arm. I have also noted three cases of cross-union between radius and ulna resulting in limitation of pronation and supination.

The only reference to fractures in aborigines in other regions is that by Dinning (1949) who has reported a number of fractures and healed fractures in museum specimens of aboriginal skeletons.

It would appear that trauma in general is a not uncommon cause of aboriginal mortality as Crotty and Webb (1960) reported this as the cause of death of 17 of the 175 autopsies they performed in Northern Territory aboriginals. In all cases the origin of the trauma was either due to accident or inflicted in brawls or fights.
Infectious disease is the most common cause of morbidity and mortality in the Western Desert aborigines, particularly in the younger age groups and this pattern is apparently reproduced in all aboriginal groups in Australia. Major differences in this pattern do however, exist between one aboriginal group and another largely on a geographical basis and also between the Caucasian and aboriginal population.

These diseases will be presented by consideration of each of the individual causative agents.
VIRAL INFECTIONS
Coryza is common in the Western Desert aboriginal population and shows a seasonal variation similar to that in the Caucasian population. It is most frequent during the winter months and is similarly preceded by prodromal shivering, sneezing, fever and headache and is accompanied by cough, temperature and nasal discharge.

Although the nasal discharge appears to be more profuse than in the white population, it is probably more apparent than real due to aborigine's habit of not removing any secretions. Although Garnet (1928) stated that the common cold was much more severe in the aborigine than in the European, I have not found this to be so in the uncomplicated case. However, in my experience the majority of aborigines do develop secondary complications particularly sinusitis, otitis media, bronchitis and not infrequently, pneumonia. These complications are much more common and severe in the young aborigine, particularly in 3 to 5 year old age group.

Apart from Garnet's (1928) reference to South Australian aborigines, there are no other references to this disease amongst other aboriginal groups, although McPherson (1939) did note the use of eucalyptus leaves as a treatment.
INFLUENZA

This disease, in my experience, is the biggest single cause of mortality among the Western Desert aborigines, even though the mortality is almost completely restricted to the infants and younger children. Unfortunately, during my stay in the Western Desert, viral studies were not available and specific viruses could not be isolated. However, various epidemics which did occur in this area in the aboriginal population were also accompanied by general influenzal epidemics in Australia, and in the Caucasian population in the Western Desert, and there is no reason to doubt that they were not due to the same virus.

In the adult aboriginal, the clinical course is similar in both the prodromal and active phase to that in the Caucasian population. However, the incidence of secondary infection is very high and sinusitis, otitis media, bronchitis and pneumonia are the commonest of these complications. It is therefore in my opinion, well worth while to give empirical antibiotic therapy in aborigines to lessen the risk of these secondary infections.

In the children and particularly in the infants, the course is much more dramatic. The prodromal stage is often remarkably short and the same secondary complications which occur in the adult, also occur in this younger age group. However, this particularly susceptible group not only develop
these secondary infections, but also develop an intractible gastro-enteritis in which death may supervene within 24 to 72 hours despite adequate attempts at rehydration and the use of antibiotics. During the 5 years I worked in the Western Desert region, this pattern of influenzal infection was the cause of death in 12 aboriginal children. Of those that recover, considerable morbidity remains and a large proportion of the younger children continue to have a chronic form of gastro-enteritis. This is probably related to destruction of the enzyme systems in the small bowel epithelium as they show evidence of both fat and disaccharide intolerance and in turn develop signs of malabsorption, accompanied by anorexia, wasting and anaemia and these effects may continue for months. It is possible that in the group of children who develop both the acute and chronic form of gastro-enteritis following influenza that there is in any case an underlying chronic gastro-enteritis from previous bacterial infection. This type of syndrome which has only been noted in the aboriginal population and not in the Caucasian population of the Western Desert region has also been reported by Tooth (1963) during a brief expedition to the Warburton Ranges area.

Influenza as a cause of both morbidity and mortality amongst other groups of aborigines, has been noted during the early days of colonisation by Stokes (1846) in New South Wales and Stretton (1892) in the Gulf of Carpentaria. Basedow (1919
and 1932) and Cleland (1919, 1928, 1962) both record numerous fatal cases during the 1919 pandemic and more recently, Crotty and Webb (1960) stated that influenza was a cause of death in 12 of the 175 autopsies performed on Northern Territory aboriginals. It is however surprising, that in this last series all the deaths occurred in the older age groups which is the opposite of my experience in the Western Desert region.

**MORBILII**

Measles have a low grade endemicity in both the Caucasian and urban aboriginal population in Western Australia, and like Britain it tends to cycle in epidemic form approximately every four years. The effect of measles on the urbanised aborigines differs in no major way from that in the white population, although the incidence of secondary infection is higher. It is in the relatively non-immune nomadic aborigines that the major morbidity and mortality is noted. The potentially disastrous and rapid spread of this disease is well illustrated by an epidemic which occurred in the Western Desert region while I was medical officer in this area.

The first warning I had of this outbreak was a radio message from Warburton Ranges Mission, which lies 450 miles east of Leonora, stating that one of the outlying camps had reported that three babies had died and also an adult woman,
and that a large number of ill aborigines were starting to trek into the mission. Matron Bergman stated that she believed the disease to be measles as her own child had similar symptoms and signs. I immediately flew to Warburton Ranges with all the antibiotics available in both the Leonora Hospital and the town pharmacy. Fortunately, a new dormitory block at the mission was in the process of being completed and we were able to utilise this as a hospital as it was obvious that the eight bed hospital at the mission would not cope with the numbers of ill aborigines. Within four days, we were treating 198 aborigines as in-patients and another 280 on an outpatient basis.

In this epidemic prodromal symptoms were particularly severe. Temperatures of 105°F were not uncommon, and accompanied by headache, generalised muscle ache, conjunctival engorgement and frequently Koplik's spots. The exanthem also appeared in the aborigines, although the native pigmentation made the rash appear dusky and only slightly pink. The spots were readily identifiable by palpation and when numerous gave the appearance of a malar flush.

The incidence of secondary complications was extremely high: Ninety-five percent of the children developed otitis media. More than 80% of the children and adults had secondary respiratory tract infection, sinusitis, acute bronchitis, bronchial pneumonia and in some 20 cases lobar
pneumonia. Despite the widespread use of antibiotics and the fact that no further deaths occurred showing the efficacy of this treatment, the resultant morbidity was remarkably high as some 40% of the children still had discharging ears and evidence of chronic sinusitis 6 months later. It was impossible to judge the increase in chronic bronchitis and the deterioration in those who previously suffered from this disease following their measles. One case of post measles encephalitis also occurred. After 4 days, I managed to induce the West Australian government to supply further medical aid in the form of a hospital registrar from the Children's Hospital in Perth - Dr. Tooth. He flew to Warburton Ranges with four nurses and they continued both the inpatient and outpatient management of the aborigines while I returned to treat the epidemics which had by this time occurred in Laverton and Leonora, although these were much less severe. Tooth (1963) referred to this epidemic in an article and stated that it was probable it had come from Alice Springs. There is however, no evidence to support this and it is my belief and there is proof supporting this, that the epidemic had been carried from Kalgoorlie by a group of aborigines returning to Warburton from a corroboree and at that time there had been increased frequency of measles in Kalgoorlie. Both the timing of the outbreak of Warburton and the pattern of the epidemic which occurred initially at Kookynie, then at Laverton and Warburton supports my theory. Further proof is given by the fact that this group of aborigines had some form
of influenzal-like illness in some of their members during their return journey from Kalgoorlie. Dr. Tooth did bring a considerable quantity of gamma globulin from Perth and this was given to all the unaffected aborigines (there being a total of some 700 at Warburton Mission at that time) but it appeared to have little effect.

Epidemics of measles in other groups of aborigines have been previously reported and in the pre-antibiotic era, evidence of the mortality associated with this disease is offered by Hackett (1865) who reported that half the tribal natives at York, West Australia, died from this disease. Other outbreaks in Australia have been reported as occurring in 1883 by Cleland (1928) and in the Shark's Bay area in 1884 by Curr (1886). Taplin however, (1879) stated that Australian aborigines do not readily take measles although he gives no evidence of this statement. Holmes (1912) reported an epidemic in the Northern Territory in which several deaths occurred and a further epidemic in 1913 with a lower mortality. More recently Langsford and Hawser (1969) have reported the mass use of live measles vaccine in 968 aboriginal children of the Northern Territory whereby they limited an apparent epidemic.

There is undoubtedly considerable morbidity and mortality associated with measles in Australian aborigines, particularly amongst the more nomadic ones, and in view of
Langsford's recent findings it may well be that all aborigines should be vaccinated against this disease in childhood.

**RUBELLA**

This disease has a low incidence of endemicity in the urbanised aboriginals of the Western Desert similar to that in the Caucasian population. I have not noticed any difference in the symptomatology between the two races, but it is more difficult to diagnose in the aborigine because of the less obvious rash which appears merely as a discoloration of the normal pigmentation. The lymphadenopathy is however identical and if this is missed it is possible for the disease to be mis-diagnosed as a common cold.

All the cases I saw in the Western Desert were confined to children within 3 and 8 years of age. No complications followed and no specific treatment was required and it is probable that amongst the urban aborigines, there is a fairly high level of immunity in the adult to the disease. I have not seen the disease in epidemic form and there are no references available to its incidence in any other groups of Australian aborigines.

**HERPES SIMPLEX**

This is commonly seen in the Western Desert aborigines, usually accompanying respiratory tract infections, particularly lobar pneumonia. The lesions frequently become secondarily
infected and spread as an impetigenous rash. I have not observed either the vulval, vaginal or gestational form amongst the Western Desert aborigines and there are no reports of herpes in any other groups of aboriginals.

HERPES ZOSTER

I have not seen any cases of herpes zoster in a Western Desert aborigine although in a comparable Caucasian population in this area there were 11 cases.

VARICELLA

Varicella was not uncommon among Western Desert aborigines in my experience; appearing in roughly the same frequency as was seen in the Caucasian population and running an identical clinical course although secondary infections of the lesions was much more common and extensive.

Cleland (1928) also noted that varicella was not uncommon in Australian aborigines and quoted Junk of Wondai, Queensland as stating that the eruption may also be found on the palate and fauces, as well as on the body surfaces. He also quotes Leighton-Jones of Darwin as stating that this disease may be epidemic at times in Australian aborigines but he (Leighton-Jones) considered that it presented no unusual features.
Although no cases of smallpox have been reported in either the aboriginal or caucasian population in Australia during this century, it has undoubtedly been a major cause of mortality in the aborigines in the past when it occurred in three major epidemics in 1789, 1828 and 1860. Each of these epidemics lasted several years and extended over the whole of the Australian continent.

Cases were first noticed although not identified at Port Jackson Bay soon after the arrival of the first fleet. Stirling (1894) and Cleland (1911) have both deduced and provided evidence that the disease was in fact introduced by Malaysian fishermen in the north before the arrival of the first English colonisers. Curr (1886) commented extensively on the distribution of the disease and Cumpston (1914) traced the history of smallpox in Australia in both the aboriginal and Caucasian population.

Initially, considerable confusion was present in the early days of settlement as to whether or not it was true smallpox which was present in the aboriginal population and in fact the disease in the aborigines was called "native pox", although Wilson in 1835 stated that both diseases were similar and commented after examination of the native pox marks, "but evidently, native pox bears a resemblance both in symptoms and consequences to smallpox. The eruptive disease
attends a fever and leaves a depression. It frequently destroys the eyes and I have referred more than one native who had thus suffered." In fact in the earlier days of settlement despite the widespread use of vaccination in the Caucasian population it was not used in the aborigines. It was not until 1832 as Bridges (1970) has noted that it was even suggested that vaccination should be extended to the aboriginal population, and this was only done when it was considered possible that the aboriginal population were in fact infecting the white population. However, it was not until 1914 that Compston positively stated that both native pox and smallpox were in fact the same disease.

Some ideas of the mortality attending the smallpox epidemics can be gained from Backhouse (1814) who stated that 140 out of 200 natives in New South Wales died from this disease in one epidemic, while Josephson (1884) discovered a cave full of skeletons of aborigines who presumably died from smallpox epidemic 40 years previously. Curr (1886) quotes Kirk who noted the degree of scarring suffered by the natives and judged by the scars of the survivors. "More men than women, and more women than children survived." He also commented that he had never seen white people severely marked. In spite of the prevelance of these epidemics amongst the aborigines, relatively few white settlers were affected due to their previous exposure in Europe and the subsequent vaccination of the children. This apparent lack of cross
infection of the races appeared to be one of the major problems in deciding whether or not native pox and smallpox were identical and in adequate steps being taken to eradicate it in the native population. Although there are no accounts of smallpox having been seen in the Western Desert region it must have penetrated to this area as Cleland (1928) noted that an aborigine with pox marks was seen at the Rollison Ranges in 1873.

The history of smallpox in Australian aborigines shows that this race is particularly at risk if another epidemic should start in this continent. Virtually none of the aborigines of the Western Desert region were vaccinated and in fact relatively few Caucasians are too. When working in this area I started an active campaign of vaccination which was largely restricted to the urban groups and this at present seems to be the only measure which can be taken if we are going to prevent another outbreak. As demonstrated by the previous rapid and uncontrollable spread of measles, smallpox when introduced to this area, would spread with the same rapidity, and certainly much more disastrous results. One would presume that a similar state of affairs exists in other aboriginal communities.

MUMPS

The only cases of mumps in the Western Desert aborigines I saw during my stay in this area were confined
to school children in the urban area. The incidence and pattern of disease, and the severity and complications were identical to that in the caucasian population. Presumably in this area the disease is endemic and most of the urban aboriginal adults have considerable immunity. It is difficult to say what immunological status to this disease is present in the nomadic aboriginal and what the effects of an epidemic in this region might be.

Binns (1945) reported a mild epidemic, affecting male and female natives in equal proportions in the Northern Territory. He stated that only mild parotitis was seen except in one case where orchitis developed.

**POLIOMYELITIS**

The incidence of this disease has decreased rapidly in Australia since the general introduction of Salk vaccine in 1956 and with the more recent introduction of the Sabin vaccine. The poliomyelitis sub-committee quoted only 1,205 cases in the years 1956-63 in the whole of Australia. This was a remarkable decrease as the previous annual report had been a few off a thousand cases per year with peaks in 1937 and 1951 of 1,500 and 1,200 cases respectively. Surprisingly, few references have been made to either the effects or prevalence in the Australian aborigines and it would appear that they have been little affected by the disease.
In the Western Desert area I saw no fresh cases of poliomyelitis, and could find no evidence of previous infection. When we consider that the habits of both the urban and nomadic aborigines place them at particular risk from the disease, it is likely that they do in fact develop an early immunity both from passive immunisation at birth and active immunisation from repeated sub-clinical infection in later years. However, one must be aware of the problem with widespread immunisation programmes which do not reach the whole of the community. It is virtually impossible to vaccinate all the desert aborigines and it may well be that in the future we will see a recurrence of poliomyelitis in the unvaccinated group of nomadic aborigines. There have been few reports of this disease affecting other groups of aborigines. Crotty et al. (1960) had no deaths from this cause in their series of autopsies in the Northern Territory. Basedow (1932) did however, note two South Australian aboriginal children who had some form of paralysis of the lower limbs which he stated may have been infantile paralysis, although from his description it seems more likely that they were suffering from vitamin deficiency probably a vitamin such as C or possibly early yaws. Kirkland (1939) noted several cases in the Northern Territory and Miles (1953) and Stokes et al. (1955) have shown the frequency of neutralising antibodies in aborigines in this area.
INFECTIVE HEPATITIS

The aborigines of the Western Desert area also appear to have some immunity to this disease. I have experienced a mild epidemic of this disease in 1962 in the Western Desert area when 13 of the white population were affected. Despite the fact that these patients came from all parts of the area including Warburton Ranges mission, there was only one full blood aboriginal who was diagnosed as also having the disease. Again it is probably that this disease is in fact present in very high endemic form in the aboriginal population due to their living habits, and that a fairly high level of immunity is present in this population and in the future, we may see an apparent increase in incidence of this disease, as living standards unimprove.

Basedow (1919) stated that the natives of the northern part of South Australia, apart from jaundice due to congenital syphilis (which I find it difficult to believe) suffer from an acute form of jaundice, which fairly frequently affected young and old. From his description that apart from the yellow discolouration of the mucus membranes of conjunctiva, the common symptoms were nausea, vomiting, headache, constipation and general despondency, it suggests that this was probably an epidemic of infective hepatitis. It is worth noting that this group of aborigines were just coming in contact with white population at that time, and it may well be that they had no inherent immunity prior to this
contact. Yirrkala has also (1963) reported an outbreak of infective hepatitis in the Northern Territory aborigines.

OTHER VIRAL INFECTIONS

I have not seen any other viral infections in the Western Desert aborigines although it is possible that some of the following may exist. Beech et al. (1953) investigated the distribution of psittacosis in budgerigars and cockatoos in Australia and although the virus was frequently isolated in these birds, no cases were reported amongst the aborigines, although a few of the inland aborigines of the Northern Territory did demonstrate antibodies.

Murray Valley encephalitis is apparently endemic in certain areas of Australia. Beech et al. (1953 & 1958) demonstrated that the viral antibodies were present in 133 of 266 aborigines tested in the Northern Territory and South Australia. Warner et al. (1957) found 3 out of 27 positive at Haast's Bluff and Doherty and Carley (1959) showed positive antibodies in 145 out of 267 aborigines in the Gulf of Carpentaria. Although no specific test appears to have been done in the Western Desert aborigines, it is unlikely that a high pick up rate would be found as Anderson et al. (1960) demonstrated that the disease was restricted to Papua New Guinea in the western low lying regions. More recently Doherty et al. (1960, 1962, 1963) have demonstrated the widespread prevalence of this disease in Queensland aborigines.
The presence of both dengue fever and dengue fever neutralising antibodies has been reported in the Queensland aborigines by Doherty and Carley (1959), and I have seen no evidence of this disease in Western Desert aborigines.

The possibility that Echo viral infections may account for some of the apparent localised outbreaks of infective "hepatitis" has been raised by Riseborough (1962). In the northern area where he was in practice there had been a mild epidemic form of apparently infective hepatitis present for some time. Investigation of this revealed that it was due to an Echo viral infection.

The trachoma will be described with diseases of the eye.

Yellow fever has never been shown to be present in any Australian aborigine, although there was some confusion in the early days of settlement with the disease which was popularly known as "Yellow Jack" in the northern parts of Australia, and which was apparently due to malaria. The appellation of "Yellow Jack" for this disease was of course frequently confused with yellow fever by immigrants.
This disease is extremely uncommon in the Western Desert aborigines despite the fact that many patients have a positive WR and Kahn reaction. I have never seen any evidence of primary, secondary or tertiary manifestations of this disease, nor have I seen it in a congenital form in this area.

The history of the apparent prevalence of syphilis in Australian aborigines is quite remarkable. It is probable that no single disease has been so misdiagnosed during the course of the century as this in the case of the Australian aborigine. Sturt (1830) commented that in the Murray River aborigines syphilis raged among them with "fearful violence". He noted that many had lost their noses and all the glandular parts were considerably affected, while amongst the sequelae he mentioned were loss of sight and skin eruptions and that he thought many of them were "hurried to premature graves". Eyre who established an aboriginal station at Murandee in 1841 is stated by Curr (1886) to have noted that many diseases had spread by the year 1844 from the white to native population, including venereal disease. Stirling (1896) recorded that during the Horn expedition both syphilis and gonorrhoea were diagnosed as being rife amongst the tribes and that he had frequently noticed destruction of both the nasal and palatal structures. Curr (1886) considered syphilis to be a major cause of death amongst the aborigines.
of Australia and he was supported in this view by Stretton (1892) in regard to the natives of the Gulf of Carpentaria. Foelsche (1881) noted the same with the tribes of the Northern Territory, and in 1912, Breinl stated that the disease was prevalent in the Northern Territory and in 1915, Breinl and Holmes described a case of tertiary syphilis in a native from the Alligator River, commenting that the disease was not uncommon, although it "only affected those who came in contact with the white and Chinese" and that they also considered that syphilis affected the aborigines much more severely "than it does those other races amongst whom it has been present for centuries". It was not until 1928 that Cleland expressed some doubt about the true incidence of syphilis in aborigines when he said, "There was a wide spread and probably corrupt opinion that the diseases syphilis and gonorrhoea are wide spread and prevalent; contribute largely to mortality and is a common factor in the rapid dying out of this race". In this article Cleland also notes some of the contemporary opinions on the prevalence of syphilis amongst the natives. He quotes Dr. Gorrie of Port Augusta as reporting that tertiary ulcers are quite common although he says he has never seen a primary or secondary manifestation and that he does not think there is any significant difference in the manifestation of the disease between white and black population, merely that the aborigine suffers from lack of treatment. Dr. Atkinson from Perth, Western Australia, stated that syphilis and gonorrhoea were particularly marked
when the Asiatic and Aboriginal populations mixed. Dr. Junk, Wondai, Queensland noted that syphilis was prevalent in his district and responded to mercurial treatment, although he also considered that most cases died before reaching the tertiary stage. Dr. Illingworth of Taroom in Queensland believed that 20% of the aboriginals were affected, but stated that he had only seen one case of secondary syphilis and "the gravity of the disease to be much less than in the white man". However the most significant statement is probably that made by Dr. Cilento, Townsville, Queensland, who stated that he believed syphilis to be uncommon and that he had never seen a primary chancre and that the lower incidence may be due to the prevalence of yaws in this area. Nonetheless, Basedow (1933) stated that syphilis was common and that there was frequent manifestations of gumma which generally affected the skin, bones and the internal organs. The most frequent bony manifestations being those of bossing of the frontal bones, thickening and anterior curving of the tibia, - the so-called "boomerang legs".

It was not until 1936 that the well documented myth of the prevalence of syphilis amongst the aborigines exploded, when Nackett proved conclusively that boomerang legs were definitely due to yaws and that all the previous references to syphilis were, in fact, manifestations of yaws and also proved that the native disease "Irkonji" was identical with yaws. From that time most reports regarding the incidence
of syphilis amongst aborigines have been of a negative nature. Thus Binns (192) commented that he saw no evidence of primary or secondary stage syphilis in the aborigines of the Northern Territory, and Davidson (1957) made the same comment regarding the natives of the Warburton Range area of the Western Desert, a statement with which I am in full agreement. It is however naive to believe that the native population of Australia has not been exposed to syphilis and probably that it is a cross immunity from yaws which is at present protecting them and it may well be that as the latter disease is cleared up, then we may see true syphilis appearing in both the urban and nomadic aboriginal groups.

**YAWS**

Yaws is endemic in the Western Desert aborigines although cutaneous manifestations are relatively rare. I have only seen two cases in this group, but bony manifestations are more common, although at present are only seen in the older aborigines.

In my experience over 40% of the Western Desert aborigines have positive W.R. and Kahn reactions but there are few complications and relatively few positive manifestations of yaws in the area. The cutaneous lesions appear to be much less frequent than in the Northern Territory and they tend to heal spontaneously leaving very little scarring. The most common manifestation in the Western Desert Region is
"boomerang legs" due to anterior bowing of the tibia, but this is only seen in the elderly aborigines. This is very uncommon in my experience in the younger age groups. The most common manifestation in the teenage and young adult group is bone pain, usually humeral, radial, or ulnar.

The actual prevalence of yaws in the Australian aborigine is in fact clearly reported in the Australian Medical Literature and modern text books tend to ignore the problem completely. The result of which, when I saw my first case in a 17 year old boy who complained of pain over lower end of the radius, when I x-rayed him I thought the x-ray pattern was that of osteoclastoma and referred him to Perth. Further x-ray studies revealed the generalised nature of this lesion and the typical pattern of yaws and he was soon cured with penicillin.

As stated earlier there was considerable confusion between the diagnosis of syphilis and yaws during the days of early settlement, but as early as 1912 both Breinl and Holmes in the same year, commented on the disease being prevalent in the aborigines of the Northern Territory and noted that it was confined to the native population. Again in 1915, Breinl and Holmes gave a more detailed account of the incidence of the disease in the Derby River area, and reached the conclusion that almost every child had been affected at some time or another, but that usually the course
was self-limiting and only a few scars remained in later life. It is noteworthy that the cutaneous lesions were the only manifestation described and probably the osseous lesions were confused with syphilis.

The earliest description that one can find of boomerang legs is that given by Stirling in 1894 who noted the condition in an aboriginal in Central Australia and again he commented on its appearance in 1896 when several cases were seen on the Horn expedition. He did not describe any particular aetiology to this condition, but he did refer to Tapinarde (1890) who had made similar discoveries in Paris regarding the bowing of the tibia. Spencer and Gillen (1899) noted several cases but did not know whether it was racial or pathological, although Smith (1906) stated that he thought it was possibly a form of rickets. Breinl (1912) reported a case of sabre tibia from the Roper River area, but stated that he saw no evidence of yaws, and again Breinl and Priestly (1916) who investigated several cases of sabre tibia, including post mortem examination, were similarly unable to decide on the cause, although they considered the deformed tibia were due to neither syphilis nor tuberculosis. In 1919, Cleland reviewed some of the literature regarding boomerang legs without reaching any definitive conclusion, although in the same year Castellani and Chambers (1919) in discussing the aetiology of boomerang legs had stated that while the aetiology was unknown "we were able to exclude syphilis,
tuberculosis, ostiomyelasia and ricketts, but that a clinically similar condition is undoubtedly a late manifestation of yaws.

Despite the description by Bahr (1914) of an identical picture in the natives of Ceylon and the radiological appearances of sabre tibia described by Pauil (1918), Woods (1920) in his extensive studies of aboriginal tibia came to the conclusion that the anterior bowing was a racial characteristic. Spittle (1923) described the relationship of the deformity of the tibia and its association with yaws in the same group of natives. However, Basedow (1925) considered the deformity to be due to the tibialis posticus muscle of the aborigines, while Gray and Cleland (1933) decided that the deformity was due entirely to the way the natives squatted.

The failure to relate sabre tibia in the aborigines to yaws seems surprising, as a considerable amount of literature had been published in other parts of the world demonstrating their association, of which the best known are by Hallenberger (1916) in the Cameroons, Clapier (1920) in Central Equatorial Africa, Botreau-Roussel (1925) in the Ivory Coast, Soutema and Eichhorn (1925) in the Dutch East Indies, Chesterman (1927) in the Belgian Congo, Herman (1928) in the Dutch East Indies, Blacklock (1930) in Sierra Leone, Bulson and Mathes (1930) in Haiti, Knott (1930) in Liberia,
and Lopes (1935) in Brazil. Furthermore, Hermans in both 1928 and 1931 suggested that the boomerang leg of Australian aborigines and sabrous shin of other races were identical and due to yaws, it was not however until Hackett (1936) proved that the native disease Irkunji, boomerang leg, and what had previously been diagnosed as syphilis, were in fact, all manifestations of yaws and that moreover the disease had been endemic before the colonisation of Australia. In this study, he also related the incidence of positive Wasserman reactions amongst Australian aborigines to the prevalence of the disease in Australia, some 44% of all aborigines showing positive WR while 88% of those who had boomerang legs also had a positive WR.

In more recent studies Binns (1945) showed that 48% of all admissions to his hospital in the Northern Territory had positive WR reactions, while Schneider (1946) also noted a similar incidence. Schneider also described ocular manifestations of yaws and from his description cutaneous lesions are common in the northern part of the Northern Territory, while, as one passes south, the cutaneous lesions decrease in severity but bony lesions become more common. Davidson (1957) showed that 60% of the Western Desert natives were WR positive in the small series he studied, and commented on the rarity of the physical signs and symptoms. As I stated earlier, in this group, cutaneous lesions are particularly rare. Davidson (1957b) still entertains some doubts as to the exact
role of yaws and syphilis in the 20-30% positive serological
WR tests he found in the natives of Western Australia in
general, but he does state "it would seem that the native
treponemal disease is one in which considerable resistance
has been developed in this population, and manifestations of
a tertiary nature are seldom seen". In 1961 Riseborough
described a group of Western Desert aborigines residing near
Cundalee mission that had extensive phalangeal osteitis due
to yaws that had resulted in some cases in a complete
resorption of several phalanges. Allen (1963) investigated
119 aborigines along the trans-Australia line, mainly around
the Cundalee mission. In these he found that 42.6% had a
positive WR; 30.9% had a positive Kahn; 45.6% a positive
V.D.R.L., and 11.8% had a positive G.C.F.T. He was unable to
reach any definite conclusion as to whether these results
were due to syphilis or yaws, however amongst these he
examined, he could find no cutaneous evidence of yaws, leprosy
or syphilis, but it is worth noting that this is a dry area
and is in fact to the southern part of the territory in which
the Western Desert aborigines reside and it is reasonable to
suppose that this disease is in fact yaws, unless one also
postulates that syphilis in the aborigines has a different
form of manifestation from that in the white population.
Anderson (1969) did state that there was a high incidence of
venereal disease in aborigines although he did not report
what type of venereal disease he was referring to, or in
which particular group of aborigines.
OTHER SPIROCHAETAL DISEASES

Pinta, relapsing fever and rat-bite fever have never been described in any aborigine, and I have seen no examples in the Western Desert area.

Weil's Disease is endemic in the north of Queensland, in the cane fields where it is transmitted by rat urine, the organism generally entering the cane-cutter's body through lacerations. Several cases in Queensland aborigines have been reported by Sinnamon and Pask (1952), and Mackerras et al. (1957), and Doherty (1958) have shown a high incidence of sero-reactors in the aborigines of this area.

No cases of Weil's Disease has been noted by myself or reported from the Western Desert area.
MYCOTIC INFECTIONS
MONILIA

I have noted monilia frequently in aboriginal infants in the Western Desert region where it presents as thrush, with the same clinical picture as in the white population. These cases have however, been restricted to the urban area, and I have not seen any in the nomadic tribes. Vaginal moniliasis is uncommon in the Western Desert and I have only seen one case in a pregnant aboriginal woman amongst the more nomadic groups, although it is not uncommon in the urban aborigines. Intertrigenous monilia infection I have only seen where the patient has been particularly obese. It is my opinion that there is probably a true lower incidence in the nomadic as compared with the urban population and that this difference is related to the thin build of the nomadic aborigines, their loose fitting clothing and the low humidity of the area. I have not seen any evidence of bronchial or pulmonary, or other systemic manifestations of moniliasis, and there are no reports in the literature regarding the general incidence of this infection amongst Austral aborigines.

TORULOSIS

I saw no evidence of cryptococcosis in the Western Desert region and to my knowledge, none had been diagnosed prior to my stay in this area, and none has been diagnosed since.

This disease was first reported in Australia by Cox
and Tolhurst (1946) who detailed several cases and reviewed the literature managed to find a total of 13 patients, all of whom were white. In 1960, Elphinstone noted an outbreak of torulosis in the Kimberley region and commented that five aborigines, all within a radius of fifty miles, had proven disease which was similar in all respects to that previously reported by Cox and Tolhurst in the white population. Crotty et al. (1960) noted that one death occurred in the Northern Territory in an aborigine from this infection, and more recently, Crotty (1965) reported that 10 cases of torulosis due to cryptococcosis neoformance had occurred in 10 cases in the past 7 years in aborigines in the Northern Territory, 8 of whom had died. In 5 of these cases there was an associated pregnancy. Sutherland (1969) reported that the incidence of this disease in Queensland aborigines was 29 per 100,000 while in the white population it was only 1.6 per 100,000.

**ACTINOMYCOSIS**

This disease is rare in Australia at any time, and I have never seen any evidence of its presence in the Western Desert aborigines. However it has been described by Dr. J. Millar in an aborigine in 1915. This case having been referred to and treated by Cleland who reported it in 1928. More recently, Crotty (1965) noted two cases in the Northern Territory in aboriginals.
MADUROMYCOSIS

This disease is apparently rare and there are only two recorded cases. The first that of an aboriginal who possibly had madura foot although there was considerable doubt about this diagnosis which was reported by Breinl and Holmes (1915) in the Northern Territory. The second was reported by Cleland (1962).

NORCARDIOSIS

Again I have noted no evidence of this disease in the Western Desert aborigines, but Crotty (1960) reported it to be the cause of death in an aboriginal adult in the Northern Territory and this was confirmed by post mortem examination. Again Crotty in 1965 saw three other cases and noted that it was apparently relatively common in aboriginal groups as compared with Caucasian race as there have been very few reports of this in the Caucasian population of the same district.

RINGWORM

This disease is not uncommon in the Western Desert area and I noticed a similar incidence in both the aboriginal and white populations, although the former tended to be later in seeking treatment. I could observe no significant difference between the course of the disease and the rapidity of the cure and the delay was taken into account. The only difference noted was in the distribution among the aborigines,
tinea capitis being more common than tinea corporis. Both tinea pedis and tinea cruris were not observed at all. In neither population were any ring worm infections of the nails seen. There are few recent reports of the incidence of ringworms in aborigines, but Taplin (1879) reported it as being common in the South Australian aborigines and stated that it was derived from calves. Foelsche (1881) stated that it was common in the Northern Territory particularly in the Alligator and Adelaide Rivers and Point Darwin tribes. Stretton (1892) also stated that it was common in the Gulf of Carpentaria. However, all these descriptions are rather vague and it may well be that some other skin disease was being discussed. Recently Donald (1958, 1959, 1960) reported a high incidence of tinea capitis in South Australian aboriginal children, of whom the majority are probably part-blood.

I have seen no other forms of mycotic disease in the Western Desert aborigines and can find no reports to their presence in other aboriginal groups, except for a case of chromoblastomycosis in a Wyndham aborigine reported by Anthony and McAleer (1963).
PROTOZOAN INFECTIONS
None of the protozoan infections have been noted by myself in the Western Desert region or reported by previous authors in this area. It is probable that the extremely low incidence of all these diseases is due to the extremely dry and hot climate. The lack of surface water and vegetation reduces possible breeding areas for vectors and the nomadic life of the aborigines further reduces the chances of contact. However, a number of these diseases have been causes of considerable mortality and morbidity in the past and some are still present in other groups of Australian aborigines.

**AMOEBIASIS**

This disease was apparently introduced to the native population by the early white and Asiatic settlers and despite the relatively poor sanitation in many areas it has surprisingly remained confined to the coastal districts of the northern half of the continent. One of the earliest references to this disease being present in the aborigines was that by Cleland (1928) who quotes Cilento as stating that both amoebic and bacillary dysentery appeared as spasmodic outbreaks in Queensland, although Cook (1936) considered it to be a mild endemic disease in the tribes of the north of Western Australia. He feared that it would rapidly spread, a fear that has not been upheld as Binns (1945) only noted two cases in his extensive series of hospital admissions in the Northern Territory. Crotty (1960)
also in the Northern Territory, reported one death from the enteric form of this disease in an aborigine. However, Cook (1949) and McKerras (1955) and Sandars et al. (1965) testify to the continuing endemicity of this disease in the wetter areas. No authors have mentioned any of the expected complications such as liver or brain abscesses and as stated above, no cases have been noted in the Western Desert region, despite the fact I have examined more than 300 stool specimens for parasitic disease.

MALARIA

This disease was apparently epidemic and endemic in the northern parts of Australia where suitable breeding grounds existed for vectors. In the earlier days of colonisation, there is no evidence to show whether or not the disease was present before white settlers arrived as it is possible that it could have been introduced by Indonesian fishermen. Ford (1942) reviewed part of the history of this disease in Australia and noted that severe epidemics in the years 1824 and 1829 were of such virulence in the Northern Territory to be locally termed 'yellow jack', a name which was often confused by would-be immigrants with yellow fever. Fuelsche (1881) had also commented on the prevalence of the disease amongst the native population and McKillop (1892) supported his statement. Ford (1942) reported that in epidemics in 1911 and 1914 on Melville and Bathurst Islands yields 20 and 30 deaths respectively among the
aborigines. Breinl, (1912) stated that the usual form was simple tertiary malaria although Holmes (1912) stated that the epidemics were sub-tertiary in the Melville Islands area and were accompanied by considerable mortality. He believed the disease had been introduced by some Malay immigrants. Hill is reported by Cleland (1928) as believing that malaria was endemic amongst the Queensland aborigines. Cook (1949) surveying the history of this disease in Western Australia, reported that a severe epidemic in the Fitzroy basin in Western Australia in 1934 had caused the death of 150 aborigines, but noted that no deaths had occurred since that date and is apparent that the various anti-malarial measures which have been taken have proven reasonably effective, as neither Ford (1942), Binns (1945), or Crotty et al. (1960) in their recent surveys have noted any serious cases of malaria in the Northern Territory although there was an outbreak at Roper River in 1955. However, the possibility that adequate breeding grounds still remain, and that significant mortality and morbidity have resulted from this disease in the past, should keep us on our guard against possible recurrences. The possibility of reintroduction to other areas in Australia is not too remote, as I have seen several cases of malaria amongst the white population of the Western Desert area. These were servicemen who had recently been in South-East Asia.
TRICHOMONIASIS

This disease is not uncommon amongst the urban groups of Western Desert aboriginal women. It is however, in my experience, rare in the nomadic groups. The incidence of this disease has not been reported in other full blood aboriginal groups.

OTHER PROTOZOA DISEASES

Trypanosomiasis, coccidiosis, toxoplasmosis, and sarcosporidiasis have not been reported in the Western Desert region, and I have seen no evidence of these diseases in this area.

Cook (1959) reported high reactor rates to toxoplasmosis in the aborigines of Cape York.
METAZOAN INFECTIONS
This group of diseases has a remarkably low incidence in the Western Desert aborigines in my personal experience, but it is apparently a common cause of both morbidity and mortality in other aboriginal groups.

**ANCYLOSTOMIASIS**

This disease has never been reported in the Western Desert aborigines and I could find no evidence of its presence in this group of people during my stay in this area.

It has however, been frequently reported in other groups of aborigines and appears to have been introduced to the aborigines of Australia by the indentured Asiatic labourers. It is however, apparently restricted to the wetter coastal areas in the northern part of the continent and Sweet (1923) stated that it had never been noted below the 30° parallel. An idea of the prevalence of this disease in this area can be judged from the references of Sawyer (1919) who noted that 80% of the northern coastal aborigines were infected, while Sutton (1923) noted that 6% of the children of the north coast of New South Wales (at the lower limit of the endemic area) were harbouring the ova. In Western Australia in 1928, Atkinson stated that 90% of the children at Beagle Bay mission were infected, while Cook (1936) stated that the disease was still prevalent in the same area despite the widespread work of the Committee for the Eradication of Hookworm and the public health measures
which had been adopted, and that little decline in the incidence of the disease had been noted. Binns (1945) observed that 12% of all admissions to his hospital were for hookworm disease, and he considers it the most common cause of anaemia in the Northern Territory aborigines, a point that had been previously established by Form (1942) who noted 108 carriers of hookworm in 114-faecal specimens examined in Bathurst and Melville Islands. He also stated that he believed it was a frequent cause of stunted growth in the natives. Schneider (1946) working in the Northern Territory, quotes Andrews as stating that of the natives with eosinophilia, 50% pass hookworm ova. In 1960, Elphinstone noticed that hookworm was still prevalent in Beagle Bay Mission, Julumburu Mission, Forest River Mission, and in the Wyndham area, all of which places are in the north of Western Australia, and he further stated that 10% of all the Kimberley aborigines lived within the area of endemic hookworm disease. Elphinstone (1966) repeated his statements regarding the incidence of the disease in Western Australia and also McKerras (1955, 1960 and 1961).

The organism implicated in this disease in Australia is ancylostoma duodenale and it is apparent that the dry interior is the major factor preventing its spread from the coastal to other groups of aborigines. It is probable that the coastal incidence is in fact increasing with continuing settlement of these aborigines into permanent camps with the
still rather primitive sanitation arrangements which are present.

ASCARIASIS

Again this disease has neither been reported in the Western Desert region, nor have I been able to establish any cases despite various searches of stool specimens for ova.

It is however, endemic in certain areas of Australia in the aboriginal population and it is probably controlled on a climatic basis, only being present in the wetter regions. The general incidence of this disease amongst the aborigines of the wetter regions is probably much higher than has been previously supposed as Cleland in 1928 was only able to cite one case as having been reported in any aborigine to that date. However, Ford (1942) noted two positive stools in the series he examined in the Northern Territory and Stevenson (1961) in a survey of the aborigines in a Victorian settlement isolated ascaris lumbricoides in 54.7% of the 95 children he examined, and at a later two year follow-up after they had been treated, 14% were still infested. A further study by the same author of the East Gippsland area of Victoria showed a general aboriginal infestation at the rate of 63.6% although no white people were infected in his control series. He stated that the low sanitation, wet climate and lack of footwear amongst the natives were the predominant factors accounting for the prevalence of the
disease. He also stated that he suspected that many of the children previously diagnosed as having pneumonia had probably had ascariasis pneumoniasis. Sandars (1961, 1962 and 1963) and Sandars et al. (1965, 1966) have shown a very high incidence in Queensland coastal and island aborigines.

It would seem from the above reports that this disease probably has a much greater incidence than has been previously suspected, particularly in localised pockets in the wetter areas.

**TRICHOCEPHALIASIS**

The distribution of this disease seems to be similar to that of ascariasis. I personally have found no cases in the Western Desert region and there are no records of it ever having been found in this area.

There is a paucity of information from other regions of Australia, but Stevenson (1961) in his East Gippsland survey recorded 75.8% as being positive carriers, although his total Victorian aboriginal incidence was only 3.3%. Sanders (1962) and Sandars et al. (1965) also noted localised pockets of infection in Queensland. On the basis of these reports, it would seem likely that the distribution of this disease is similar to that of ascariasis with a fairly general endemic incidence in the aboriginal population of the wetter areas with local pockets of very high prevalence.
and that the major factors in the high incidence are poor sanitation and hygiene.

**STRONGYLODIASIS**

Again there is no evidence of this disease being present in the Western Desert aborigines either in my own or others' personal experience. The only reports available on this disease is by Ford (1942) who observed one case amongst the Bathurst Island aborigines, although McKerras (1951), (1955) noted heavy infestation in the islands, and Walker-Smith (1969) who reported small bowel obstructions in an aboriginal infant due to strongyloidiasis.

**ENTEROBIASIS**

This disease is not infrequent in the Western Desert aborigines, although it is more common in the urbanised aboriginals than in the more easterly groups. The disease appeared to have a similar frequency in both the white and aboriginal populations in the Western Desert area, although the urbanised aborigines had a higher incidence than the white people but this was offset by a lower incidence in the more nomadic groups. During a two year period at Leonora, I treated 21 aboriginal children for this disease.

    Ford (1942) noted that enterobiasis was present in 23 of 114 stools examined in the Bathurst and Melville Island area and both McKerras (1955) and Sandars (1962) have also
noted heavy infestation in the aborigines of North Queensland.

FILARIASIS

I saw no evidence of this disease in the Western Desert aborigines and there are no reports of it having been present.

It has however, been recognised by Richards and Cilento (1923) in the aborigines of the north of Queensland, who stated that it only occurred west of the mountain range amongst the natives of the north of Queensland and he considered the parasite to be *F. bancrofti*, the vector being *culex fatigans*. There was no indication of either the incidence or severity of the disease in this report, although Cleland (1927) stated that Illingworth of Taroom, Queensland, considered it to be not uncommon. More recently, Ford (1942) stated that he could find no evidence of filariasis in the Northern Territory and this has been supported by McMillan (1967) who examined 374 full blood aborigines for the presence of filaria and could not find any positive carriers. McKerras (1950, 1951, 1953, 1957) also found no evidence of filariasis in several Queensland surveys.

DWARF TAPEWORM

*H. nana* infestation has only been reported by Stevenson (1961) in the aborigines of Victoria with a general
incidence of 3%, although 7.4% of the aborigines of the East Gippsland area were affected, and by McKerras (1955) and Sandars (1962) in Queensland where the disease is prevalent. I noted none in the Western Desert area.

CYSTICERCOSIS

This disease has never been reported in an aboriginal from any part of Australia and I have noted none in the Western Desert Region.

ECHINOCOCCOSIS

I found the incidence of this disease extremely difficult to verify in the Western Desert aborigines. As noted earlier, a high percentage of these people are positive Casoni reactors, but there doesn't seem to be any association between this test and the actual presence of hydatid disease, as I could not establish a positive diagnosis of disease in any of those patients who had a positive test. I could find no references in this area to hydatid disease ever having been diagnosed and in discussions with Davidson (1960) he too, could find no evidence of the disease in this area. As I have described earlier (page ), this test requires further investigation in the Western Desert. On purely theoretical grounds however, it is unlikely that there would be a high incidence of the disease in this region, as the climatic conditions of low humidity and high
temperature, plus the relatively nomadic nature of the aborigine's movements, would make it unlikely for them to be at high risk.

There is however, no doubt that the disease is present in other aboriginal groups. Again these seem to be associated with the wetter areas of Australia.

The first reported case of hydatid disease in an aborigine, was that by Hudson (1861) to be followed later by Gunning (1884) who reported two cases, and Curr (1886) who stated that hydatid disease of the liver was known in the aborigines, but he gave no further details. The disease was apparently fairly common in South Australia as Stirling observed that during the preceding eleven years from 1896, no aborigine had died in the Adelaide Hospital "except from tuberculosis, hydatid disease or accident", but unfortunately he gives no statistics. Cleland (1928) noted several cases and commented on the examples of the disease in aborigines as demonstrated in the Adelaide University Pathology Museum. The overall incidence was probably however, lower than is suggested in these reports, because despite the impression amongst medical practitioners that it is not uncommon neither Ford (1942), Schneider (1943), Binns (1945), Elphinstone (1960), nor Crotty (1961) noted any cases in their fairly extensive surveys of aborigines in the Northern Territory, Kimberley Region, northern part of Western Australia.
or Bathurst and Melville Island area. In addition, the work of Casely-Smith (1958) confirms that a positive Casoni test in the Australian central desert aborigines bears no relationship to infestation with hydatid disease, a fact that has also been emphasized by Warner et al (1957) in relation to the Haast's Bluff area.
ARTHROPOD DISEASES
This disease is relatively common in the Western Desert aborigines, particularly in the more urbanised groups. Sporadic cases occurred in both the more settled and nomadic groups of aborigines during the time I was in the Western Desert area. The clinical course was identical to that seen in the white population of the same area, but in the aborigines secondary infection was much more common and in all the cases I saw, the disease was restricted to the hands. The relapse rate in aborigines was high unless they were admitted and treated in hospital under supervision.

This disease is apparently relatively common in other groups of aborigines and in 1928, Cleland stated that it had apparently been introduced to the aborigines of Point MacLeay by returning servicemen, when the incidence rapidly rose. In a survey of his contemporaries to establish the prevalence of the disease, he stated that the incidence varied from rare to 90%, and that when present, this disease was frequently associated with impetiginous sores. Ford (1942) noted several cases in the Bathurst Islands aborigines, but Binns (1945) made no mention of the disease in the Northern Territory mainland. Davidson (1957) saw several cases in the Warburton area in the Western Desert.
PEDICULOSIS

I found this disease to be less common than scabies and was in fact surprised at the relatively low incidence of this disease in the Western Desert aborigines. The majority of the cases I noted were in the school children in Leonora, and the incidence was almost identical with that in the white children in the same area. Despite lower standards of hygiene, the incidence was remarkably low at Laverton and Warburton ranges and most uncommon in the adults. All these cases were pediculosis capitis.

Basedow (1932) stated that pediculosis was common amongst aborigines, the children particularly being affected and their heads covered with excoriations and he stated that he had observed body and pubic varieties as well. He also stated that pediculosis corporis predominated in the west coast aborigines, but pediculosis capitis was more common in northern aborigines. Whether in fact there is a true decrease in incidence from that time or whether the drier and less populated central areas are less conducive to infestation is not known, but Davidson (1957) commented on the low rate of infestation in the Western Desert natives when he examined them at Laverton, and at Warburton Ranges, which is in complete agreement with my personal findings.
RICKETTSIAL INFECTIONS
Although Q fever and scrub typhus have been described in the Australian white population, there have been no specific reports of these diseases affecting any aboriginal and I observed none in the Western Desert region. It is however, possible that these diseases may be more prevalent in the aboriginal population than has been previously considered, as Warner et al (1957) observed one aborigine at Hasst's Bluff who demonstrated a positive antibody titre to Q fever out of 36 aborigines tested, and Beech et al (1953) noted several aborigines with antibodies in Northern Queensland.

Snow (1953) considered that several deaths in aborigines at Beagle Bay in 1922 were due to murine typhus.
BACTERIAL INFECTIONS
Under the conditions in which I worked in the Western Desert regions of Australia, it was impossible to culture organisms and to obtain their sensitivity. The following description of the various bacterial infections present amongst these people is therefore based purely on clinical analysis. There have been very few studies of the normal flora of Australian aboriginal groups and the only one which has produced significant results is that by Rountree (1969) who examined the nasal flora of the Australian aborigines of the Pitjantjatjara tribe in the Musgrave ranges. In a group of 132 adults he found that 6% had staphylococcus while proteus was present in 63.6% of these individuals. In 148 aborigines aged under 15 years of age, 18% had staphylococcus in the nasal flora and 12.8% had proteus. He found that only 1 out of every 46 persons with staphylococcus aureus had an organism which was penicillin sensitive demonstrating the wide use that was made of penicillin in this region and also indicating the possible changes that were occurring in the normal aboriginal organisms with the use of wide spectrum antibiotics.

Pneumococcal Infections

The most common form of apparent pneumococcal infection in the Western Desert aboriginal is lobar pneumonia. I admitted 26 patients to hospital for treatment of this condition between 1960 and 1963. The majority were
children between 3 and 8 years of age, and 4 died. The disease in the aborigine is much more acute than in the white person. The probable cause is that they are much more debilitated at the commencement of the disease and most have already got some chronic respiratory disease, mainly bronchitis or bronchiectasis in the young, or chronic bronchitis and emphysema in the older age groups.

There are few early reports of lobar pneumonia as a specific entity but it is probable that they form a large proportion of the many cases loosely referred to as 'chest infections' by Taplin (1879), Curr (1886), Smith (1878), Foelsche (1881), Brienl and Holmes (1915), and Basedow (1919). It is also probable that many were not diagnosed, death probably occurring before medical attention was received or being treated by lay people and these aspects may well account for Cleland's (1928) survey only being able to quote two sources, that of Gorrie in Port Augusta who stated that he had not seen many cases, and Jones of Darwin who said he had only seen one case and the child had died. However, the more recent references do indicate that it has been relatively common. Basedow (1932) regarded it as being relatively frequent and Ford (1942) stated that quite a number of cases had occurred in the Melville and Bathurst Islands. A more exact statement of Binns (1945) based on clinical diagnosis with radiological and bacteriological confirmation, showed that 5% of all aborigines admitted to
hospital in the Northern Territory in that year (a total of 400 patients) had lobar pneumonia. Similar accounts with relative incidence of the disease were made by Schneider (1946) in the Northern Territory, and Elphinstone (1960) in the Kimberley region. Crotty (1960) in his series of post mortems observed that in 19 the cause of death was pneumonia, but he does not state which type, or which particular organism was implicated, and that in a further 6 cases the cause of death was lung abscess. Again the aetiology was not given.

Pneumococcal meningitis occurred in one young aboriginal woman I treated in the Western Desert area. This followed lobar pneumonia and the specific organism was proven in this case. She fully recovered from this illness. There are no other reports of pneumococcal meningitis or pneumococcal peritonitis in any other areas.

**Streptococcal Infections**

Disease due to streptococcus is relatively high in both the Western Desert and general Australian aboriginal groups and will be considered under the following four headings:

**Acute Tonsilitis** is common in the Western Desert area in my experience. It occurs particularly in the wet season and in the urban aborigines of this area it may
reach almost epidemic proportions particularly in the children and it is usually complicated by bronchitis, sinusitis and otitis media. Moreover, in this age group, acute episodes are usually superimposed on chronic tonsillar disease and an accompanying chronic suppurative otitis media. I have not seen any peritonsillar abscesses but this may well be due to relatively early treatment being instituted. In many respects the aboriginal children of the Western Desert region present a picture similar to that seen in Glasgow slum children of the pre- and immediate post-war era, having chronic suppurative otitis media, associated with chronic infections of the upper respiratory tract, of both the tonsils and the adenoids and frequently with sinusitis, in turn giving rise to chronic post nasal drip, chronic bronchitis, sinu-bronchitis and bronchiestasis. The distribution of acute tonsilitis in other groups has been briefly mentioned by Curr (1886) as being relatively common, while Basedow (1919) cited several cases in the north of South Australia. Ford (1942) also observed several while Binns (1945) noted four cases in his series on the Northern Territory. Elphinstone (1960) in discussing the health of the Kimberley aborigines, mentioned it as the disease of the upper respiratory tract with which he most commonly met.

Erysipelas has not been reported in Australian aborigines apart from one doubtful reference by Basedow (1932), and I have seen no cases in the Western Desert area.
Scarlet Fever. During the time I was in the Western Desert area I saw no cases of scarlet fever in the aboriginal population, although several cases occurred in the white population of this region.

The disease has however, been described in other Australian natives by Taplin (1886) who considered that they did not contract it easily, although Curr (1886) refers to an epidemic in the Cape York peninsula which was accompanied by a high mortality. Cleland (1928), referring to the aborigines of Queensland, thought that the disease was comparatively rare. More recently Snow (1952) made no reference to its occurrence in aborigines, in his review of the history of this disease in Western Australia.

From the information available it would appear that scarlet fever has probably run a similar course in the aborigine to the white population and that it probably was relatively common in most aboriginal groups and accompanied by a fairly high mortality at the end of the last century. More recently its incidence has diminished both in the white and aboriginal population, and it is now a rare disease which when present is seldom accompanied by severe systemic upsets. It is a natural decrease in the prevalence of the disease rather than any specific immunity which probably accounts for its rarity in aboriginal groups.
Rheumatic Fever. Despite a careful search for both past and present occurrence of rheumatic fever, I could find no evidence of this disease in the Western Desert aborigines, in either the nomadic or urban groups, although six cases occurred in the white population who are similar in numbers to the aboriginal population of this region. It is difficult to account for this apparent difference between the white and the native population in this area, but it has been both my own and other doctors' experience in this region, that acute rheumatic fever is comparatively common in the white population but extremely uncommon in the aborigines. The low incidence of this disease in this area has also been confirmed by Davidson (1952) and Lewis (1962) during a brief paediatric survey of this area. Discussions with Webb (1964), Cook (1962) and Allen (1962) confirm these findings, although Allen stated that he had seen one case of rheumatic fever but that was in an aborigine from outside the Western Desert region. It is also notable that acute rheumatic fever is relatively common in the part or half caste aborigines of this region.

Rheumatic fever has however, been reported in other Australian aborigines, but the references are often poorly supported and it is difficult to deduce the relative frequency of this disease. Cleland (1928) in an attempt to
form some assessment of this incidence quoted Gorrey as stating that rheumatic fever was common in the aborigine and Illingworth who commented that he had only met one case with a post traumatic mitral stenosis, these practitioners referring to Queensland and New South Wales respectively. Cleland himself details a number of "heart conditions" which appear to be purely murmurs, with no aetiology described and with no evidence that they were due to rheumatic fever. Basedow (1932) referring to the South Australian aborigines, did not differentiate between acute rheumatic fever, rheumatoid arthritis and muscular or joint pains of uncertain aetiology, tending to consider all of these in the one group. More recently Ford (1942) noted no cases of either acute rheumatic fever or post-rheumatic cardiac involvement in the natives of the Bathurst and Melville Islands, but Binns (1945) in the Northern Territory, stated that he had treated five cases of acute rheumatic fever but comments that two of these patients had yaws. Elphinstone (1960) noted no cases amongst the Kimberley natives and Crotty et al (1961) in the Northern Territory stated that one death occurred from acute rheumatic carditis and three from chronic rheumatic carditis. However it is not specified whether these were full-blood or part-blood aborigines. Gandevia (1967) in a survey of chronic respiratory disease in the Pintubi and Walkiri aborigines noted no evidence of post rheumatic heart disease. Jose et al (1969) reported a relatively
high incidence of rheumatic carditis in aboriginal children in Queensland, but did not indicate their degree of caste, and it is probable that very few of the total survey were full-blood.

**Staphylococcal Infections**

Disease due to this group of organisms is comparatively common in both the Western Desert and other groups of aborigines. The most common types are the skin infections. Both carbuncles and boils were seen in the Western Desert region in roughly the same frequency as they occurred in the white population. The clinical course was identical in both groups. There was however, a significant difference in the lack of carbuncles in the aborigines as compared with the white population in whom it was particularly common in this climate. Staphylococcal impetigo was particularly common in the aboriginal children and much more frequently seen than in the white population.

**Staphylococcal Skin Infection** has been observed in other aboriginal groups. Foelsche (1881) stated that carbuncles and boils were frequent occurrences in the Northern Territory aborigines and Curr (1886) quoted Garson as stating that large blind boils were particularly common in children of 14 years of age. Cleland (1928) cites both Jackson and Armour in personal communications as considering that boils are as common in the native as in the white
person while Basedow (1932) stated that they were a common occurrence in the aborigines of South Australia. No more recent statement has been made in the literature.

**Staphylococcal Pneumonia** is probably more prevalent than realised in aboriginal children, although it has not been specifically described, and I had personal experience of two cases in Western Desert aborigines, one of whom died (aged two years of age), while the other survived (aged three and one half year). In both of these cases the organisms were cultured after transference to Perth. Discussions with paediatricians confirmed that this is not an infrequent occurrence. There are however, no other existent references to this disease entity in other aboriginal groups.

**Staphylococcal osteomyelitis** has not been reported in any of the older or more recent literature, except by Crotty et al (1960) who considered it to be the cause of death in one of the aborigines in the Northern Territory. I have seen no cases in the Western Desert region, and no evidence of old osteomyelitic disease, although it is possible that some of the amputees may have had their operations for this reason. Mackay (1938) reported osteomyelitis in aboriginal skeletal remains.
Gonococcal Infections

This disease has traditionally been considered to be rife amongst the aborigines. However I saw no evidence of it being present in the Western Desert aborigines, there being no cases of salpingitis, ophthalmitis or urethritis during the time I worked in this area. Mann (1954, 1957) confirmed the lack of ophthalmological gonococcal disease in the Western Desert aborigine.

Gonorrhoeal infections have however, obviously been extremely frequent in other aboriginal groups and in the past Brienl (1912) and Holmes (1913) referring to the Northern Territory, considered the disease to be endemic while Basedow (1919) referring to South Australia, stated that gonorrhoea was rampant in both sexes in this area. Cleland (1928) in quoting a number of communications from various parts of Australia, commented on the apparent prevalence of the disease in the Australian aborigines and stated that orchitis, balanitis, and arthritis were particularly common complications, although salpingitis and endometritis appeared to be rare. Hackett (1936) stated that he had demonstrated microscopically gonorrhoeal ophthalmitis in 100 aborigines and that it is most frequent at Alice Springs, Darwin and Victoria River, but relatively uncommon in the Musgrave Ranges, from which he deduced that it must have been introduced into Australia by the early settlers and had not yet fully penetrated into the interior.
Cook (1936) considered the disease to be common in Western Australia and Binns (1945) noted eleven cases in the Northern Territory of which two had orchitis, while Fryberg (1966) reported a number of cases at Palm Island. It would appear that the incidence of this disease had considerably reduced in the post war era and this has almost certainly been due to the use of antibiotics for respiratory and enteric infections in aborigines, which has also cured any incidental gonorrhoea.

Over the past decade relatively few cases of gonorrhoea have been reported to the Public Health Department from any of the Australian population, although in common with other countries over the last three years there has been a rapid rise in the incidence. Unfortunately it is impossible to establish whether or not this is affecting the aboriginal population, as race is not stated on the notification form. In my own personal experience in the urban aborigines of Western Australia notably around Perth, the incidence of gonorrhoea was increasing and one would expect a similar increase maybe occurring in the full-blood population.

Meningococcal Infections

The only report of meningococcal infection in an aborigine was that by Binns (1945) who noted one case of meningitis. I have not seen any disease myself from this
source in the Western Desert area, and Snow (1955) made no mention of any specific effect on aborigines in his review of meningococcal infections in Western Australia.

**Haemophilus Influenza Infections**

In my opinion this is probably not an uncommon secondary infection in influenzal cases, and may well be present in other forms of viral infections as a few cases which I have managed to have examined bacteriologically from the Western Desert region have shown the presence of this organism. There are no reports from any other sources as to the occurrence of haemophilus influenza, either in association with other influenza infections or as a secondary infection in bronchitis and pneumatic patients. It is possible however, that this may have been the major factor in the 1919 pandemic of which Cleland (1928) states as having produced a 25% mortality amongst the native patients.

**Diptheria**

Diptheria has not been reported in Western Desert aborigines and I have seen no evidence of it. It is, however, an uncommon disease in the whole of Australia and was not seen in any of the white population of the Western Desert region either, although epidemics of this disease have occurred in Australia as recently as 1955 when 480 cases were reported. No specific mention has been made of its
occurrence in aborigines apart from one case in an aboriginal woman reported by Holmes (1913) in which she recovered and in which the course of the disease was not significantly different from that seen in the white population, and by Kirkland (1939) who stated that the disease was endemic in the Northern Territory.

Although a varying number of aboriginal children have had triple antigen injections, there is probably a majority of the adult population who have never had immunisation, and there must be a large aboriginal population at risk from this disease in the community.

**Salmonella Infections**

I have seen no evidence of salmonella, typhoid or paratyphoid disease in the Western Desert aborigines. There is however, considerable information available to designate it as a major cause of mortality in earlier days of settlement of Australia in both the white and native populations. Smith (1878) states that this was a common cause of disease in the aborigines and this has been substantiated by Curr (1886), while Cleland (1928) gives more detailed accounts of the disease in four aborigines from which it would appear that the clinical signs and symptoms and course are identical in both the white and aboriginal races, and Kirkland (1939) notes the commonness of the disease in the Northern Territory. The major
epidemics in the Western Desert area occurred in the early part of the century in Kalgoorlie, Coolgardie and Norseman. There is no history available as to how this affected the aboriginal population and Snow (1956), in the history of typhoid in Western Australia, gives no details of the effect apart from the Caucasian population.

**Coliform, Proteus and Pseudomonas Infections**

These, in my experience, are an uncommon cause of disease in the aborigine of the Western Desert. However, urinary tract infection in the female, particularly common during pregnancy, is usually associated with E.coli and in addition I have seen one urinary tract infection in an elderly female diabetic in which pseudomonas was present. I have never seen any other systems involved by any of these organisms, although it is worth remembering that Rountree in his survey of nasal flora found proteus to be particularly common in aborigines. There are no other records of the prevalence of these diseases due to this group of organisms in other groups of aborigines.

**Shigella Infection**

Bacillary dysentery is apparently common in all groups of aborigines. In the Western Desert region they formed approximately 10% of all cases I saw, the majority of these (90%) occurring in children under 10 years of age, of which 60% were below the age of 4 years. The disease
is seen 3 times as frequently in the town aborigines as in the more outlying tribes, although this may well be due to the greater ease with which medical attention can be sought. The disease pattern is sporadic, reaching epidemic proportions during summer months. It is noteworthy that gastro-enteritis and bacillary dysentery are frequently complicated by bronchial pneumonia and vice-versa. In those children that die there are marked inflammatory small bowel changes, frequently gastric mucosa changes, and widespread bronchial pneumonia. In the cases I admitted (321), there were 7 deaths and the recurrence rate was high. A chronic state of apparent damage to the small bowel mucosa is not infrequent and this is associated with a long lasting malabsorption syndrome in which inanition, anorexia and anaemia are common complications.

The high incidence in Australian aborigines has been present ever since the time of settlement and has been noted by Curr (1886) who referred to its frequency in the Eukla district, Smith (1878) and Taplin (1879). In 1923, Cilento mentioned that frequent outbreaks occurred in Queensland. Basedow (1919) noted that almost the whole aboriginal population of the north-east of South Australia suffered from these gastro-intestinal disorders, while Cleland (1928) mentioned that, of 88 deaths in aborigines, 7 were due to dysentery. The incidence is still high as shown by Cook
(1949) who, referring to natives of the western parts of Western Australia, mentioned it as one of the major causes of morbidity and mortality in this region. Ford (1942) stated that the disease was frequent in the Bathurst and Melville Islands, but Binns (1945) also working in the Northern Territory, noted that it was only the cause of 1% of his admissions. Schneider however, (1946) also in the Northern Territory, agreed with Ford as to the frequency of the disease. Davidson (1950) stated that 115 admissions in the Kimberley region were due to gastro-enteritis and dysentery and these figures were supported by Elphinstone (1960). Crotty et al. (1961) in a series of 175 postmortem examinations, stated that the cause of death in 27 of them was due to either bacillary or unspecified dysentery, all the deaths except one occurring in children under 4 years of age. Surprisingly, Davidson (1957) stated that "intestinal infection of the Western Desert natives is not common". This has not been my experience, and one presumes that this false impression of the incidence of this disease was due to his extremely brief stay in this area.

Leprosy

This disease was introduced to the Australian continent with the immigrant Chinese and Kanaka labourers in the 19th century, who rapidly passed the disease to the aborigines. The disease was first reported in an aborigine
by Ashburton Smith (1892) in Queensland. Other cases in the Northern Territory and Queensland were reported in 1893 and 1894 by McKillop. In 1906 Smith drew attention to the fact that leprosy had been detected in five full-blood aborigines in the Alligator River district. The disease continued to spread, and further cases were noted by Breinl (1912), Holmes (1913), and Breinl and Holmes (1941) in the Northern Territory. It also spread into the northern parts of Western Australia, and continued southwards, and by 1928 Cleland had noted numerous cases and commented on the frequency. For some reason, the disease never passed beyond the 20° latitude in Western Australia, and attempts to confine it north of this level were enacted by Parliament in 1941 in which travel by aborigines across this boundary was forbidden without special departmental consent, a rather ridiculous and unnecessary law that was rescinded in 1951. This follows... the recommendation of the Bateman report in 1948 which stated that the disease had apparently remained climatically restricted to the coastal regions above this parallel and that there was evidence of considerable movement of aborigines across the boundary despite the law. Ford (1942) and Binns (1945) noted a few cases in the Northern Territory. Cook (1949) in discussing the northern area of Western Australia was in agreement with Davidson (1960) that a considerable decrease in the incidence of the disease had occurred with the introduction of new chemotherapeutic agents. Elphinstone (1960) stated that there
were only 484 known cases of leprosy in the aboriginal population of the Kimberleys. Davidson (1960) stated that the aboriginal population, which he regards as a non-immune population to which the disease has only recently been introduced, show a 3 to 1 ratio of lepromatous to tuberculoid forms, which is the opposite to other countries in which the disease is established. He also concluded that amongst the aborigines there is no evidence that it is contracted in infancy or childhood and that prolonged and repeated contact is conducive but is not essential for infection to occur. He also observed that the white population, allowing for the few opportunities of contact, show a relatively high incidence of infection, the types of infection being similar to the proportion noted in the aborigines. No leprosy has ever been reported in the Western Desert area and I have seen no evidence of this disease myself, although Elphinstone (1963 and 1971) in a review of the health of the aborigines in the Sandy Desert area did note some cases of leprosy. The Sandy Desert area lies north west of the Great Western Desert and geographically is not separated by any major distance and it is possible that leprosy could be introduced to the Western Desert natives. The true incidence of leprosy is well documented in the annual reports of the Public Health Commissioners of each Australian State.
**Tuberculosis**

This disease is uncommon in the Western Desert aborigines as I have only seen 2 active cases, both pulmonary, of whom one was diabetic and also had renal tuberculosis. This low incidence is quite surprising in view of the fact that the local white population has one of the highest incidences of tuberculosis in Australia, due to the association between tuberculosis and silicosis in the gold mining industry. In the Western Desert aborigine, the low incidence has also been confirmed by Davidson (1957) and Lewis (1963) in the brief examinations in groups of these aborigines.

Tuberculosis has however, long been regarded as an important cause of mortality in the aborigine, particularly by earlier authors many of whom were probably including other forms of acute and chronic pulmonary disease in the statistics. Some assessment of the frequency in which the disease was noted in the aborigines may be gained from Curr (1886) who quoted Parkes as stating that consumption was prevalent in the York area of Western Australia while in the Eucla district, the chief causes of death were consumption and dysentery, and he also noted that in the Beltona tribe of South Australia tuberculosis had reduced the population from 115 to 50 during the years 1865 and 1883. In 1892, Watson demonstrated specimens of tuberculosis in aborigines and McKillop (1892) stated that consumption was
the most common cause of death in the aborigines of Daly River district. Stirling (1896) during the Horne expedition, observed many cases and stated, "The extreme susceptibility of the Australian race to tuberculosis when brought into contact with civilisation has frequently been observed". He also stated that cavitation of the lungs was infrequent in the aborigines, a view with which Cleland (1912) agreed when he discussed some aspects of the pathology of the disease in natives. In several papers between 1912 and 1915, Breinl and Holmes commented that the disease only occurred in those aborigines who had contact with civilisation and did not occur in the bush native. The disease in general ran a rapid course, proving fatal in a few months and they considered that the disease had largely been introduced by the Chinese immigrants. Basedow (1919) stated that in the north-east of South Australia he found 28 of 250 natives examined to have active tuberculosis, and that probably the same number had it in an inactive form, so that some 25% were probably infected. These high frequency rates received some support from Cleland (1928) who demonstrated the continued prevalence of the disease by quoting numerous personal communications and by examining material from pathology museums and hospital records. Binns (1945) noted that tuberculosis accounted for 5% of the admissions to his hospital, and both Ford (1943) and Cook (1949) stated that the disease was common in the
aborigine. However, the most accurate survey was conducted by King, Edwards and Gibson (1951) amongst the aborigines of the north of Western Australia. Using mass radiography techniques, they examined 3,209 full-blood natives and discovered only 15 with evidence of infection. Of these, 12 had fibrotic lesions, 1 was a mixed lesion and 2 were active lesions. The incidence was therefore less than 5 per 1,000 of the population, a lower incidence than in the general white population of Australia. The low incidence was certainly not due to lack of contact with the organisms, as of 2,657 natives tuberculin tested, 1,191 were positive reactors, and the conversion rate was higher in the town than desert natives as one would expect. It would appear from these reports that the aboriginals have in fact, developed considerable immunity to the disease and it may well be that the earlier reports of the prevalence were exaggerated or the disease was in fact, more common and the mortality accentuated by concurrent alcoholism and other debilitating diseases. Environmental factors may also play a major part as Crotty (1960) noted that tuberculosis was a cause of death in 19 aborigines, 17 of these being pulmonary and it is difficult to account for relatively high death rate in the Northern Territory as compared with King's et al findings in 1951 in the north of Western Australia, unless there are still susceptible groups present in the aboriginal population.
Granuloma Inguinale

This is another disease which has apparently undergone a dramatic reduction in its incidence in Australian aborigines and during the five years I was in close contact with the Western Desert aborigines I saw no cases.

Curr (1886) stated that the disease was prevalent around Newcastle in 1879 and also stated that previously had been much more acute, while Goldsmith (1901) noted it in Europeans, Asiatics and aborigines of South Australia, and although he considered it to be of venereal origin, he did not think it was highly contagious. Further references by Smith (1906), Higginbotham (1909) and Breinl (1912) all emphasise the widespread distribution of the infection. Holmes (1912 and 1913), Breinl and Holmes (1915) and Breinl and Priestly (1916 and 1917) published further accounts of the disease in aborigines of the Northern Territory and noted that 18 cases were treated at Darwin Hospital in 1913. In 1919, Basedow stated that it was frequent although it was possible that some of his cases were in fact, yaws, and in 1921 Mapleson discussed the treatment and considered that some of his cases had been cured. Nonetheless, the disease apparently continued unabated as testified by Cleland (1928), Hackett (1936) and Basedow (1932).
Even in 1945 Binns noted that 50% of the admissions to his hospital in the Northern Territory had evidence of infection, both sexes being equally affected and further confirmation of the frequency of the disease was given by Ford (1942), Musso (1921, 1925). Schneider (1946), Cleland (1947) and Cook (1949) in differing parts of Australia. Following these reports there seems to have been a rapid decline in the disease, as neither Davidson (1957), Mann (1957) or Lewis (1962, 1963) noted any cases during their visits to the Western Desert area. The decrease in other areas where the disease had been rife, was noted by Elphinstone (1960) who states that the Kimberley region had shown a marked decline of incidence from when he had visited it 12 years previously, the drop having been so dramatic that he had only seen 3 cases in the previous year and he considered the decrease to be due to the widespread use of antibiotics for other infections. It is probable that the widespread use of antibiotics in other areas for other diseases had accounted for this remarkable decline in the disease.

Pertussis

I have seen both sporadic cases and minor epidemics of whooping cough in the Western Desert aborigines affecting both the urban and desert natives. Clinically the courses of the disease in children and adults is identical in both native and white population and the distribution is similar,
the majority of the patients being children of school or immediate pre-school age. Of 33 native children, 26 were between 4 and 7 years of age. The aboriginal children did however, show a higher incidence of secondary chest infection, the most common being broncho-pneumonia, but in all cases satisfactory recovery occurred.

Whooping cough in epidemic form has been previously reported in aborigines by Smith (1878) and Curr (1886), although they gave no details of the disease as it affected aborigines. Brienl (1912) noted that whooping cough appeared sporadically in the aborigines of the Northern Territory and affected both children and adults. Cleland (1928) referred to an epidemic on Thursday Island in 1922, again without any details, though he cites Parker of Victoria as saying that the native's resistance to whooping cough is weak. Kirkland (1939) also reported sporadic outbreaks in the aboriginal population.

No more recent reports are available on the current prevalence and affects of this disease in any area.

Melioidosis

Cook (1961, 1962) found no evidence of this disease in the Cape York area in 208 aborigines tested, but Crotty et al (1963) reported 2 cases in the Northern Territory.
There is no evidence of this disease in the Western Desert area.

Clostridial Infections

These are comparatively common infections in Australia in the white population, but none have been reported in the aborigines in any detail. I have never seen clostridium welchii infection in any aborigine but one of the amputations in the Western Desert area was supposed to have been performed for this disease. C. Tetani infection was the cause of death in two of Crotty's (1960) post mortems, one of which was tetanus neonatorum which they state was the only case of its kind of which they have ever heard. Sutton (1966) reported a high carrier rate in aborigines for C. welchii.

In the Western Desert area I have never seen tetanus in an aboriginal patient, although Roberts (1959) had previously observed one from this area. Botulism has never been reported in an aboriginal.

Other Bacillary Infections

Other bacillary infections such as cholera, plague, tularaemia and anthrax have been well documented in regard to their history in Australia, particular by Cumpston (1926) and by Snow (1951). In none of the literature is there any reference to the affect of these diseases or prevalence of
the disease in aborigines. Similarly brucellosis, which has been reviewed by Snow (1954) has not been reported in an aboriginal patient, although in the area of which it is prevalent, that is the south-west of Western Australia, there are very few full-blood aborigines. None of these diseases has been seen by myself in the Western Desert area or have been reported in this area by any other authority.
DISEASES OF THE CARDIO-VASCULAR SYSTEM
THE HEART

The heart is probably more commonly affected in the aborigine by disease processes than is generally realised. I found chronic heart failure to be relatively common in the elderly Western Desert aborigines where it is associated with chronic bronchitis and emphysema, and it is probable that this is the form of heart disease with dropsy reported by Smith (1878) in the Murray River natives, and some of the cardiac cases reported by Cleland (1928). However, I found chronic left heart failure to be uncommon. I have seen no cases in the Western Desert region. Binns (1945) noted two cases of essential hypertension without cardiac decompensation in the Northern Territory, and I have seen 3 similar cases in the Western Desert area. Crotty (1960) however, noted the same condition with congestive cardiac failure in the elderly aborigines of the Northern Territory and he also stated that one death was due to myocardial infarction. I have not seen any evidence of myocardial insufficiency in the aboriginal population of the Western Desert native, and recently Woods et al (1966) in an electrocardiographic survey of part of this area reported very few abnormalities.

Bacterial endocarditis has been reported by Crotty (1960) in a middle-age native, and he also observed a woman who had pericarditis, the aetiology of which he did not present. I have however, noted mild pericardial
effusions in several children dying of pneumonia at post mortem examination, but whether in fact, true pericarditis existed is difficult to say.

There have been no reports from other areas and I have seen no evidence of syphilitic disease of the heart, pericardial effusions, apart from those mentioned above, pericardial tumours, myocardial degeneration, myocardial inflammation or myocardial tumours in aborigines.

THE BLOOD VESSELS

I find hypertension to be extremely rare in full blood, Western Desert aborigines and artherosclerosis to be present in only mild degree of those post mortems which I conducted. I have never seen any evidence of Monckeberg sclerosis or true arteriosclerosis being present, although Crotty (1960) stated "Arteriosclerosis is not uncommonly found at autopsy of elderly aborigines", but it is probable that he was dealing with a much more urbanised group of aborigines and some of these may have been part-caste. In the various surveys conducted of aborigines' blood pressure by Nye (1937), Casely-Smith (1959), Abbie and Schroeder (1960) and Von Dongern et al (1962), there was no report of any evident arteriosclerosis or marked hypertensive disease. Similarly, there had been no reports of inflammatory arterial diseases having been reported
amongst the aborigines and this also applies to the neurotrophic vascular disorders. Varicose veins were reported by Basedow (1932) as being common amongst the South Australian aborigines, but this is not so in my experience in the Western Desert area. The veins of both the upper and lower limbs in this region are prominent, due to lack of subcutaneous fat and with age they become more tortuous, but I have not seen any case in which incompetence of the long and short saphenous vein, or incompetent perforating veins could be demonstrated. Similarly, I have seen no evidence of gravitational ulcers or other trophic skin changes in these people due to venostasis.

Phlebothrombosis, thrombophlebitis and migratory plebitis or phlegmasia alba dolens have never been described nor seen by myself, except for two cases of milk thrombophlebitis following blood letting ceremonies in male adult aboriginals in the Western Desert region.

THE LYMPHATICS

This system may be involved in acute infective processes, giving lymphangitis and lymphoedema, but none of the primary lymphatic disorders have either been seen by myself in the Western Desert region or described in other groups of aborigines. Similarly, no tumours of the vascular system, either benign or malignant have been described.
DISEASES OF THE RESPIRATORY SYSTEM
THE NOSE AND PARANASAL SINUSES

I have described the frequency with which these organs are affected in the infectious diseases, both under the exanthemata and respiratory infections. In my personal experience it is common to find chronic paranasitis, chronic nasal congestion in adults, often associated with hypertrophic changes in the turbinates and causing nasal discharges in all groups of Western Desert aborigines.

Epistaxis is in my experience extremely uncommon and I have never seen a nasal polyp in an aborigine. I could find no examples of allergic rhinitis or nasal congestion due to allergy in the Western Desert region. Binns (1945), Hughes (1966) and Malekerinos (1969) have also commented on the frequency of nasal and paranasal infection in the Northern Territory and other aboriginal groups. Neither they nor I have noticed any of the complications which may accompany these diseases, namely osteomyelitis, orbital cellulitis, meningitis, frontal lobe abscess or cavernous sinus thrombosis.

Many of the earlier authorities, Brienl and Holmes (1915), Cleland (1928) and Hackett (1936) all commented on the destruction of the nose, variously diagnosed as being due to syphilis or yaws, (but as previously mentioned, almost certainly due to yaws), but I have seen no examples
of this in the Western Desert area, although there was one native woman who had lost the tip of her nose, but she claimed emphatically that the defect was due to the tip being bitten off in a domestic fight. No neoplasms of the nasal and paranasal areas have been described.

THE LARYNX

Disease of the larynx is uncommon in the Western Desert region and I have only seen four cases of acute laryngitis in children, all associated with laryngotracheobronchitis, and two cases of chronic laryngitis in elderly aborigines. I have seen no evidence of specific laryngitis in this region and there are no reports of the incidence of these diseases in other groups of Australian aborigines.

THE TRACHEA AND BRONCHI

As I have previously described under Infectious Diseases, these organs are frequently diseased and form a major cause of morbidity in the aborigines of the Western Desert area. Acute bronchitis is an extremely frequent complication of any of the exanthemata and other viral infection. In my own experience it is present in 90% of patients with measles. Tooth and Lewis (1963) reported its occurrence in 80% of aborigines with measles and I have also noticed its presence in 50% of aboriginal patients with chicken pox.
The high frequency of bronchitis has been reported by Wilson (1835) who stated that acute bronchitis was very prevalent in the Northern Territory. Smith (1878) referring to the natives of Victoria, Basedow (1919, 1932) referring to the natives of South Australia, and Cleland (1928) in reference to the aborigines of Queensland, all confirmed its frequency. Binns (1945) stated that half of his admissions for respiratory infection were due to acute bronchitis and Davidson (1957, 1958) and Elphinstone (1960) commented on the high incidence of this disease in the north of Western Australia.

In my experience children not uncommonly have laryngo-tracheo-bronchitis and Crotty and Webb (1960) noted one death due to obstruction from this disease.

Chronic bronchitis associated with emphysema is extremely common in the Western Desert area and in my estimation it is present in 80% of aborigines over the age of 45 and is the major cause of morbidity in this group in this region. Its commonness in other regions has been shown by Crotty and Webb (1960) who noted that eleven of the deaths (forming almost one quarter of the patients in their series over 60 years of age) were due to chronic bronchitis and/or emphysema, but they make the pertinent comment "This does reflect their frequency in elderly aborigines in whom they are an extremely frequent source
of disability. These conditions were present in many natives who died from other causes."

Bronchospasm is a frequent accompaniment of acute bronchitis, but I have never observed bronchospasm in an aborigine from a true allergic cause, although Illingworth (1929) noted what he considered to be a case of asthma. No substantiated references to asthma have appeared in the literature.

The lungs are most commonly affected by pneumonia and broncho-pneumonia, both of which have already been discussed, as have tuberculosis, hydatid disease and neonatal pneumonia.

Pulmonary oedema was present in six children in the Western Desert area associated with influenza, but neither pulmonary infarction nor pulmonary haemorrhage have been noted in this area by myself. Crotty and Webb (1960) did note one case of haemorrhage into a cystic lung in the Northern Territory, and also six cases of lung abscess in adults, although the aetiology is not stated.

Emphysema as noted above, is commonly associated with bronchitis, and bronchiectasis is not an uncommon accompanying feature in these cases. Frequency of emphysema has also been noted by Basedow (1932) and
Recent surveys have in fact emphasised the widespread respiratory disease which exists in the Australian aborigine. Crotty and Webb (1960) have already been mentioned. Gandevia (1965 and 1967) in a survey of the Western Desert aboriginals, found an extremely high incidence of chronic respiratory disease and Maxwell et al (1968) in a survey of 338 children in Central Australia and the Northern Territory, showed that 37% had some clinical abnormality, 30% had radiological abnormality in the lungs and 16% had actual pulmonary consolidation at the time of examination. They also noted that 69% of these children had previous recurrent ear, nose and throat disease.

There is no reference in the literature to silicosis or asbestosis in full-blood aborigines, probably because of the small number employed in these industries, and again because they only work for a limited time. I have noted one case of bronchogenic carcinoma in an elderly aboriginal woman. No other lung carcinomas have been reported from other areas, except by Cleland (1927) who described a case in South Australia.
Pleural Cavity

Pleurisy is a not uncommon accompaniment with lobar pneumonia in my experience in the Western Desert area, and pleural effusion occurred in four cases. Empyema is rare and only one case was observed in the Western Desert area and this was secondary to staphylococcal pneumonia. I have seen no cases of either spontaneous or secondary pneumothorax in this area.

Cleland (1928) did note several cases of pleural effusion in aborigines, but he did not comment on the aetiology, while Basedow (1932) reported several examples of both pleurisy and empyema, but again without details.

The Mediastinum

There have been no reports of disease of the mediastinum in the literature, but mediastinum lymph gland involvement occurred in a case of bronchogenic carcinoma reported above, in the Western Desert area.
DISEASES OF THE DIGESTIVE SYSTEM
THE MOUTH

The only common disease of the mouth in the Western Desert aboriginals in my experience is thrush in children, which has already been described. Gums commonly show pyorrhoea in the elderly aborigines in this area, but alveolar abscesses are rare. A marked feature in the Western Desert aborigines is the attrition which occurs in their teeth, but dental caries is common in the urban aborigines, although rare in the desert native. Glossitis is also not uncommon in the elderly aborigines in this region and is probably due to subclinical malnutritional states, as it usually responds to adequate hospital diet. This has also been referred to by Davidson (1957) who noted several cases of glossitis in the Western Desert area, but was unable to attribute any particular cause to it.

Diseases of the mouth which have been reported by other authors are, gangrene and carcinomas, which have been noted by Brienl and Holmes (1915), Stirling (1911), Basedow (1932) and Hackett (1936), although it is possible that some of these cases were in fact yaws. There are no recent records of either of these diseases and similarly Vincent's angina, lichen planus and leucoplakia have not been reported either. The attrition of the teeth has also been noted by Stirling (1896), Basedow (1932), Gray (1933), Hackett (1936) and Wilson (1954). Hackett (1936) also
reported bleeding gums in a Central Australian aborigine
due, he considered, to vitamin C deficiency, but I
personally have not seen any evidence of scurvy in the
Western Desert aborigines.

THE SALIVARY GLANDS

The only disease of the salivary glands I have seen
in the Western Desert area apart from mumps which have
already been discussed, was an ascending parotitis in an
elderly comatose female. No other infections have been
reported, either in the Western Desert area or in other
groups of aborigines, except for a case of chronic
enlargement of the parotid gland noted by Gray and
Cleland (1932).

THE PHARYNX

Tonsillitis has already been described by myself
in the Western Desert area, and particularly in children,
chronically infected hypertrophic tonsils (palatine) and
adenoids are particularly common and associated chronic
pharyngitis, sinusitis, bronchitis and bronchiectasis.

Cleland (1928) also drew attention to the frequency
of tonsillitis in aborigines and Crotty (1960) reported
carcinoma of the pharynx as the cause of death in one of
the Northern Territory aborigines.
THE OESOPHAGUS

I have seen no disease of the oesophagus in the Western Desert aborigines and can find no reports referring to this area.

Crotty (1960) noted oesophageal varices associated with cirrhosis of the liver in a patient in the Northern Territory.

THE STOMACH

In the Western Desert region, diseases of the stomach are extremely rare apart from the common gastro-enteritis which has already been described. I have never seen any evidence of peptic ulceration in this area and have not seen any of the complications of this disease or of hiatus hernia.

Matthew (1889) stated that "indigestion" was common in aborigines and Basedow (1932) also commented on numerous cases seen, although from his brief description, many of them were apparently infective in origin, and probably cases of gastro-enteritis, although it is possible that acute and chronic gastritis may have been more common than other areas where liquor was freely supplied to them. No congenital abnormalities have been noted but Cleland (1928) reported two cases of carcinoma of the stomach in the Northern Territory and Crotty (1960) in the same area, noted
secondary neoplastic disease of the liver which he considered was probably due to primary in the stomach, and I also have seen one case of carcinoma of the stomach, in an elderly male aborigine in the Western Desert area. Although no further cases have been reported in the literature, practitioners with whom I have personally communicated, both Thomas (1958) and Allen (1960) are of the opinion that neoplastic disease of the stomach is relatively common in this race, but could give no absolute figures.

SMALL INTESTINE

I have never seen any diseases of the small intestine apart from infective diseases which have been previously detailed, in the Western Desert aborigines. Chronic changes in the mucosa in infants following acute bacillary infections have been noted previously and both by myself and also by Lewis (1962), and these account for the malnutritional disease that may follow acute attacks of enteritis. More recently Harris (1970) has studied these changes in South Australia and shown that they are associated with a disaccharide intolerance. Edwards (1970) in the Walgett area has found the same pattern of malnutrition following repeated attacks of enteritis. There are no reports of regional enteritis having been noted in any aboriginal group.
THE LARGE INTESTINE

As has been previously detailed, the large intestine is frequently involved in infective disease of both bacillary and parasitic origin, although in the Western Desert region parasitic disease is rare. I have never seen non-specific ulcerative colitis or diverticulitis of the colon in this group of aborigines. In my experience acute appendicitis in the full-blood aborigine is also extremely rare, as I have never seen a case in the Western Desert region. I have in fact removed the appendix in three patients, all full-blood aborigines who complained and gave symptoms of acute appendicitis, but certainly there was no evidence of infection in the removed organ, and I believe all three of them had non-specific mesenteric adenitis which was probably associated with a viral infection.

Basedow (1932) stated that he observed two cases of appendicular colic in native women in South Australia, although from his description it is much more likely that they had enteritis. Cleland (1928) quotes Illingworth as stating in a personal communication, that he saw an acute appendicitis in an aboriginal male, but however cast some doubt on his statement by also declaring that he had never seen a case of "acute abdomen" in an aborigine. The only case of neoplasia reported of the large bowel was noted by Crotty (1960) when he stated that carcinoma of the colon
was present in one of the post mortems he performed in the Northern Territory.

**ANAL CANAL**

The only disease of the anal canal I have observed in the Western Desert aborigines are several mild cases of haemorrhoids, but these are infrequent and in one patient I observed a third degree tear which had occurred in a previous pregnancy, and had left her with slight faecal incontinence. This was subsequently repaired without any problems. In the older literature, reference to yaws and granuloma inguinale affecting the anal margins, have been presented. No neoplastic disease of the anal canal has been published, but Basedow (1932) reported a case of fistula-in-ano in an aborigine.

**THE LIVER AND BILIARY TRACT**

Disease of these organs are the most frequent cause of acute abdomen in the Western Desert aborigines in my experience. Cholelithiasis is surprisingly common. I have seen four cases in the Western Desert area and know of two more from this region seen by Dr. Roberts (1963). In two of these patients the main symptoms were biliary colic and of the other four the presentation was that of cholecystitis. One also had an ascending cholangitis. I also observed two cases of neoplasia of the liver in this region, one being a metastatic deposit from a primary carcinoma of the
stomach while the other was considered by the Department of Surgery of the University of Western Australia, to be a primary carcinoma of the gall bladder with secondary hepatic deposits. Both these patients were elderly male aborigines.

Involvement of the liver and biliary tract in infectious diseases, such as infective hepatitis, hydatid disease, amoebiasis and malaria, in both the Western Desert and other groups of aborigines has been previously presented. There are no reports of pyogenic abscesses, portal pyaemia, or portal veinthrombosis in the literature. Curr (1886) reported that the Gippsland aborigines were fast dying out from diseases of the liver and enlargement of the liver was prevalent in Cape York natives. He gives no details from which we can diagnose the disease, although it is probable in the latter area, malaria may have been responsible. Basedow (1919) frequently noted hepatomegaly in the aborigines of South Australia, and as previously discussed, this was probably more likely to be infective hepatitis than as he considered congenital syphilis. Cleland (1928) reported a 10 year old aboriginal youth with hepatomegaly due to congestive cardiac failure. No further details of this case are available. Cleland (1928) also reported several specimens of cholelithiasis in the Adelaide University Pathology Museum.
Tumours of the liver have been reported on a variety of occasions. Watson (1895) demonstrated an "adenoma of the liver" from an aborigine. This was re-examined by Cleland (1928) who thought it was a carcinoma probably secondary to primary bowel tumour. Bancroft described a carcinoma of the liver he saw in 1913. Crotty (1960) noted two cases of carcinoma of the liver in the Northern Territory, one of which he considered primary and the other a metastatic deposit.

THE PANCREAS

Apart from diabetes mellitus I have seen no evidence of pancreatic disease in the Western Desert aborigines and there are no reports on this subject from any other areas.

THE PERITONEAL CAVITY

Peritonitis is in my experience extremely rare in the Western Desert aborigines. I have never seen a case of primary peritonitis and the only secondary peritonitis with which I have been involved is that associated with salpingitis which has been previously described as relatively uncommon. It is not surprising that the incidence is low due to the lack of appendicitis, gastric and duodenal ulcers, torsion of the bowel and other causes of peritonitis. Cleland (1928) did refer to a case of tuberculous peritonitis and hydatid disease has
already been considered. Bowel obstruction appears to be extremely rare in the aborigines. I have seen none in the Western Desert natives and the only report of bowel obstruction is that by Walker-Smith (1969).
In my experience these are much more rare in the aboriginal population than in the white population. The umbilical infant type of hernia always regresses spontaneously and amongst the adults of the Western Desert region I have only seen two inguinal hernias in males, both of which repaired satisfactorily. There are no extant accounts of complications of hernia which I have been able to find and there have been no observations made on the incidence of femoral hernia in the aboriginal population.
DISEASES OF THE URINARY SYSTEM
THE KIDNEY

The only common disease of the kidney I noted in the Western Desert area of Australia was pyelonephritis. This was relatively common in females, particularly during pregnancy and coliform bacilli seem to be the most common organism, apart from one case in which Proteus was involved. I have only seen one case of renal calculi in this area and this was a staghorn calculus in association with renal tuberculosis in an elderly male aborigine. I was impressed by the fact that acuteglomerulo-nephritis was quite common in the white population in the Western Desert area, but no cases were noted in the full-blood aboriginal population. Renal calculi are also common in the white population of this area.

Kidney disease has been described by many other authors such as Smith (1878), Curr (1886) and Roelsche (1881) all who mention dropsy in the aborigine as a not uncommon occurrence, although they do not definitively state what the aetiology is. Cleland (1928) quotes Browning as stating that Bright's disease with albuminuria, retinal haemorrhage, oedema of the hands and face, with the patient dying in a coma, was common amongst the aged natives of Port McLeay in South Australia. Unfortunately there are no details by which we can verify this statement, and it is probably
significant that neither Ford (1942), Binns (1945), Schneider (1946), Cook (1949), Davidson (1957), nor Elphinstone (1960) in their fairly extensive survey of various groups of full-blood aborigines ever noted any cases of glomerulo-nephritis, of either acute or chronic variety. However, Crotty and Webb (1960) did state that two autopsies showed evidence of this disease and they also noted a further case of renal tubular necrosis. None of the above authors noted any apparent renal or ureteric calculii or renal neoplasms and apart from the staghorn calculus described above, I have not seen any evidence of these diseases in the Western Desert aborigines.

THE URETERS

I have seen no evidence of ureteric disease in the Western Desert area, except secondary inflammatory changes due to renal or bladder infections, and no evidence of ureteric colic due to uretero-lithiasis has been observed.

THE BLADDER

Acute cystitis is quite common in the female particularly in those who are elderly and those with diabetes mellitus, in the Western Desert area. As with renal infections the common organisms are B coli. Cystitis is relatively common in pregnancy in the aborigine
but does not seem to have quite the same incidence as in the surrounding white race and is rare in children and I have only seen one case in a child in the Western Desert area. I have only seen cystitis on two occasions in the male aboriginal patients. Minor degrees of cystocele are not uncommon in the Western Desert aboriginal females, but I have never seen any congenital abnormalities or tumours of the bladder the the only cases reported from other regions of Australia are by Cleland (1928) who recorded cystitis in a male and in a female aborigine.

THE URETHRA

The male urethra and prostrate will be considered with the male genital system. There are no reports of abnormalities of the female urethra in the Australian aborigines. In the Western Desert region, the only diseases I noted were urethritis associated with cystitis and urethritis associated with trichomonas infection and one case with a urethral caruncle. No neoplasms have been described.
DISEASES OF THE BLOOD

AND

RETICULOENDOTHELIAL SYSTEM
ANAEMIA

In my experience of the Western Desert aborigines anaemia is particularly uncommon except in children between the age of 2 and 4 years. In this group anemia, which is a hypochromic microcytic type, is invariably associated with chronic gastro-enteritis, often following what appears to be an upper respiratory tract infection, which has previously been described and is in turn followed by a malabsorption syndrome.

In the adults of this region I have only seen one case of hypochromic microcytic anaemia and that was in a patient with menorrhagia due to fibroids. I have not observed anaemia in pregnant women in the Western Desert region although it is common in part-caste aborigines, and aborigines living in the more urban parts of Western Australia.

In the adult males several cases of normochromic normocytic anaemia have been observed but this has always followed blood letting ceremonies or trauma and these very rapidly regenerate to normal. The low incidence of anaemia in this area amongst these aborigines has been confirmed by Pitney and Davis (1957) and Davidson (1957).
Hypochromic microcytic anaemia has however, been widely reported in other groups of Australian aborigines by Brienl and Holmes (1913), Basedow (1919, 1932), Cleland (1928), Hackett (1936), Binns (1945), Schneider (1946), Cook (1949, 1960), Cawte (1962), Collins (1968) and Jose et al (1969), all of whom have related it as being secondary to either malaria, yaws or hookworm. The incidence therefore of hypochromic microcytic anaemia is largely restricted to the coastal regions of Australia. Morbidity associated with anaemia has been emphasised by Crotty particularly in reference to children and in their series in 1960 they recorded it as the cause of death in one case, and stated that it was a concomitant factor in 69% of deaths occurring under 5 years of age (44 out of 64 deaths).

I have never seen in the Western Desert area nor have there been reports from any other area, of pernicious anaemia, congenital haemolytic anaemia, sickle-cell anaemia, thalassaemia or the microcytic anaemias associated with sprue, coeliac disease or liver disease in aborigines.

Haemolytic anaemias associated with malaria and snake bite have been described, while Petiwijeff (1963) considers that he has seen two cases of acute haemolysis of red blood cells due to ingestion of a native plant in the north-west of Western Australia.
The only cases of leukaemia described in aboriginal patients are those referred to by Crotty and Webb (1960) as being chronic lymphatic leukaemia in a Northern Territory aboriginal, and one of myelocitic leukaemia reported by Dorgan et al (1962) in Western Australia. Secondary thrombocytopenic purpura due to snake venom was noted by Feelsche (1881), but there are no reports of haemorrhagic diathesis from other regions of Australian aborigines.

THE LYMPH NODES

The lymph nodes disease has only been seen in associated with acute and chronic inflammatory disease in the Western Desert aborigines. None of the primary neoplasms of lymphatic tissue has been noted and the only degenerative change is that of one case of myeloid disease reported by Crotty (1960).

SPLEEN

The only disease of the spleen I have noted in the Western Desert aborigine is traumatic rupture of that organ. It is almost certain that that spleen was involved in tuberculosis, malaria and typhoid fever, but no specific references to this involvement have been reported. However, Crotty et al (1960) did report amyloid changes in the spleen of one of the Northern Territory
aborigines. None of the haematological disorders, lipoidosis or neoplasms of the spleen have been reported.
DISEASES OF THE MALE GENITAL SYSTEM
CONGENITAL ANOMALIES

Congenital abnormalities of the male genital system have been previously described and are apparently relatively common in other areas of Australia, although I have not noted any in the Western Desert aborigines.

THE PENIS

This is not infrequently involved in non-specific inflammation in children, balanitis is particularly common in the young males in the Western Desert region as already discussed under Cultural Characteristics and it is my belief (and the proof for this is detailed in the appendix), that this is the reason why circumcision at puberty is practised in the central areas of Australia. Circumcision itself has frequently been followed by acute infections, almost certainly due to the lack of cleanliness at the time of performing the operation and several of these patients have had to be admitted to hospital to clear up the infection. Following circumcision, meatal ulceration is not uncommon, and this I believe is the basis of the rite of subincision in later life and again evidence is presented for this in the appendix.

The specific inflammatory diseases of this organ, and granuloma inguinale, has been described as being frequent by Curr, (1887), Cleland (1928), Hackett (1936), and Basedow (1932). Although it is now apparently uncommon
as demonstrated by Elphinstone (1960) and I have seen no cases in the Western Desert area.

Gonorrhoea has apparently shown an identical epidemiology to granuloma inguinale, and as previously mentioned, syphilis is rare. I have not seen any cases in the Western Desert area and chancroid and lymphogranuloma venereum have been neither noted in the literature nor seen by myself in the Western Desert region.

Two epitheliomas of the penis have been reported by Cleland (1928) who also stated that balanitis, phimosis and smegmosis were extremely common. The incidence of balanitis, phimosis and smegmosis were also noted by Basedow (1932) and Ford (1944), but it must be remembered that they are referring to uncircumcised tribes, as they are rare in the male adult aboriginal population of the Western Desert area, but not uncommon in the younger males prior to puberty.

THE TESTES AND EPIDIDYMIS

The only description of disease of these organs is by Binns (1945) who described two cases of acute epididymal orchitis of unstated aetiology, and I have seen one epididymal orchitis secondary to a B. coli urinary tract infection in the Western Desert area. There are no
recorded episodes of testicular torsion, neoplasm or Klinefelter's syndrome in Australian aborigines. I have however, treated two hydroceles of the tunica vaginalis in adult male aborigines, both of these apparently due to trauma. Haematocele, spermatocele and varicocele have not been noted.

THE PROSTATE GLAND

In my experience, hypertrophy of the prostate gland is extremely rare in the elderly male aborigines. I have never seen urinary retention due to this cause, but Crotty and Webb (1960) state this to be the cause of death in one of their post mortem examinations. There are no reported cases of carcinoma of the prostate.
DISEASES OF THE FEMALE GENITAL SYSTEM
THE VULVA

This organ in my experience, is seldom subject to disease in the Western Desert aborigines and I have only seen one case with condylomata acuminata and three with intertrigo. Although earlier reports by Cleland (1928), Basedow (1932) and Hackett (1936) commented on the frequency of inflammatory pudendal lesions due to soft sore granuloma inguinale and yaws, and Basedow also noted the frequency of condyloma. In the Western Desert region, I have seen one case of acute Bartholin's abscess. Kraurosis vulvae, leucoplakia and carcinoma of the vulva have not been described.

THE VAGINA

Both moniliasis and trichomonal vaginitis have been presented under the section of infectious diseases. Senile vaginitis with non-specific inflammatory changes are not uncommonly seen in elderly females in the Western Desert region. There have been no reports of neoplasms of the vagina in other aboriginal groups, but I have seen secondary invasion of the vagina from primary carcinoma of the cervix in the Western Desert region, which was later followed by a recto-vaginal fistula.

THE CERVIX

There are no reports of inflammatory changes in the cervix in aborigines and although I have not seen any
evidence of acute cervicitis in the Western Desert region, chronic cervicitis is quite commonly associated with leucorrhoea and the formation of Nabothian follicles. These changes are often associated with cervical lacerations, erosions and ectropion. Congenital erosions have not been noted, but one endocervical polyp was removed from a middle aged woman of the Western Desert region. Carcinoma of the cervix has been described by Crotty (1960) in a Northern Territorial aboriginal woman and I have seen a case in the Western Desert region of stage III, according to international classification. Subsequent to this I have had reports of three other cases of carcinoma of the cervix from the Western Desert area and my experience in other areas in Australia suggests that this is one of the most common forms of carcinoma encountered in the female aborigine.

**THE UTERUS**

In my experience, this organ is less subject to disease in the aborigines than in the Caucasian races. No uterine inflammatory diseases have been described apart from those associated with recent pregnancy and neither have pyometra, endometriosis, adenomyosis, cystic hyperplasia or endometrial polyps. Fibroids have however, been noted by Cleland (1928) in an aboriginal woman and I have seen two cases in the Western Desert area. No other forms of neoplasia of the uterus have been recorded.
FALLOPIAN TUBES

I have only observed one case of acute salpingitis in an aboriginal girl in the Western Desert area. However, my experience in Perth suggests that salpingitis is relatively common in detribalised groups of aborigines and it is probable that a rise in incidence in salpingitis will be observed as the incidence of gonorrhoea continues to rise. Ectopic pregnancy is considered later.

THE OVARIES

Basedow described three women with ovarian cysts, but without any details and I have seen only one serous cystadenoma in the Western Desert. No other definite ovarian neoplasms have been described.

PREGNANCY

Abortion and miscarriage, in the Western Desert area, are rare, and on investigation there is no evidence to support the generally held concept that the frequency of miscarriage and the rate of self-induced abortion are high in the aboriginal population. This is also supported by later work I undertook in Perth regarding the obstetrical performance of detribalised aborigines (Morrison, 1969).

Ectopic gestation has not been described in the literature but one full term extra-uterine pregnancy in
a full blood aborigine of the Western Desert region was delivered by Roberts (1963). Multiple pregnancies are not uncommon as I have seen three in the Western Desert area, one of which had hydramnios associated with one of the sacs. In the Western Desert area itself, I have never seen pre-eclamptic toxaemia, eclampsia, hyperemesis gravidarum, essential hypertension, puerperal psychosis or any form of microcytic or macrocytic anaemia of pregnancy, although Crotty (1960) claims toxaemia to be the cause of death in one of his post mortem examinations and although eclampsia and pre-eclamptic toxaemia have been described in part aboriginals by Cleland (1928) and Allen (1963). Cleland also noted two cases of puerperal psychosis.

Pyelitis and cystitis in pregnancy have been previously mentioned and appear to be slightly less frequent in the aboriginal than the Caucasian population.

Ante-partum haemorrhage is infrequent. I have only seen one case of placenta praevia (grade II) and no cases of accidental haemorrhage. Breech presentations were not uncommon in my deliveries in this area, (39%), while persistent occipito-posterior positions form 16% of all deliveries I have undertaken. This is presumably due to the anthropoid shape of the aboriginal pelvis but it does not seem to give any rise to problems during
confinement. Uterine inertia is in my experience, uncommon and it may be that squatting during labour improves uterine function. Cervical dystocia has neither been noted by myself nor observed nor recorded in the literature. There are no other descriptions of labour in aborigines available except for a normal case reported by Cleland and Hackett (1937), a malpresentation reported by Cleland (1962), and delivery of a normal fetus by Poidevim (1957).

Post-partum haemorrhage was the cause of death in one of Crotty’s (1960) series, and it may also have been a factor in two other deaths reported by him in pregnant women who died after delivery, although there are no details of these maternal deaths. I have not seen any major post-partum haemorrhages in the Western Desert area and one patient with a retained placenta had very little bleeding despite a journey of 220 miles to obtain medical assistance. Premature rupture of the membranes in the aborigine is unusual, although one case was reported by Cleland (1928). Similarly premature delivery only occurred in three patients in several hundred deliveries in the Western Desert region in my series.

Trauma in childbirth to the soft tissues is not uncommon. Basedow (1932) described a third degree tear, Cleland (1928) reported a vesicovaginal fistula and I have
seen a third degree tear of two years' duration which
was repaired after delivery with good results.

Minor degree of cystocele and rectocele and
vulval varicosities are not uncommon. I have never seen
personally or noted in the literature, any cases of
procidentia, enterocele of major degrees of rectocele
or cystocele. Retroversion of the uterus is common
and first and second degree prolapse are occasionally
seen.

THE BREAST

Congenital anomalies of this organ have not been
described and I have seen no example in the Western Desert
aboriginal females. However, acute mastitis has quite
commonly occurred in my experience, having treated well
established abscesses in 6 cases, all of which required
surgical incision. Another 2 which were diagnosed early
subsided with antibiotics and conservative measures.
In the Western Desert region it is not uncommon to see
marked asymmetry of the breasts and on two occasions it
was thought that previous acute mastitis had in fact been
the cause of one breast shrinking, while continual feeding
on the other breast had resulted to a certain degree of
hypertrophy. Because of the lack of alternative diet, it
is important for the infant to maintain secretory activity
of the breast tissue. Although I have never seen any
evidence of the custom, it has been reported by Basedow (1920) and Phillips (1969) that non-puerperal lactation has occurred on several occasions amongst Australian aboriginal women.

I have only seen one carcinoma of the breast in the Western Desert region and cannot find any other reports on this disease from other groups in Australia.
DISEASES OF THE ENDOCRINE SYSTEM
Basedow (1932) refers to a case of goitre in an aboriginal woman which he considered might be a malignant thyroid, but unfortunately no details were given to substantiate the diagnosis in this person. Rankin (1902) reported a possible case of gigantism in an aboriginal female and Dowling (1937) presented a pituitary disorder (pan-hypopituitarism) in aboriginal sisters. There are no other references in the literature to diseases of the thyroid, parathyroid, pituitary, thymus or the renal or adrenal glands, apart from diabetes mellitus which is considered later under diseases of metabolism. No references are obtainable to disease of the pancreas, while disease of the ovaries and testes are considered under the relevant genital systems. No diseases of these glands, except as detailed elsewhere, have been observed in the Western Desert aborigines.
DISEASES OF METABOLISM
OBESITY

The most common metabolic disease amongst the aborigines of the Western Desert region in my experience is obesity. It is particularly common in the urban aborigines, but a number of the older aborigines in more eastern areas are also remarkably obese. These invariably are patients who have settled in the mission area and no longer join the tribe in their active nomadic movements. In both groups of aborigines, it is much more common in the female than in the male.

This metabolic disorder has also been noted by other authorities and has been mentioned by Cleland (1928) and Basedow (1932). Elphinstone (1960) studied the nutrition of the aborigines in the Kimberley region and stated "obesity is the only sign of malnutrition". In all regions, the obesity seems to be associated with a very high carbohydrate intake and is associated with the adoption of a western type of diet.

DIABETES MELLITUS

This disease I found to be surprisingly common in the Western Desert area where 6 elderly aborigines have been diagnosed and the disease confirmed in these patients by laboratory examination. All of these cases appeared to be mature onset diabetes and no juvenile or perinatal diabetes has been reported. Of these patients, 4 were
DISEASES DUE TO DEFICIENCIES
SPECIFIC VITAMIN DEFICIENCIES

I found no evidence of specific vitamin deficiencies in the Western Desert aborigines although one has the impression that sub-clinical deficiency states may well exist, particularly in the young children and the elderly aborigines.

There have however, been a number of reports of vitamin deficiencies from other areas. Thus Vernon (1928) stated that beriberi was not uncommon in the Thursday Island aborigines. Basedow (1932) quotes Wilson of Port Hedland as also observing beriberi in that area. No recent reports however, have been made of this disease.

Scurvy was stated by Basedow (1932) to be present in various localities including the Hermansberg Mission and Hackett reported a case (1936) from the Central Desert. More recently Kalokerinos (1973) has stated that he believes vitamin C deficiency to be the major aetiological factor in the upper respiratory tract infection from which aborigines suffer, leading in turn, to other malabsorption syndromes. Barton (1972) however, has stated that he doubts this and I personally have not found this to be so in the Western Desert region. No other forms of specific vitamin deficiency in the absence of other disease have been described in any other group of aborigines.
This in my opinion is common in both the children and elderly aborigines of the Western Desert region. In the children, it is associated with recurrent upper respiratory tract, respiratory tract and gastro-intestinal tract infections. These have been stated previously to cause a malabsorption syndrome in which probably all forms of vitamin deficiency are present in subclinical state, apart from protein and probably carbohydrate deficiency as well. From the evidence presented in the chapter on the physical characteristics of aborigines, I believe that on an adequate balanced western diet, the aborigines' potential for growth is almost identical to that of the white population and certainly from our growth curves it would seem that both the elderly and particularly the infants fail to achieve this growth rate. It is difficult of course to say whether the primary problem is that of malnutrition in the first instance or whether, as I believe, is more commonly due to malnutrition secondary to recurrent infection. Davidson (1952) stated that a few infants who he saw in the Western Desert area were possibly suffering from a minor form of kwashiorkor with minimal oedema of the ankles but no hepatosplenomegaly. However the more recent nutritional surveys in this region of Davidson (1957), Curnow (1957), and Mann (1957) in the Western Desert area, all fail to show any sign of under nutrition. Schneider (1946) referring to the Northern Territory and Elphinstone
(1960) referring to the Kimberleys, agreed that deficiency diseases were rare in these areas, but one has a general impression that we may be understating the general state of nutrition of the aborigine as we are tending to rely on parameters of growth which were established in the earlier days of colonisation and tend to expect all aborigines to be rather short in stature and extremely thin and wiry. The probable cause of the under-nutrition in the infant is undoubtedly, due to recurrent infection. In the elderly aborigines, the problem is probably the reliance on available mission stores as their total food intake. This consists mainly of tea, sugar and flour, and they forego their natural food supplies which, as has been described earlier, have a high protein content. I believe it probable as time passes, and more reliance is placed on western types of food, more overt disease due to under-nutrition will become apparent in this race, until they are sufficiently educated to realise the nutritional value of various foodstuffs.
There are no reported occurrences of hay-fever, drug allergy or serum sickness among the full blood aborigines in any region and I have observed none in the Western Desert area. Asthma has previously been discussed and as noted, all of these cases were related to infection and none due to a true allergy have been seen.

Rheumatic fever, rheumatoid arthritis and glomerulonephritis have been detailed under the relevant sections and there are no accounts, and I have never seen any examples of disseminated lupus erythematosus, dermatomyositis, polyarteritis nodosa or scleroderma in a full blood aborigine.
DISEASES DUE TO PHYSICAL AND CHEMICAL AGENTS
SUNSTROKE AND HEAT EXHAUSTION

Both these conditions were stated to be common causes of death amongst the aborigines by Curr (1886), Taplin (1879) and Cleland (1928). There are however, no recent references to these effects and I have seen no cases amongst the Western Desert natives. I believe that most of the previous references probably related to dehydration due to some other intercurrent disease, most probably enteritis or possibly pneumonia or bronchopneumonia. No authorities have made any mention of the effects of either motion or atmospheric pressure in aborigines, but in my experience, travel sickness is remarkably common, while the reduced pressure effects in air transport frequently produce sinus headaches and otalgia in aborigines, often of a severe degree which are probably due to Eustachian tube congestion and blockage related to upper respiratory tract infection and chronic sinusitis.

ALCOHOLISM

According to Curr (1886), Smith (1887), Foelsche (1881), Cleland (1928) and Basedow (1932), alcoholism was apparently rife amongst the aborigines until the legislation of 1905 and 1911 forbade the supply of alcohol to aborigines. Since that date there have been very few reports of alcoholism in aborigines apart from the popular press.
Drunkenness in the Western Desert aborigines is restricted to the various town areas. These are the only places where alcohol can be supplied. All the missions forbid the importation of alcohol and are consequently "dry." Alcohol is now of course, freely available to aborigines from the normal retail outlets and in my experience, the general incidence of drunkenness is related to both the responsibility of the publican supplying the liquor and the local police officials. I have not noted any marked difference between the effects of alcohol in the white and the aboriginal population, if one takes into consideration the type of drink which is imbibed and the speed and quantity with which it is drunk. In my experience, neither alcoholism nor drunkenness is more prevalent in the aboriginal population than the white population when one allows for the relative social problems facing both groups of the population, and social class.

OPiUM ADDICTION

This is no longer a problem in aborigines following similar legislation to that originally applied to alcohol, but was common in the past as noted by Holmes (1912) and Cleland (1928). This addiction rose in various groups of aborigines in other parts of the continent which had mingled with a fairly high Chinese population. No cases of opium addiction have been reported in the Australian aborigines in the past 40 years.
I have never seen any venomous snake bites in the Western Desert region in an aborigine although I have seen 2 in the white population. However, in this area, venomous snakes are relatively rare.

Snake bite poisoning was however, stated by Curr (1886) to be one of the common causes of mortality in aborigines before the advent of white settlers, a statement which I find difficult to believe and is in direct opposition to that of Foelsche (1881) who stated that the aborigines knew how to treat snake bite and moreover stated "I have never heard of a native dying of snakebite".

Reference has already been made to the possibility of 2 aborigines in the Port Hedland area dying of acute haemolysis due to ingestion of an unknown chemical in a native plant described by Fetwijeff (1963).
DISEASES OF THE SKIN
ACNE

I observed this disease frequently in the Western Desert aborigines particularly amongst the younger males and females, and miliarias is not uncommon in the latter. Acne has also been reported to be prevalent amongst the Thursday Island and South Australian aborigines by Basedow (1920 and 1932), and Cleland (1928) respectively.

DERMATITIS AND ECZEMA

In the Laverton and Warburton area of the Western Desert region I saw 3 children who suffered from a form of contact dermatitis in which the primary irritants were thought to be a combination of sweat and dust, but no true allergic sensitizing agents were discovered despite extensive testing at Princess Margaret Hospital in Perth. These 3 children all improved when transferred from this region to Perth, but all relapsed on coming back to this area. The major problems with these children were the occurrence of secondary infections. I have not seen any true eczema in a full blood aboriginal in this region, although Stirling (1886) stated that eczema was common and Gorrey, as quoted by Cleland (1928) and Basedow (1932), also report one case each.

PSORIASIS

This was stated by Smith (1878) and Stirling (1896) to be common. The latter stating that he had seen it in
the aborigines of Central Australia. I have never seen psoriasis in a full blood aborigine and I can find no recent reference to this disease. None of my personal correspondents have seen this disease either.

**IMPETIGO**

As previously described in "Infectious Diseases" this is not uncommon in the Western Desert aborigines and it presents no difference between the white and aboriginal races in either the clinical course or the response to treatment.

It is very difficult to assess the true incidence of impetigo prior to 1914, although Collins (1798), Smith (1878), Taplin (1879) and Curr (1886) all comment on the frequency of the disease, but there is no doubt that considerable confusion existed at that time in a differentiation of impetigo, native pox, smallpox and chicken pox. Hayward (1884) stated that "native pox" was impetigo contagiosa, while Thomas (1884) could see no difference between the native pox and the various impetiginous diseases as occurred in Britain. Verco (1884) said that he could not recognise native pox from impetigo or varicella. The confusion continued until Cumpston (1914) proved that smallpox and native pox were identical, and that impetigo in the aboriginal was identical with impetigo in the white race. However, impetigo probably was
prevalent as both Basedow (1919 and 1932), and Cleland (1928) remarked on its frequency quoting many contemporary sources for this statement, but since these reports there have been no comments from literature regarding the present incidence.

**WARTS**

The occurrence of warts (verruca vulgaris) has been described by Basedow (1932) who stated that they were common in the aborigine, although Gray and Cleland (1933 and 1934) noted only three cases, while Black and Cleland (1938) in another examination of aborigines of Central Australia did not observe any. Basedow also stated that the central tribes treated warts by pricking them with acacia leaves (acacia verticulita).

In my experience verruca vulgaris is not uncommon in the Western Desert region, but no cases of verruca plantaris were noted and none have been reported in the literature.

**SEBACEOUS CYSTS**

These were quite common in the Western Desert area in my experience and they have also been noted by Basedow (1932) and Gray and Cleland (1933) who also commented on their frequency.
OTHER SKIN DISEASES

Ptyriasis is stated by Stirling (1911) to be common in the Central Australian aborigines and Junk, quoted by Cleland (1928), considered lichen planus to be "not uncommon", and Gorrie (1928) observed a case of pemphigus in a four month old child who died. I have never seen any evidence of any of these diseases in the Western Desert aborigines.

Infantile eczema, rosacea, chronic discoid lupus erythematosus, dermatitis herpetiformis, urticaria, drug eruption, erythema multiforme, atopy and neurodermatitis, have never been reported in Australian aboriginals and I have seen no examples of these diseases in the Western Desert area.

SUBCUTANEOUS TISSUES

Apart from infections which have been previously described, lipoma, in my experience, is the most common condition seen. The majority of the cases I saw in the Western Desert varied between small and moderate size, though my predecessor removed one weighing 12 lbs. from the right scapula area of a male adult aborigine. Lipomas are apparently common in other groups of Australian aborigines, as several have been reported by Cleland (1928), Basedow (1932), and Gray and Cleland (1933).
MALIGNANT DISEASE OF THE SKIN

Cleland (1928) described three cases of squamous cell carcinoma (one of the cheek, two of the penis) and one basal cell carcinoma, while Stirling (1896) in the Horn expedition also observed what he considered to be a squamous cell carcinoma of the foot. Black and Cleland (1938) reported malignant melanoma in an aboriginal woman.

Neither hyperkeratosis nor any other premalignant or malignant tumours have been reported in the literature and I have seen no evidence of malignant neoplastic disease in the skin or subcutaneous tissues in the aborigines of the Western Desert region.
DISEASES OF THE LOCOMOTIVE SYSTEM
MUSCLES AND TENDONS

Both trauma and infection of these organs have already been considered, although in two aborigines in the Western Desert following spear wounds, I have seen herniation of the vastus lateralis muscle through the sheath although this was symptomless. None of the muscular dystrophies, myasthenia gravis or any neoplasms or striated muscle have been described and I have seen no examples of these conditions in the Western Desert region.

Numerous accounts of "rheumatic pains", "muscular pains", "fibrositis" have been recorded by Smith (1878), Curr (1886), Cleland (1928), Basedow (1932), Hackett (1936) and Ford (1942), but it is impossible to diagnose the exact condition causing these pains, apart from Hackett's description of "Irkunji" which is due to yaws. In my experience, it is not uncommon to find localised tender areas in various muscles in the Western Desert area of uncertain aetiology, though the majority are apparently due to exertion or muscle trauma with resulting muscular spasm.

Tenosynovitis is not uncommon in the Western Desert area and I have seen several cases usually of the extensors of the forearm, particularly the extensor pollicis longus.
Ganglions have been reported by Cleland (1928), Gray and Cleland (1933) and Basedow (1932). The latter noting many examples, but I have seen no evidence of this condition in the Western Desert.

THE BONES

The only common disorders of the bones are those which have been previously described, namely trauma and yaws.

None of the congenital disorders of bone formation, von Recklinghausen's disease, hypertrophic osteotropathy, fibrous dysplasia or any neoplasms of bone, either benign or malignant, have been described in the aborigine and I have seen no examples in the Western Desert area. Barry (1968) described a case of Paget's disease which apparently occurred in a full blood aborigine. There are no other extant examples of this disease. Although osteoporosis has not been described in the Australian aborigine, I found it to be not uncommon in the elderly age groups in the Western Desert area, more so in the females than the male. In my opinion, it is probably nutritional in origin, although it is possibly due to other metabolic changes. There have been no reports of osteomalacia or of bony changes due to rickets although it is possible that Basedow's case (1919) which he considered to be infantile paralysis could possibly have been this disease or scurvy.
The joints

These areas are frequently affected by trauma and in the elderly aborigines in the Western Desert region, there are many examples of osteoarthritis which may result from previous trauma or as degenerative changes with age. In the younger aborigines one can frequently trace osteoarthritis to direct injury, but this condition has not been reported elsewhere.

Suppurative arthritis which was probably gonococcal in origin, has been described by Breinl and Holmes (1915), Cleland (1918), Basedow (1932 and Hackett (1936) but there are no recent references to this condition and it seems now to be apparently rare, I have certainly seen no examples in the Western Desert region. Gray and Cleland (1933) noted a case of ankylosis of the knee joint which they thought was due to previous acute arthritis although they did not state what they thought the aetiology was. Mackay (1938) has described various bone and joint disorders in aboriginal skeletons.

Rheumatoid arthritis has never been noted in an aborigine, except in the extremely doubtful reference by Cleland (1928) who diagnosed the disease on the partially complete skeleton of an elderly female aboriginal at Innaminka. It is almost certain from his description that the actual disease was osteoarthritis and I have
not seen any evidence of this disease in the Western Desert aborigines. Similarly none of the syndromes of diseases associated with rheumatoid arthritis have ever been reported in a full blood aborigine. Arthritis due to gout or rheumatic fever have previously been considered.

Bursitis is common and I have seen several examples - four of the olecranon, two subdeltoid, one prepatellar, and one infrapatellar in the Western Desert aborigines. All were of the acute simple type and none were infected. Basedow (1932) and Gray and Cleland (1937) have also reported many examples. Most of these being either of the elbows or of the deltoid region.
DISEASES OF THE NERVOUS SYSTEM
Organic disease of the central nervous system due to infection by specific organisms has already been described, and is apparently the same clinically in both the white and aboriginal races. In addition, Crotty (1960) noted a cerebral abscess in an aboriginal, but has given no details of the cause.

Cerebro-vascular accidents have been described by Smith (1878), Cleland (1928) and Crotty (1960) referring to both cerebral haemorrhage and thrombosis, and I have seen several cases of both in the Western Desert area, the former having been associated with hypertension, and all occurring in elderly aboriginals. Subarachnoid haemorrhage and cerebral embolism have not been described. "An acute chorea" was described by Smith (1878), and Cleland (1928) also noted an example, but in each instance no details have been given to the clinical cause. Binns (1945) stated that he had observed a case of Sydenham's chorea and also an example of true catatonia.

Epilepsy was noted by Crotty (1960), and I have seen two examples of grand mal in the Western Desert area. In neither case could an organic cause be found, but it is possible that it was due to previous trauma.
No congenital malformations of the brain, spinal cord or their coverings or the associated blood vessels have been recorded and there is no evidence that any of the hereditary familial neuropathies, metabolic disorders, or the demyelinative diseases exist in the aboriginal population. Saint (1962), in a state-wide examination of multiple sclerosis, stated that the disease had never been seen in a full blood Western Australian aboriginal and this is certainly true of the Western Desert area.

Although cerebral degeneration associated with age and accompanied by senility is not uncommon, the specific degeneration such as Wilson's disease, Huntington's chorea, Herxheimer's disease, lobar sclerosis, Friedreich's ataxia, syringomyelia and tubo-sclerosis have never been described in an aboriginal and I have seen no evidence of any of these diseases in the Western Desert area. The only reference to Parkinson's disease is that made by Newland (1895) in which he refers to an aborigine of the Encounter Bay district named Shaky Jack whom he describes as being afflicted by a species of palsy which Cleland (1928) thought might be this disease. However the original description is too vague to put any definitive diagnosis to it.

There have been no references to neoplasms of the central nervous system in Australian aborigines, although
I have seen two gliomas in a brother and sister, but who were part-aboriginal.

Organic disease of the peripheral nervous system is most commonly due to trauma or infection; the former predominates in the Western Desert region, although in the coastal region leprosy must be considered as a significant factor.

FUNCTIONAL DISORDERS

These in my experience, are extremely rare amongst the aboriginals, and I have only seen one example of a post-puerperal depression and this was not in the Western Desert region, and one elderly native man who became maniacal when he was incarcerated in a cell after being transferred to Kalgoorlie jail. When returned home, he rapidly reverted to his normal state.

Minor behaviour disorders are not uncommon in children who have been separated from a parent either because of school commitments or hospital treatment, but again these usually subside rapidly after they return home. Lickiss (1970 and 1971) has commented on the problems which beset aboriginal children in trying to cope with a large city environment and points out the relatively common social deviations that tend to occur in this group. Amongst the adult aborigines Cawte (1964) has extensively
studied the ethnopsychiatry of the Kimberley aborigines and has evidently found most of the psychological and psychotic disorders to be present in these natives relatively frequently. No confirmation or disproof of this work in this region is available, but the extreme difficulties involved in accurately interviewing such a group would render any conclusions suspect in my opinion.

From my own experience in the Western Desert region I can quote two cases in which psychiatric diagnoses were wrongly made. The first patient was a 45 year old adult aboriginal man whom I saw frequently because of apparent abdominal pain. Despite thorough examination, I could find no abnormality and the only significant feature in his history was the fact that he wished to get an invalid pension. Because of his frequent appearance and the fear that I was missing some serious disease, I referred him to Royal Perth hospital for further investigation by the department of surgery. They too could find no organic disease and referred him to the psychiatrist for assessment. The report I got from the psychiatrist stated that this man was uncommunicative, appeared to be withdrawn and could neither read nor write, and they considered him to be of subnormal mentality and considered that he should probably be certified to institutional care for his own protection, but as the letter stated, because they were not "absolutely sure of the norm for this ethnic
group, they would let him return to the Western Desert."

I must say that I found this statement quite incredible and an excellent example of the lack of insight that many practitioners have into problems associated with the aborigines because this man was in fact, extremely intelligent, his main desire being to get an invalid pension and he was certainly clever enough to mimic symptoms of possible organic disease. It was not surprising that he could neither read nor write having been brought up in a tribal manner in culture which has no written language, some 400 miles from the nearest school and again it was not surprising he was withdrawn and uncommunicative when his initial history taking had been rapidly followed by a barium meal, barium enema, cholangiogram, sigmoidoscopy etc. The fact that he was nearly incarcerated in an institution filled me with horror.

The second case was reported to me by Dr. Ida Mann (1960) and also concerned an aborigine from the Western Desert region. This was a young male adult who was serving a jail sentence in Fremantle as a result of spearing another aborigine in whom he severed the femoral artery, subsequent to which the opponent died. This man was not in fact arrested for over a year since he went bush north of the Warburton ranges and could not be found, but was eventually arrested in South Australia. It was by
chance that Dr. Ida Mann was visiting the prison hospital seeing another aboriginal patient when the prison psychiatrist stated that he had just seen an interesting aboriginal patient whom he was going to certify because of apparent repeated attempts at suicide. Dr. Mann said that she would like to see the patient and asked how the diagnosis of repeated attempts at suicide had been reached. The psychiatrist stated that the aborigine had numerous scars on his arms in which he had obviously attempted to slash his vein. Dr. Mann examined the patient and informed the psychiatrist that these were the normal scars of tribal venesection and had absolutely no relation to attempted suicide. I am glad to say that patient has now been released having completed the jail sentence and is now back in the Western Desert area enjoying a normal life.

It is these personal experiences of psychiatric diagnosis coupled with my knowledge of the lack of psychiatric conditions in the aborigines of the Western Desert which make me chary of reported frequencies of psychological upset in aboriginal patients in their normal environment. I do not doubt however, that there will be an increase in both psychological and psychotic disorders amongst the full blood aborigine as they become more integrated into western society and become more exposed to the stresses and strains of western civilization.
DISEASES OF THE EYE.
The Conjunctiva

Conjunctivitis is the most common form of eye disease amongst the aborigines of the Western Desert area, and by far the most prevalent type of conjunctivitis is due to trachoma. Although approximately 70% of the Western Desert aborigines have active trachoma, few show any signs of complications such as scarring, although the frequency of complications increases with both age and geographic location, those in the western part of the Western Desert are having a higher rate than the more eastern sectors. The apparent low rate of complications in the younger age groups is probably due to the blanket treatment by Lederkyn which is given every six months to all aborigines in this area. This is sufficient to keep the disease in abeyance although reinfection will occur but not to the degree whereby complications are superimposed, and Mann (1957) considered that the degree of complications was related to the frequency of secondary infections. Surveys of trachoma in the Western Desert area were conducted by Mann in 1954, 1957 and 1961 and during this time, the overall rate of infection has shown little decline due to the apparent rapid reinfection of most of the patients. In 1961 in her surveys to Cosmo Newberry Mission and
Warburton Ranges Mission in which I accompanied her, the infection rate was 90% and 70% respectively to the areas.

There is some evidence that trachoma was probably endemic in the Australian aboriginal before white settlement, as Dampier (1688) describes a disease in the aborigine that certainly fits this pathology. It was also described as being widespread by Stephens (1889). The prevalence of the disease was also attested to by Wade (1916), Basedow (1919), Cleland (1928), Basedow (1932) and Black and Cleland (1930). More recently Schneider (1946) stated that 90% of the aborigines in the Northern Territory were affected, those in the dryer area showing a greater number of complications, and he considered this to be due to constant irritation by dust.

Both catarrhal and membranous conjunctivitis are also frequently seen in the Western Desert area, especially in the children and this has been substantiated by Mann (1957) and they appeared to be much more common in the town than the desert aborigines. Schneider (1946) also mentioned the frequency of catarrhal conjunctivitis in association with upper
respiratory tract infection in the Northern Territory.

Gonorrhoeal conjunctivitis has been previously discussed.

THE CORNEA

Trauma to the cornea is common, and scarring following corneal abrasion, laceration and penetrating injuries are frequent in the Western Desert area. Corneal opacity following trachoma and its complications are unusual in the Western Desert area but are seen in some of the more elderly aborigines, although it has apparently been not uncommon in other areas as noted by Cleland (1928), Basedow (1919 and 1932), Black and Cleland (1938) and Schneider (1946). More recently however, Mann (1957) has disagreed with this.

THE SCLERA, IRIS AND CILIARY BODY

These structures are seldom affected except by trauma and infection following penetrating injuries. Scleritis, episcleritis, sclerokalacia perforans and blue sclerotics have not been described and none of the allergic forms of uveitis have been recorded. No tumours of this region have been described and I have seen no example of these diseases in the Western
Desert area.

THE LENS, VITREOUS, CHOROID AND RETINA.

Traumatic and senile cataract are not uncommon in my experience and these have also been noted by Schneider (1946) and Mann (1954 and 1957). I have seen several cases of mild hypertensive retinopathy in aborigines, with slight nipping of the A-V junctions, but I have never seen malignant retinopathic changes.

THE LIDS

Blepharitis is common in the Western Desert region and it has also been described by Basedow (1919 and 1932), Schneider (1946) and Mann (1957).

I have seen several cases of ectropion in the elderly aborigines in the Western Desert area and two cases of entropion associated with trachomatous cicatrication. Basedow (1919) stated that he considered the former of these two diseases to be a sequel of gonorrhoeal conjunctivitis.

Although Basedow (1932) considered hordeolum to be uncommon, I have seen several cases and Mann (1957) also reported several in the Western Desert
area. Chalazion is stated by Schneider (1946) to be common in Northern Territory but I have only seen three examples in the Western Desert area.

**LACRIMAL APPARATUS**

Schneider (1946) stated that chronic lacrimal cystitis was not uncommon in the Northern Territory in association with old trachoma, but I could not confirm this is the Western Desert area. I did however, note in this region that chronic lacrimal cystitis is not uncommonly seen in infants in association with upper respiratory tract infections.

**NEOPLASMS OF THE EYE AND ORBIT.**

No neoplasms of this region have been reported in the literature and I have seen no examples in the Western Desert area.
DISEASES OF THE EAR.
THE AURICLE.

I have noted in the Western Desert area that trauma is relatively common, particularly in women in domestic arguments, the lesions ranging from abrasions to lacerations and haematoma auris. Human bites are not uncommon, and are occasionally complicated by perichondritis. No congenital deformities have been described.

THE EXTERNAL MEATUS.

Furunculosis is in my experience, uncommon, and otitis externa is invariably secondary to a chronic otitis media. Dermatitis, seborrhoeic eczema and otomycosis have not been seen by me or recorded in the literature. Impacted wax is extremely rare as I have not seen a case in a full blood aborigine. Foreign bodies are also remarkably infrequent, those few seen in the aborigines having been insects in children's ears. None have been observed in adults, and this is probably due to the profuse growth of hair that normally protects the external meatus.

THE TYPANIC MEMBRANE.

Traumatic rupture is rare and I have only seen one case associated with a fractured skull in an adult
male aborigine and also one case of acute otitic barotrauma. Perforation of the ear drum however, in association with acute and chronic otitis media is extremely common and this is discussed below.

THE MIDDLE EAR.

The frequency with which infection of the middle ear occurs in the aborigines has been noted by Curr (1886), Foelsche (1881) and later emphasised by Cleland (1928) and Basedow (1932). Binns (1945) noted that in his series in the Northern Territory he had 12 cases of acute otitis media associated with tympanic perforation. Davidson (1957) and Elphinstone (1960) also commented on the frequency of the condition in the Western Desert and Kimberley natives respectively.

I have found both acute non-suppurative and acute suppurative otitis media to be extremely common in the Western Desert area, few children not being affected at some time, while many have recurrent otitis media which is frequently associated with the exanthemata (particularly measles), respiratory tract infections, and, in the younger age groups, gastro-enteritis. After the age of 10, the frequency of otitis media rapidly diminishes and it is uncommon in adults. As
mentioned previously, particularly noticeable was the incidence of acute otitis media during the measles epidemic at Warburton Ranges Mission in 1963, when approximately 90% of the children developed this complication, many of these subsequently remaining resistant to treatment and developing a chronic suppurative state. Many of these children had also a chronic suppurative otitis media prior to the measles infection. Tooth and Lewis (1963) have given some details of the acute stage of the epidemic, but unfortunately gave little indication of the long term morbidity that resulted from it. The chronic state of suppurative otitis media is usually associated with enlarged pharyngeal lymphatic tissue and other infective states. In the end spontaneous resolution normally occurs during the teenage, and the disease is seldom seen in the adult. Despite the prevalence of both acute and chronic suppurative otitis media, complications are few. No cases of mastoiditis, cerebral abscess, petroidosis, sigmoid sinus thrombosis or pharyngeal abscess have been reported in the literature or seen by me in the Western Desert area. Granulations are quite common, while polypi are much more rare, while choleseomata have not been personally encountered. On several occasions during acute infections, I have
noted some labyrinthine upset in the older children, but this generally settles rapidly, while in infants transient weakness of the facial nerve has also been seen.

Otosclerosis has never been described in an aboriginal patient and I have seen no examples of this disease. Recently both Pittock (1970) and Lickiss (1970) have both commented on the prevalence of chronic otitis media in aboriginal children in New South Wales and Clements (1953, 1961) has commented on the frequency of diminished hearing in aboriginal children due to chronic ear infections.

THE INNER EAR.

Labyrinthitis, Ménière's disease, specific infections or tumours of the inner ear have neither been observed nor reported.
DISCUSSION OF THE PATHOLOGY

OF THE

WESTERN DESERT ABORIGINES.
The most significant feature in the preceding description of the pathology of the Western Desert and other aborigines is the paucity of reliable information, despite the relatively large full-blood Australian aboriginal population (estimated at 45,000) and the duration of colonization.

The factors leading to this are complex. Many of the early reports of diseases in aboriginal tribes were made by non-medically qualified persons, usually missionaries, although some, such as Taplin (1879) and Curr (1886) had some medical knowledge. The majority of the earlier statements must not therefore be given unqualified acceptance. In the early days of settlement most of the aborigines existed in areas outside the confines of medical attention, and most practitioners who treated any aborigines did so in extremely small numbers. Even today, despite the extension of medical services and the more ready access of the great majority of aborigines, their actual medical care is conducted by only a fraction of the medical practitioners in practice in Australia, with the result that only a few people have the necessary experience of sufficiently large numbers of aborigines to comment about the diseases to which they are subject.

The major problem, however, is the lack of
available records for comparison between populations. Although certificates of the cause of death, still-births and the notification of communicable disease apply to the aboriginal population, as well as the white, there is no method by which the two races are separated in the statistics of the Registrar-General's office. Thus comparisons of mortality and morbidity cannot be made, and this defect will continue unless Government legislation is altered, a recommendation which is supported by Moodie (1969), Tatz (1970) and Matlak (1970).

Despite these limitations, it is possible to discern a pattern of disease in both the Western Desert and other aboriginal groups, and it is apparent that differences exist not only between various aboriginal groups, but also between the aborigines and the white population of Australia, and it is from these two major aspects that the pathology of the Western Desert aborigines will be discussed.
THE PATHOLOGY OF THE WESTERN DESERT ABORIGINES

IN COMPARISON TO

OTHER AUSTRALIAN ABORIGINAL GROUPS
It is evident that prior to colonization the aborigines did not live in a disease-free environment, but it is probable that the spectrum of disease to which they were subject was far less extensive than at present.

Although it is impossible to be sure which diseases were endemic in the Australian continent, due to a lack of adequate documentation, it is likely that the major causes of morbidity and mortality were traumatic, infectious diseases, and deficiency and neoplastic states.

It is probable that both the incidence and types of trauma were similar to that seen in nomadic aborigines today, of which the only remaining major groups are the Western Desert and Central Desert aborigines, as most forms of trauma are closely related to their cultural habits and it is unlikely that they varied greatly on a geographical basis.

The deficiency states would, however, be closely related to the availability of an adequate and varied diet, and this must have been largely dependent on climatic and environmental conditions. It is most likely that the Western Desert as well as the other Central Australian aborigines were at much greater risk than other aboriginal tribes from both acute and chronic deficiency states, and that both general malnutrition as well as specific
vitamin deficiencies (of which Vitamin C lack would probably be the most common) were not infrequent causes of morbidity and mortality in the central areas.

There is remarkably little information on which one can base the probable pattern of infectious disease prior to colonization, but it is likely that both trachoma and yaws were endemic. The evidence for the former is tenuous but it is certain that not only was the latter present but that all groups of aborigines had developed relatively high degrees of immunity to it. It is apparent that some variation in the type of lesions due to yaws was present, probably due to climatic conditions, the skin manifestations being more common in the wetter areas, and the osseous lesions more common in the drier areas especially in the Western Desert and Central Desert areas.

Which other infectious diseases were endemic is impossible to state with accuracy but it is likely that malaria was endemic in the sub-tropical regions, and there is evidence that epidemics of smallpox did occur, but whether this disease was endemic or introduced by casual immigrants such as Malay, Indonesian and New Guinea fishermen who were cast ashore is impossible to say. It is likely that other diseases were periodically...
introduced from these sources, but the effects would be most evident in the aborigines at the point of contact, the more isolated and nomadic Western and Central Desert aborigines being less likely to be infected.

Malignant neoplasms are a not uncommon cause of death in the Western Desert and other aboriginal groups today but to what extent the type and frequency has been influenced by Westernisation and a possibly changing age pattern is difficult to evaluate.

The effects of colonization on the epidemiology of diseases in Australian aborigines was much more complex than the mere introduction of a variety of new diseases to a non-immune population. The early immigration of Asiatics and Polynesians to Australia introduced a vast variety of tropical diseases in addition to the more common European types, but fortunately the former tended to be limited climatically within the continent and restricted to the aborigines of the Northern and more humid regions. However, the mortality, morbidity and frequency of these diseases were exacerbated by the marked changes which occurred in the aboriginal environment. These changes were the compound effects of a decrease in the nomadic habits of the aborigines and the establishment of more permanent camps,
generally in relation to the newly developing towns; the breakdown in hygiene which this entailed; the change in diet from a largely meat to a sub-standard carbohydrate diet; overcrowding in unsanitary conditions; the exposure to various addictions and venereal disease; the acquisition of garments, which the aborigines had no experience of maintaining in a hygienic condition; contamination of water supplies; and what is still one of the major public health problems in Australia today, the rapid increase in the fly population due to the establishment of the cattle industry. In addition the introduction of new animal species, many of which became wild with their accompanying parasitic infestations, of which the pig with taenia, and the dog with echinococcus are the major examples. These particularly affected the aboriginal population due to their normal custom of undercooking food.

The effects were most prominent in the coastal regions where most of the early immigrants settled and the Western Desert aborigines were relatively unaffected apart from the infectious diseases which occurred in epidemic forms in this region by virtue of social contact with other aboriginal groups, as the earliest contact with white explorers only occurred in the late 19th century and no settlement was achieved until the gold
field discoveries in 1893.

The Western Desert aborigines were further protected from the effects of these diseases by the climatic conditions which have prevented the spread of tropical diseases to this region, and their still largely nomadic way of life and dry atmosphere which have, in my opinion, formed the major factors in preventing the establishment of the parasitic infestations.

It would appear that the only disease introduced to these aborigines by the colonists, to which they had a specific immunity was syphilis, and this was due to yaws being endemic. The other infectious diseases had the same effect on the aborigines as has been seen with other non-immune racial groups such as the North and South American Indians, the Polynesians and the Eskimos. In a similar manner the aborigines have developed some immunity to these diseases and the epidemiology of these diseases has shown variations, especially during this century.

The major changes have been those which have followed 'pari-passu' with the control of specific diseases in the white community of which tuberculosis, typhoid fever and paratyphoid are the most important. Relative
immunity to tuberculosis has also been attained, and the more urbanized aborigines are frequently exposed to the more common exanthemata and have also developed an immunity similar to the white population.

There has, however, been a notable, and in my opinion, purely fortuitous decrease in such diseases as granuloma inguinale gonorrhoea and yaws. The reason is not due to specific treatment of these diseases but the incidental curing of them by antibiotics given for other infections, mainly upper respiratory tract infections and gastro-enteric conditions.

The effect of these modifications on disease patterns has affected the Western Desert as well as other aboriginal groups, and today the differences which exist in epidemiology between the various aboriginal groups are due to environmental conditions rather than specific racial characteristics.

There is, however, a changing pattern of disease associated with assimilation. The most apparent of these in the Western Desert region is the occurrence of metabolic disorders in the more urbanised groups, of which mature onset diabetes mellitus, cholelithiasis, and obesity are the most prominent. Assimilation does
produce many more complex effects and these are best seen in relation to pregnancy. The metabolic effects appear to increase the fetal size, and obstructed labour, which is very rare in the nomadic aborigine, is not uncommon in the urbanized person. The incidence of obstructed labour is not only due to the larger fetal size but also to a relative decrease in pelvic dimensions which is probably related to malnutrition in the female child. A detailed account of the effect of assimilation as shown by the obstetrical performance of the urbanized aborigine has been previously published - Morrison (1968) and is contained in the Appendix of this thesis.

In general, variations in disease patterns between the Western Desert and other aboriginal groups are related to variations in climatic and other environmental conditions, and not to any specific racial factor. The major factors which support the high incidence of disease in the aboriginal population today are the poor living conditions, poor diet and problems of hygiene, rather than any specific racial lack of immunity, and many of these factors are increased with partial assimilation.
THE PATHOLOGY OF THE WESTERN DESERT ABORIGINES

IN COMPARISON WITH

THE WHITE AUSTRALIAN POPULATION
The differences in epidemiology which occur in these two groups may be considered under the following sub-sections:

1. **Diseases with a higher incidence in the aboriginal population.**

   The infectious diseases are, as a group, much more common in the aborigines than in the white population, and the major cause has been ascribed by Cook (1949) to the environmental conditions of the aborigine.

   The close and crowded living conditions, which are frequently both cold and damp are, in association with detribalization and the concommitant insanitary surroundings (due to the lack of appreciation of the necessity of careful disposal of the extreeta), and also the poor diet, are adequate reason to account for the increased frequency of this group of diseases. The other cause of the increased frequency has been considered by Cleland (1928), Basedow (1932), and Cook (1949), to be a lack of immunity in a previously unexposed race, which has not yet reached equilibrium with the changed environment. However, these two factors are, in my opinion, not sufficient to account for all the differences in the pattern of infection between the aboriginal and white populations. Close scrutiny of the infectious diseases disclose three sub-groups with marked variations in frequency.
(a) The parasitic infestations and gastro-enteritis.

The higher frequency of amoebiasis, hookworm, hydatid disease, bacillary and bacterial infections, (which are the only common diseases of the group in the Western Desert aboriginal) and other intestinal parasites are much more frequent in the aboriginal than the white population within the climatic region of localization. The increased incidence of this group of infections can be explained on the basis of a lack of hygiene, and there is no evidence that either a lack of immunity or genetic differences are involved.

(b) Viral infections causing common colds and influenza.

Both of these diseases occur in the aboriginal population much more frequently than in the white population of the same area, and they vary in several aspects. Firstly, the number of complications are much greater, particularly those of the respiratory system, although in the uncomplicated cases the duration of the disease is much the same as in the white population. The major difference is in their recurrence rate, a feature that I have particularly noticed in the Western Desert region. Attacks succeed each other in many cases with such rapidity that any antibody production must be of a fleeting nature, and therefore in this group, superficially
there is apparently some deficiency in the protective mechanisms.

The exemthemata are not more frequent in the aborigines than in the white population in my experience, but are followed by a greater number of complications due to the chronic respiratory disease from which most of the aborigines suffer. One particular aspect of the epidemic of measles in the Warburton Ranges in 1961 was that it was continuous with an epidemic of influenza in this region, which was still present, although starting to decrease in incidence. The very close juxtaposition of these two viral epidemics, both of which affected almost the entire population, leads one to wonder whether the aborigines may have a defective interferon mechanism, and this could well account for the rapid recurrence rates of coryza and influenza, although specific immunity to the exemthemata are clinically the same as in the white population.

(c) A group of infections characterised by tuberculosis, poliomyelitis, and infective hepatitis, which one would expect to be more common in the aborigine than in the white population on account of their poor environmental conditions. These three diseases occur, in fact, much less frequently than one would expect. All three are endemic in the white Australian population, although they
have decreased recently, and the latter two have on occasions reached epidemic proportions, although poliomyelitis has greatly decreased during the past ten years due to extensive vaccination in the white, but not the aboriginal, population.

Despite the close contact between the white and aboriginal populations, these diseases appear to be much less frequent in the latter, and the low frequency must be due to some form of immunity. The studies of King et al. (1951) show that the aborigine has reached a state of true immunity towards tuberculosis, and is now in satisfactory equilibrium with the environment, and through the studies of Cleland (1927), Curr (1886) and Taplin (1878) one can deduce that this has occurred during the last 50 years. It is probable that the same pattern has occurred in relation to poliomyelitis and infective hepatitis. The present low incidence of these diseases is almost certainly due to the babies having acquired immunity from the mothers, which protects them during their early life, while sub-clinical recurrent infections helps to preserve their immunity. This means, therefore, that these diseases are endemic within the aboriginal population and does as such represent a major health hazard to the white population in contact with them, and that the true incidence is higher but of a largely sub-clinical state.
2. Diseases with a similar incidence in the aboriginal and white population.

This group includes the congenital anomalies and the examathemata. The congenital anomalies have been fairly well recorded and give an impression that they are more common in the aborigine than in the white population, but if one allows for the fact that physical abnormalities are more easily recognized and therefore more commonly commented on even by lay authorities, then it is probable that the relatively high incidence in the aboriginal population is exaggerated. It is my experience that they are no more common, and, it is noteworthy, that, apart from diabetes mellitus, none of the metabolic or associated diseases of genetic origin such as the "inborn errors" of metabolism have been reported in the aborigine.

The examathemata show a similar incidence in the white and native populations. The majority, after one attack, having a prolonged immunity. They do, however, differ in two respects. The first is that due to the more isolated nature of some of the aboriginal groups the disease appears in a more epidemic form and affects many older people; the second is increased frequency of respiratory complications, which have already been noted and which in many cases are due to the pre-existing respiratory disease which in turn is related to, or
probably due, to the high frequency and recurrences of coryza and influenza.

3. Diseases which exhibit a low incidence in the aboriginal population.

There are a large number of clinical entities which are apparently more rare or non-existent among the aborigines, and of these we have already discussed infective hepatitis, poliomyelitis and tuberculosis, and considered the apparent reason for the low incidence. The others may be considered in the following groups:

(a) The surgical diseases of the abdomen.
Abdominal malignancies are apparently as common in the aborigines and the white population, and trauma is also as common. However, the major differences lie in the low frequency of the "acute abdomen" in the aboriginal group. Appendicitis is rare, and I have never seen or heard of an undoubted case of acute appendicitis in a full-blood aborigine, despite the many full-blood aborigines who live in the Western Desert environment. Peptic ulceration is also extremely uncommon, although acute cholecystitis and cholelithiasis are not infrequent in the detribalized aborigine; while inguinal hernias are also occasionally seen, although I have never seen an acute obstruction. The low incidence of abdominal surgery accounts for the infrequency of bowel obstruction due to post-operative
adhesions. The most common form of acute abdomen is seen in the adult aboriginal female population, and this is acute salpingitis, which again is much more commonly seen in the detribalized aborigine.

(b) The group consisting of interrelated diseases of hypersensitivity, auto-immunity, collagen disease, allergy and various others of a more indefinite aetiology.

(i) Within the hypersensitivity and allergic group of disease atopy; serum sickness; anaphylactis; drug, dust and food hypersensitivity; and allergy have never been described, and I have never seen an example in the Western Desert area, despite the much more frequent use of the sulphonamides and penicillin as well as the more potent antibiotics in the aboriginal as compared with the white population. No proven cases of contact dermatitis, naticaria or hay fever have been recorded or observed, although all three are common in the white population of the Western Desert. Cleland (1928) did note one case of asthma in an aboriginal but my experience is that asthma of an allergic nature is unknown, although it is frequently seen in both the white and part-aboriginal populations. Therefore, even allowing for some difficulties in diagnosis, and the comparative rarity of some of the conditions described, there is undoubtedly a decreased
incidence of these manifestations in the Western Desert aborigines, despite the more extensive use of the common drugs associated with these conditions. While diseases of hypersensitivity are rare or absent, it must be borne in mind that hypersensitivity itself can still be demonstrated in the aborigine if we accept that the Mantoux reaction is a form of hypersensitivity to the tubercle bacillus and if we believe that the mechanism of Mantoux conversion is the same in both the aboriginal and white races.

(ii) In the diseases of the auto-immune group, the idiopathic haemolytic anaemias, pernicious anaemia (which may belong to this group), idiopathic thrombo-cytopenic purpura, Hashimoto's disease of the thyroid, and ulcerative colitis have never been recorded in an aborigine. Similarly, despite the fairly frequent traumatic loss of one eye, sympathetic ophthalmitis has never been noted. Nephritis may also be considered in this section as there is some evidence, Walters and Israel (1964) that auto-immune mechanisms may be the principal cause of this disease. Cleland (1928) in his review of Australian aborigines, gave no reference to this disease in the literature and in a survey of practitioners' opinions, only one stated that "Bright's disease" had been observed. Even this statement is very
doubtful, as none of the symptoms describe acute glomerulonephritis or chronic glomerulonephritis. It is also significant that in the more recent literature pertaining to large groups of aborigines and their diseases neither Basedow (1932), Binns (1945), Ford (1942), Schneider (1946), Cook (1949), Davidson (1957), Lewis (1963) or Elphinstone (1960) mention acute or chronic glomerulonephritis, and I have seen no evidence of this disease in Western Desert aborigines, although I have noted it in the part-caste and white population. The only report of its occurrence in Australian aborigines is by Crotty and Webb (1960), who observed two cases but give no details as to the type, although the age groups given would suggest a chronic form. Moreover, the material they had available would make it extremely difficult to distinguish between full and part-blood aborigines, with the result that we must accept this account of acute glomerulonephritis with considerable reserve. It would appear that acute glomerulonephritis (Ellis type I) is extremely rare amongst the aboriginal population, and that both membranous (Ellis type II) and chronic glomerulonephritis are also uncommon when compared with the white population. The rarity of acute glomerulonephritis is surprising as streptococcal (group A, beta-haemolytic) infections are more common in the aboriginal than in the white population, and moreover apparently no
difference exists between the partially assimilated and
the nomadic groups in the low frequency of glomerulo-
nephritis.

(iii) The diseases of collagen are closely related
to the hypersensitivity groups. Systemic lupus
erythematosis, poly-arteritis nodosa, polymyositis,
dermatocytosis and scleroderma have never been reported
in an aboriginal subject, although this is not surprising
considering the rarity of these diseases in any race.
However, the complete absence of any reference or reports
of rheumatic or rheumatoid arthritis amongst the aborigines
is of significance as none of the authors quoted in the
previous section have noted its occurrence in their
separate series. Furthermore Campbell (1936), Gray and
Cleland (1932 and 1934) and Black and Cleland (1938) who
specifically examined groups of full-blood Central Desert
aborigines for pathological deformities, made no mention
of any example, although they noted many cases of osteo-
arthritis. Similarly in the Western Desert area, although
Vos (1964) has found many aboriginals with high titres
of $T^A_j$ antibodies (which in the white population is often
associated with rheumatoid arthritis), has seen no
evidence of this disease in the full-blood population,
although it is not uncommon in the part-caste and white
inhabitants of the area.
The incidence of rheumatic fever in the full-blood population is much more difficult to assess as a review of the literature gives apparently conflicting opinions. Cleland (1928) in his survey of diseases of the aboriginals quotes Gorrey of Queensland as stating that rheumatic fever was common, while Illingworth of N.S.W. stated that he had only seen one case and that was a post-rheumatic mitral stenosis, and he considered it to be rare. However, neither practitioner gives any further details and fails to specify whether they are considering full-blood aborigines only or including part-caste aborigines, both of whom are being covered by Cleland's survey.

Cleland himself gives some details of "heart conditions" in aborigines but none are specifically due to rheumatic fever. Even more confusion is apparent in Basedow's (1932) series where he presents a confused mixture of acute rheumatic fever, arthritis of differing aetiology and non-specific muscular and joint pains. Within his descriptions there is no evidence of the occurrence of acute rheumatic fever, and he mentions no possible cases of post-rheumatic heart disease. Binns (1945) in his series of hospital admissions in the Northern Territory noted five cases of acute rheumatism, but as
his admissions consisted of both full-blood and part-caste aborigines and he failed to differentiate between the two groups, there is doubt as to which group was affected and furthermore two also had yaws. Crotty and Webb (1960) also noted two cases, one of acute rheumatic fever and one of rheumatic heart disease in their series in the same area, but again the possibility of part-caste aborigines being included in this series is present. Neither Ford (1942) in the Bathurst and Melville Island areas, Cook (1949) in the North of Western Australia, Davidson (1957) in Western Australia nor Elphinstone (1960) in the Kimberley region have observed acute rheumatic fever or post rheumatic heart disease in full blood aborigines.

In the Western Desert area, the disease does not appear to be present, despite streptococcal infections being common, and I have never seen a case of rheumatic fever or heart involvement in this area. This is also confirmed by Davidson (1957) for the Laverton and Warburton Range area, and by Lewis (1963) for the Warburton Range area. Allan (1960) and Cook (1960) also concur with these findings in reference to the aborigines in the Mount Magnet and Meekathara region. More recently Woods (1964) and Gamdevia (1967) have investigated the
Warburton aborigines for heart and chest disease, and neither found any evidence of rheumatic heart disease.

On the evidence available, it is apparent that acute rheumatic fever and its complications are virtually or completely non-existent in the central areas of Australia amongst the aborigines, despite recurrent streptococcal infections, poor living conditions, adverse climatic conditions and sub-standard nutrition, and although in my experience the disease is commonly encountered in both the white and part-caste population of the area. Whether or not this low incidence is universal in the native population is difficult to judge, as some of the cases reported, if not all, for the Northern Territory are almost certainly part-caste, and further investigation is required to assess the true frequency in the full blood population of this area. Nevertheless, the overall incidence is apparently much lower in the aborigine than in the white population. The only mechanism at present that can be seen for this difference is a purely racial one, and it is possible that this racial difference may occur largely in the central group of Australian aborigines.

(iv) The final group contains a number of diseases which are of unknown aetiology, but are considered to
bear some relationship to "stress". These are essential hypertension, pre-eclamptic toxaemia and eclampsia. All three conditions are rare in the aboriginal population. Neither eclampsia nor pre-eclamptic toxaemia have been reported, and I have seen no evidence of these conditions in the Western Desert area, while only a few cases of essential hypertension have been described in the literature. The latter has only been noted in elderly aboriginals, and may well be within the normal range for the age of the subjects. The rarity of essential hypertension has been confirmed in other parts of Australia in the extensive series recorded by Abbie (1960) and Von Donopren (1962) who has also noted that while a slight elevation of blood pressure (both systolic and diastolic) occurs in the detribalized aborigine, the mean is still below that of the white population. Parallel changes occur in the serum cholesterol levels as has been shown by Charnock et al. (1959).
SUMMARY AND CONCLUSIONS
This study of the Western Desert aborigines of Australia was undertaken within the confines of general practice, and is subject to both the limitations and the opportunities that this entails. The aborigines of this region have been considered from three aspects: the cultural, the physical and the pathological, because it is only against the background of these three integrated facets that adequate medical therapy can be given.

Within each of the three major aspects, variations between the norms that pertain to the Western Desert aborigine and the differences and similarities between them and other aboriginal and the white inhabitants of the continent are noted.

The culture of the Western Desert aborigine has been considered from the aspect of family life, the environment, diet, and the customs surrounding birth, initiation, infanticide, geriatricide, marriage, and the arts, the language, the psychology of the people. Most of these aspects have considerable ceremony attached to them, and are surrounded by a double mythology, that of the aborigines themselves and that of the anthropologists who have studied them. In most cases there is a simple, rational explanation for the various customs, nearly all
of which are a form of preventive or social medicine and when considered against the background of their environment prior to colonization it is obvious that such customs as infanticide and geriatricide were evolved from necessity. Cannibalism has never been established in its true sense in Australia, and is an erroneous concept, as also is the belief in the widely reported practice of abortion. In my experience, the widespread belief that the aborigine does not relate sexual intercourse with conception is also erroneous, and supportive evidence that circumcision and subincision have been environmentally determined is also given. The particular medical implications of their diet, the aboriginal pattern of aggression, the complex marriage and kinship relationships and their psychology are also discussed. The discussion of the effect of Western civilization on the culture of the aborigines in general, and the Western Desert aborigines in particular, is traced historically with details of the main groups of settlers which affected them, the majority of these influences being adverse, by upsetting the balance of their environment and decimating the aboriginal population. The more recent effects of increased government aid to aborigines and the extension of the medical services to include the majority of aborigines has now been shown to have the opposite effect, and evidence of a large population explosion with the
magnification of the problems that exist at present, is to be expected in the future.

Their physical structure has been considered in detail, both from the author's work and from the available literature; because clinical measurements are of little importance unless the normal parameters of the population under consideration is known. The details include the normal growth curves for height and weight, the mean blood pressures, blood cell counts, haemoglobin estimations and such biochemical estimations as are known. Descriptions of the normal adult male and female with their range of body characteristics is given, and the pattern of growth for the child detailed. In the discussion of the gene frequency variations between the Western Desert aborigines and peripheral groups of aborigines, there is evidence that the Western Desert aborigines are derived from at least two distinct racial groups, giving for the first time support to the theory of a hybrid origin of the Australian aborigine. In the comparison between the aboriginal and Caucasian racial groups, attention is drawn to differences that exist between them as regards accepted normal physical, clinical and biochemical estimations, and it is concluded that true racial as opposed to tribal environmental factors may be responsible for some of these differences. Evidence is given that
the aborigines probably possess a more efficient iron transfer mechanism in the gut wall than the white population, and it is also postulated that the apparently abnormal reactions shown by the Western Desert aborigines to the Casoni test may be due to some variation in the P blood group similar to the variations within the E subgroup of the Rh factor which have already been shown to exist.

A survey of the diseases that may affect the aborigines in general and the Western Desert natives in particular has been made, using both personal observation and the available literature. A discussion of the epidemiology of these diseases between the various groups of aborigines and the white population shows that marked variations occur and particular attention is drawn to the lack of disease of collagen, immunity, hypersensitivity or stress in the native population.

This thesis has been prepared with several objectives. Firstly to provide a comprehensive picture, where none has previously existed of the Western Desert aborigines, and to present a comparative study of this group with other aboriginal tribes, and also with the Caucasian race, and thus, it is hoped, will be of some value to other medical practitioners dealing with aborigines and to researchers interested in this group of people.
Secondly, to present the author's experiences and researches on this particular tribal group and to correlate them with the available literature on the subject. Thirdly, to present the author's theories and conclusions on the various aspects studied and to emphasise the opportunities available for more elegant and intensive investigation of particular facets, many of which could be of fundamental importance. These include such aspects as the optimum method of assimilation of the aborigine in view of their cultural background and the present effect that Western civilization has had on it. Of the importance of the effect of assimilation on their physical characteristics and the eventual definition of parameters of physical normality when assimilation has occurred. Clinical investigation of the epidemiology of disease, the apparent lack of inborn errors of metabolism, the low or non-existent incidence of disease of hyper-sensitivity or auto-immunity, appendicitis and peptic ulceration, all require follow-up, as does the effect of assimilation and Caucasian nutrition in the aetiology of cholelithiasis and diabetes mellitus in the aborigine.

It is evident that the aborigine is considerably disadvantaged in present Australian society, and that a multi-factorial approach, of which medical attention is only one aspect, is required if he is to achieve a standard
of living similar to the white population. The most pressing requirements are for a broad based educational policy which will adequately bridge the gap between their traditional culture and Western civilization, and which emphasises the problems posed by the adoption of a Western form of living and nutrition, coupled with an active social welfare and public health campaign to ensure adequate nutritional and hygienic standards, and which retains as far as possible some of their traditions.

This policy can only be successful if its proponents have both the knowledge of the needs of the aborigines and the humanity with which to implement it, and it is in the hope of improving both these aspects that this thesis is presented.
ACKNOWLEDGEMENTS
A thesis of this description cannot be prepared and presented without the help of many persons.

While it is impossible to mention all of them, I would particularly like to express my gratitude to Miss Sadie Corner, M.B.E., Matron of Leonora Hospital, for the unstinted help she gave in both obtaining a detailed history from aboriginal patients, in teaching me the rudiments of the Wongi language, and for the insight and understanding of the Western Desert aborigines she imparted; to Mr. G. Vos, Serologist, King Edward Memorial Hospital, Perth for many stimulating discussions about the Western Desert aborigines; to Constable N. Hopkins, Laverton Police Station, whose knowledge of the local aborigines is encyclopedial; and to Mr. T. Long, Native Welfare Department, Leonora, for his unfailing and helpful cooperation.

Last, but by no means least, I must acknowledge the help of the Western Desert aborigines themselves. Their uncomplaining and happy disposition, despite their many adversities and their inherent generosity of both material goods and spirit were an experience for which I will be forever grateful.


ACTS OF PARLIAMENT - WESTERN AUSTRALIA (1911) "Aborigines Act - Amendment Act 1911" No. 42, Govt. Printer, Perth.

ACTS OF PARLIAMENT - WESTERN AUSTRALIA (1936) "Aborigines Act - Amendment Act, 1936". No. 43, Govt. Printer, Perth.

ACTS OF PARLIAMENT - WESTERN AUSTRALIA (1940) "Native Administration Act - Amendment Act 1940", No. 37, Govt. Printer, Perth.

ACTS OF PARLIAMENT - WESTERN AUSTRALIA (1941) "Native Administration Act - Amendment Act, 1941", No. 4 Govt. Printer, Perth.


ACTS OF PARLIAMENT - Western Australia (1954) "Native Administration Act - Amendment Act, 1954". No. 60, Govt. Printer, Perth.

ACTS OF PARLIAMENT - Western Australia (1954) "Native Welfare Act, 1954". No. 64, Govt. Printer, Perth.


ALLEN, J., (1960) "Personal Communication".


BERGMAN, H., (1961) "Personal Communication".


References:


BLAYTHE, N., (1961) "Personal Communication."


CANNING, G., (1963) "Personal Communication."


COOK, C.E., "Personal Communication".


DAVIDSON, W.S. (1960) "Personal Communication."


FETWIEFF, B., (1963) "Personal Communication."


JAMES, S., (1882) "Occident and Orient, Sketches on Both Sides of the Pacific." Pub. Robertson, Melbourne.


KIRKLAND, W.B., (1939) "Endemic and Epidemic Disease in the Northern Territory." Health 17:121.


LEWIS, I.S., (1962) "Personal Communication."


MACKINTOSH, N.W.G., (1951) "Fingerprints of Australian Aborigines of West Arnhem Land and Western Australia." Oceania 22:299.


MANN, I., (1961) "Personal Communication."


RISEBOROUGH, A.W., (1962) "Personal Communication."

ROBERTS, W.D., (1963) "Personal Communication."


ROTH, W.E., (1897) "Ethnological Studies among the North-West-Central Queensland Aborigines." Govt. Printer, Brisbane.


SMYTH, R.B., (1878) "The Aborigines of Victoria." 


STURT, C., (1833) "Two Expeditions into the Interior of Southern Australia During the Years 1828, 1829, 1830 and 1831." Pub. Smith, Elder, London.


THOMAS, J.T., (1958) "Personal Communication.


VOS, G.H., (1964) "Personal Communication."


VOS, G.H., KIRK, R.L., STEINBERG, A.G., (1963) "The Distribution of Gamma-Globulin Types, Gm(a), Gm(b), Gm(x) and Gm-like in South and South-East Asia and Australia." Amer. J. Hum. Gen. 15:44.


WOODS, J.D., (1879) "The Native Tribes of South Australia." Pub. Wigg, Adelaide.


The First Human Blood, ---/---, which Lacks the 'D-like' Antigen

The 'D-like' antigen has been demonstrated in all Rh positive and Rh negative human bloods including the rare D-/D- and rfr (ref. 1), as well as in rhesus and other lower monkey red blood cells, but it was not found in common laboratory animals\(^2,3\). One of the characteristics of this antigen is its capacity to induce formation of 'anti-D-like' antibodies in guinea pigs. These red cells which possess this antigen after exposure to its antibody yield eluates exhibiting the 'D-like' specificity.

The unique blood of a normal, female Australian aborigine, Mrs. E. N., which is deprived of all known antigens of the Rh–Hr system, was described recently by Vos, Vos, Kirk and Sanger\(^4\). As this rare blood was neither Rh positive nor Rh negative, an excellent opportunity was provided to determine whether it carried the 'D-like' antigen. If the ---/--- red cells lacked the 'D-like' property, the question arose of the probable theoretical implications of the relationship of the 'D-like' antigen to basic Rh substance.

Joint parallel studies of the ---/--- blood and the 'D-like' antigen were undertaken based on three criteria: (a) absorption with ---/--- red blood cells; (b) elution of the antibody; and (c) antigenicity in guinea pigs. These investigations demonstrate that this exceptional ---/--- blood devoid of Rh–Hr antigens also does not possess the 'D-like' property.

'Anti-D-like' reagents prepared by absorptions with Rh negative or Rh positive red blood cells did not produce potent reagents since all random bloods possess the 'D-like' antigen; however, using ---/--- red blood cells for absorptions, more potent reagents were obtained which would suggest that these red cells did not carry the 'D-like' property (Table 1).

Eluates were prepared according to the technique described previously\(^5\). In contrast to Rh negative bloods, the red blood cells of E. N. failed to yield eluates with 'D-like' specificity as indicated in Table 2. While we used a guinea pig anti-rhesus reagent, Vos and his co-workers used a reagent prepared from guinea pigs injected with rr red blood cells and confirmed the results shown in Table 2.

Frozen glycerinated ---/--- blood on arrival from Australia was maintained at -70°C. As a control a freshly drawn specimen of Rh negative blood was
Guinea pig serum (No. 351) diluted 1:10 and absorbed twice with washed red blood cells

<table>
<thead>
<tr>
<th></th>
<th>R₁R₁</th>
<th>R₁R₂</th>
<th>R₁r</th>
<th>R₂R₂</th>
<th>R₂r</th>
<th>r'r</th>
<th>r'r</th>
<th>r'r</th>
<th>rr</th>
<th>rr</th>
<th>---/---</th>
</tr>
</thead>
<tbody>
<tr>
<td>++/---</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0/---</td>
</tr>
<tr>
<td>R₁R₁</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0/---</td>
</tr>
<tr>
<td>rr</td>
<td>+s</td>
<td>+s</td>
<td>+s</td>
<td>+s</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0/---</td>
</tr>
</tbody>
</table>

Guinea pig No. 351 was injected twice with Rh negative red cells. Readings were taken after incubation for 45 min. at room temperature, light centrifugation and resuspension of the sedimented red cells.

Table 2

<table>
<thead>
<tr>
<th></th>
<th>R₂R₂</th>
<th>R₁R₁</th>
<th>R₂r</th>
<th>R₂r</th>
<th>rr</th>
<th>rr</th>
<th>rr</th>
<th>rr</th>
<th>---/---</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absorbed with rr</td>
<td>+ ±</td>
<td>+ ±</td>
<td>++</td>
<td>++</td>
<td>±</td>
<td>±</td>
<td>0</td>
<td>0</td>
<td>---/---</td>
</tr>
<tr>
<td>Eluate from red blood cells:</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>---/---</td>
</tr>
<tr>
<td>rr</td>
<td>+ +++</td>
<td>+++++</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0/---</td>
</tr>
</tbody>
</table>

Readings were taken after incubation at room temperature for 1 hr., light centrifugation and resuspension of the sedimented red cells.
also preserved in glycerine, frozen and maintained at -70°C. To avoid the loss of red cells incidental to deglycerinization, thawed, unwashed samples were used for injections. (Guinea pigs injected with glycerine revealed no toxic effects.) At three-day intervals each of two series of guinea pigs received two injections of 2-0 ml. of the bloods which contained approximately 1-0 ml. of sediment. When tested ten days later by two absorptions at 1:5 with rr red blood cells, of the 20 guinea pigs inoculated with the control rr blood, 12 produced 'anti-D-like' antibodies; however, none of the 20 guinea pigs inoculated with -/-/- blood showed 'anti-D-like' specificity. Four of the 20 guinea pigs inoculated with -/-/- blood produced an antibody exhibiting anti-N specificity which was not observed in the pre-inoculation specimens. Since the -/-/- red blood cells are of genotype NN, the source of stimulation is obvious.

The immunization experiments carried out in Australia were made with heat extracts of group O, rr red blood cells and -/-/- red blood cells. While the five guinea pigs injected with heat extracts of -/-/- red cells failed to produce 'anti-D-like', two of these five produced an antibody also identified as anti-N. Of the four guinea pigs injected with heat extracts of rr red blood cells, three showed very weak 'anti-D-like' specificity when absorbed with rr red cells. After absorbing these sera with the -/-/- red blood cells, all four gave 'anti-D-like' specificity of stronger activity than that obtained on absorptions with rr red blood cells.

The view that the E. N. genotype, -/-/-, is the result of suppression or deletion of that part of the chromosome bearing the Rh locus has been considered by Vos et al. Another possibility may be that -/-/- represents basic material remaining after loss of products of the Rh-Hr genes by one or another genetic mechanism, but, on the other hand, it could represent basic material on which the Rh-Hr genes deposit their products.

Based on our previous observations and the results of this joint study, it would appear that when a gene produces an Rh-Hr antigenic determinant, it simultaneously produces a 'D-like' antigen. We would like to offer the hypothesis that the 'D-like' material constitutes unaltered basic Rh substance which, by a series of mutations in man, has evolved into the complex Rh-Hr situation as it exists to-day. Such a theoretical possibility was mentioned as early as 1952 by Murray and Clark. In any event at this stage of investigation one can conclude that the 'D-like' antigen is associated with all random human
bloods bearing any one or more of the antigenic
determinant groups of the Rh–Hr system.

We thank the King Edward Memorial Hospital
for Women, Perth, for financial support, and Dr.
G. A. Kelsall for his co-operation. We also thank the
Western Australia Department of Native Welfare
for facilitating the collection of blood from Mrs.
P. N.

PHILIP LEVINE
M. J. CELANO
Ortho Research Foundation,
Raritan, New Jersey.

G. H. Vos
King Edward Memorial Hospital for Women,
Subiaco, Western Australia.

J. MORRISON
Leonora Hospital,
Leonora, Western Australia.

1 Levine, P., Rosenfield, R. E., and White, J., Amer. J. Human
2 Levine, P., Celano, M., Fenichel, R., and Sligher, H., Science, 133,
332 (1961).
3 Levine, P., Celano, M., Fenichel, R., Pollack, W., and Sligher,
4 Vos, G. H., Vos, Dell., Kirk, R. L., and Sanger, R., Lancet 1, 14
(1961).

Printed in Great Britain by Fisher, Knight & Co., Ltd., St. Albans.
THE ORIGINS OF THE PRACTICES OF CIRCUMCISION AND SUBINCISION AMONG THE AUSTRALIAN ABORIGINES

JOHN MORRISON, M.B., Ch.B., D.Obst.R.C.O.G.1,2
King Edward Memorial Hospital for Women, Perth

Circumcision and subincision are practised among many of the Australian Aboriginal tribes, and are surrounded by so many customs and myths that it is impossible to state categorically how they originated. The customs and rites have been well documented by such authorities as Love (1830), Elkin (1838) and Spencer and Gillen (1899, 1904), and there is little one can add to these descriptions. The myths have originated with tribal folklore and from various anthropologists' interpretations of these customs, particularly those surrounding circumcision, and have led to so fanciful theories as that it represents a symbolic return to a matrarchal society (Cook, 1964), or is a form of contraception (Elkin, 1888). The latter is now not generally believed, although the former appeal because it is de novo, and the theory (Cook, 1966) were based on the oft-repeated, and I firmly believe erroneous, statement that the Aborigines do not relate sexual intercourse with conception, and therefore cannot practise contraception. Despite this, I believe that, by carefully analysing the customs, the practices and the environmental factors, one can reach a logical conclusion as to how both circumcision and subincision were incorporated in the tribal customs and have attained the place they now occupy among the cultural characteristics of the Aborigines.

CIRCUMCISION

The practice of circumcision is of undoubted antiquity in the Australian continent, and Abbe (1861) has stated that he believes that the custom was introduced by the original immigrants some 6,000 to 10,000 years ago, after crossing the Surat straits. However, not all tribes practice circumcision, and those that do are mainly situated in the interior of Australia. If Abbe's hypothesis is correct, then it would be expected that the custom would be universal among the tribes that indulge in circumcision. Therefore, it would have been deleted in a random manner in the distribution of the custom within the Australian continent shows that it is practised by all the central tribes and disappears only towards the periphery, although some of the coastal tribes also practise circumcision. This therefore suggests that the custom has arisen de novo in Australia—if one believes in a uniracial origin of the Australian Aboriginal, as Abbie does. If, however, one believes in a dihybrid or trihybrid origin, then it is possible that the distribution of the practice reflects from an older race; but, again, this is unlikely because of the relatively widespread distribution.

If, however, we consider the distribution of the custom in relation to the climatic conditions, we soon realize that it is almost entirely restricted to the low rainfall area—an area characterized not only by the low humidity, but also by dry, dusty conditions and high winds, and a notable lack of surface water. These factors, which also tend to occur in other parts of the world where circumcision is performed, are well known medically to lead to recurrent inflammation under the foreskin (balanitis), and I have seen numerous cases among the male children of the western desert area. These recurrent episodes of inflammation and their attendant adhesions between the glans of the penis and foreskin in turn cause marked complications after puberty—complications for which circumcision is normally performed in western society, or which are prevented in Jewish society by circumcision at birth and by the Mohammedans by circumcision before puberty. As none of the surrounding ethnic groups practice circumcision, it is probable that the custom has arisen de novo among the Aborigines, and that it has almost certainly arisen in the area of environmental risk factors. It is also likely that the custom was transferred to some other aspect. In this case it is concentrated on the age of the boy, with the inevitable result that in time the practice is considered a significant event associated with puberty and the attainment of the status of manhood, until in those tribes among which it is practised the emphasis is completely removed from the practice as a simple measure of preventive medicine. It becomes the status of initiation, which is the mysticism of "initiation".

SUBINCISION

This practice, delicately labelled by Curr (1886) "the horrible rite", consists of incising the urethra on the underside of the penis from the glans backwards for a variable distance, but frequently as far as the peno-scrotal junction. There are four outstanding features about this rite. In the first place, it is practised only in Australia, and in the second, it is performed only by those tribes which indulge in circumcision. The third factor is that not all tribes that circumcise perform subincision, or, if they have no subincision, it is purely a preventive measure to stenosis in western society consists of either dilating the meatus, or, if it is badly stenosed, incising it and thereby removing the meatal ulceration, and among these lack of hygiene and dry, dusty, hot, abrasive environments predominate. Each episode of inflammation is followed by a greater production of scar tissue, which finally constricts the meatus, preventing adequate urination. That these complications do occur in the recently circumcised native can be seen in the western desert area, where on several occasions I have noted meatal ulceration. The treatment for meatal stenosis in western society consists of either dilating the meatus, or, if it is badly stenosed, incising it and thereby performing a subincision.

That the practice of subincision among the Aborigines is for exactly the same purpose is supported by the distribution of the rite, by the time at which it is performed (after circumcision), and by the form of the operation. It is therefore logical to reason that the practice of subincision is purely a preventive measure to...
obviate the complications of meatal stenosis, in turn a frequent complication of circumcision. Again, one can easily visualize the change that has occurred in the practice with the passage of generations. As the original reason is gradually forgotten, or as a folklore and tradition are gathered around the practice, the emphasis shifts to the age at which it is performed; and as it follows at a variable time after circumcision, it becomes a significant feature of the elevation of the “initiated” man to a higher tribal status. As the significance is altered, so does the actual operation vary, some tribes performing purely an anterior meatotomy, while others incise the whole penile urethra, either at a single operation or more frequently in stages at successive corroborees.

ANALOGOUS PRACTICES

Critics may question the preceding arguments, on the grounds that the Aborigines are a primitive race and have no surgical skill, and that the entire concept is theory. They can be easily answered by the fact that the natives do possess considerable surgical skill in other fields apart from the one under consideration; that the Aboriginal I.Q. has never been properly assessed, but most authorities, such as Albie (1961), would agree that it must approximate to the “Caucasian norm”; and that many aspects of tribal custom are based on a highly developed system of preventive and social medicine when related to the environment of this race (Morrison, 1967).

Much more pertinent to the argument is the exhibition of identical customs in a more recent and racially separate society living in the same environment—the Caucasian inhabitants of the eastern goldfields. They may be selected as a representative group, but the pattern can be seen in other white Australian communities. These people, despite the fact that this area was occupied only some 70 years ago (the major goldfield rushes of Western Australia occurring between 1889 and 1898), are all descended from European immigrants, the majority being of British extraction. They therefore have no long-standing tradition of circumcision; yet the vast majority of the early goldfield settlers’ descendants insist that all male children be circumcised. The reason for this can be easily discovered. Since the frequency of balanitis, due in the early days to the environment and to lack of washing facilities, and its associated complications, often necessitated circumcision, and since the operation is much more severe in the adult than in the child or infant, within a few years it became a routine as an act of preventive medicine to circumcise male infants at birth. At the present time these conditions no longer exist, and in fact they have not existed since the goldfields water supply was completed in 1904; but within that short space of time the custom became firmly established, and it has remained so ever since. No parents, when questioned as to why they wish their baby circumcised, can give any reason other than “It’s necessary”, “Aren’t all boys circumcised?”, and they feel that the male child will not be “initiated” into western society unless the operation is performed. It is striking that within two or three generations in a civilized society, the origin of the custom should be almost completely forgotten.

Among the older male goldfield residents meatal stenosis is not uncommon; many require frequent dilatation, and not a few have had anterior meatotomy performed, and thus have had a form of subincision performed.

CONCLUSION

A careful appraisal of the customs of circumcision and subincision in the Australian Aboriginal leads to the conclusion that both practices have arisen in the Australian continent as the result of environmental factors. Circumcision has preceded subincision, and both have originated as a form of preventive medicine, and as a response of an organized society to a specific environment. It is only later, after the reason for the origin of the customs has been forgotten, that significance has been transferred to the age of the individual undergoing the practice, and thus the practices have developed into the rites incorporated in the initiation and other status ceremonies.

REFERENCES

Curtis, L. (1964), Personal communication.
THE BIRACIAL ORIGIN OF THE AUSTRALIAN ABORIGINES

JOHN MORRISON, M.B., Ch.B., D.Obst.R.C.O.G.

King Edward Memorial Hospital for Women, Perth
THE BIRACIAL ORIGIN OF THE AUSTRALIAN ABORIGINAL

JOHN MORRISON, M.B., Ch.B., D.Obst.R.C.O.G.

King Edward Memorial Hospital for Women, Perth

The possible origins of the Australian Aborigines have attracted many theories, of which a uniracial origin as proposed by Wood Jones (1934) and Howells (1937) has received most support. Birdsell (1949), on the basis of certain physical characteristics and the ABO and the MN blood groups, proposed a trihybrid theory of origin. The initial enthusiasm for this concept rapidly waned, and Abbie (1951) stated that the differences noted by Birdsell could be explained purely on the basis of random genetic drift or isolation and in turn again supported the uniracial theory.

There are, however, two factors which seem to have almost universal support, and these are that any emigrant waves of population to the Australian continent must have occurred across the Surat Straits, and that any Melanesian influence is minimal.

POSTULATES

On the premise that any immigration occurred across the Surat Straits, then we may put forward three postulates, as follows.

1. If the Australian Aborigines are of uniracial origin, then they should be a homogeneous group, and any differences which can be detected between the various subgroups must be explainable on the basis of isolation, random genetic drift, mutation, or the effects of environment.

2. If the Australian Aborigines are of biracial origin, then the secondary wave of immigrants would have been arriving and attempting to settle along the northern coast, which is the most fertile area of Australia. The attempt to displace the primary settlers would almost certainly have led to strife. If the secondary wave were numerically small, then presumably they would either have been eliminated or assimilated by the primary race, but if it were numerically large, then the primary settlers in this region would in turn have been eliminated, or assimilated, or displaced initially along the coastal region and subsequently towards the centre of Australia, this being the least hospitable region. If complete assimilation between these two races occurred, then again the Australian Aborigines would be a homogeneous group. If, however, this did not occur, a gradient of racial characteristics would exist between the coastal and central Aborigines.

3. If the Australian Aborigines are of multiracial origin, the situation would be similar to that in the second postulate, but much more complex and more difficult to estimate.

It is therefore on a comparison of the physical characteristics of the Central Australian and the northern
and coastal Aborigines that we may expect to find any existing proof that they are not of uniracial origin.

**COMPARISON OF AUSTRALIAN ABORIGINALS**

**Somatographic Features**

The general characteristics of the northern and coastal Aborigines, as described by Foelsche (1881), Howells (1937), Love (1936), Mackintosh (1951a) and Birdsell (1946), show that they superficially form a homogeneous group, and apparently are different from the Central Australian Aborigines as described by Campbell *et alii* (1926, 1927, 1936), whose description corresponds closely with that of the Western Desert natives, with whom I have had close contact.pector, in an extensive review of the craniology of the Australian Aboriginal, also noted geographical variations. These two major groups do correspond to a certain extent to Birdsell's "Carpenterian" and "Murraylian" types, respectively, but the general somatography is of little help, as much of it may be environmentally induced. However, there are amongst the physical characteristics four features which are controlled by simple Mendelian inheritance, and which can therefore act as genetic markers and be evaluated in the two groups. These are fair skin pigmentation, fair hair colour, straightness of hair and the dermatoglyphics, of which the first three are autosomally related.

These first three features are rare in the northern and coastal natives, but they are not uncommon amongst the Western Desert and Central Australian Aborigines. The incidence of deepening pigmentation and hair colour and straightness is greatest in the male, less in the female, and least amongst the children, of whom some 20% are very fair. The climate of the interior of Australia is such that these features are not likely to have been induced environmentally. The dermatoglyphics have been studied by Cummins (1961), Campbell *et alii* (1956) and Macdonald (1956), and there is a significant decrease in the percentage of whorl patterns between the northern and coastal natives on the one hand and those in Central Australia on the other.

**Blood Group and Serum Factors**

Over the past few years, a considerable amount of work has been done on the distribution of these genetically inherited characteristics amongst the Australian Aboriginals, and they may be divided into two groups, as follows.

**Factors of Similar Distribution in all Australian Aboriginals**

These consist of the Rhesus and Duffy groups, which are rare, while the Lutheran groups are 100% absent amongst the various groups studied in both the coastal and Central Australian regions. In the Lewis system, approximately 7% of subjects are Lewis-positive, while the F group shows a fairly even distribution (Simmons, 1958); but the sera are small to be significant. Within the Kidd group there are no marked local variations, and Vos (1964) has demonstrated that a large percentage of subjects have anti-Jka amongst those who are Kidd-negative.

**Factors Showing a Gradient of Genetic Frequency from the Peripheral to the Central Tribes**

The ABO Group.—The presence of B in the Cape York Peninsula is almost certainly a recent introduction, according to Simmons *et alii* (1958). For all practical purposes, this subgroup is not present amongst Australian Aborigines. In fact, all examples of the A subgroup are A$_o$. The isoenetic distribution of these has been studied by Birdsell and Boyd (1940), and the studies have been extended by Birdsell and Boyd (1940) and Simmons *et alii* (1954, 1958), who have demonstrated that the highest distribution of A$_o$ is present in the Western Desert native, rising from a genotype frequency of 0-218 in the north to 0-446 in the centre of Australia.

The Rhessus Group.—Although all Aborigines are Rh-positive, apart from the one known subject who has complete deletion of all Rh factor (discussed by Levine *et alii*, 1962), there is a considerable genetic gradient in the distribution of the subgroups, according to Wilson *et alii* (1944), Simmons *et alii* (1944, 1946, 1953) and Simmons (1958). Rp is maximal in the northern coastal region with a frequency of 0-185, and minimal in the Western Desert with a frequency of 0-085; the corresponding frequencies for R$_2$ are 0-121 and 0-207, while R$_C$ has a maximum in the Western Desert of 0-446 and in the north of 0-025. Vos *et alii* (1962), in a study of the B$^-$ subgroup, showed that the gene frequency decreased from the Western Desert from 0-47 to 0-34 towards the coast, and was not present in the northern regions.

The MNS System.—No Aborigines possess the S factor, but there is a decline of S from the Northern Territory to 0-18 in the Western Desert (Wilson *et alii*, 1944; and Simmons, 1958).

**Go Serum Factor**.—The incidence of Go, according to Kirk *et alii* (1963), is highest in the Western Desert, where the frequency is 0-956, and diminishes peripherally to 0-727 in Cape York. Central Australian group of natives has a frequency of 0-840 to 0-194. The distribution of Gc$^b$ is of interest, as it has been described only amongst Australian Aborigines, and the frequency distribution varies from 0-035 in the Cape York Peninsula to 0-194 in the Western Desert.

**The Gm Serum Factors**.—The clinal distribution of Gm$^a$ decreases from 0-730 in the Western Desert to 0-577 in the Kimberleys, the ratios for Gm$^{ab}$ are respectively 0-270 and 0-256, while Gm$^b$ is present at the periphery with a frequency of 0-167 and is not present at all in the Western Desert area, according to Kirk *et alii* (1958).

**The Haptoglobins**.—The distribution of Hp$^a$ decreases from 0-54 in Cape York to 0-37 in Central Australia, Hp$^b$ is the reciprocal. These factors have been investigated by Budtz-Olsen (1958) and Kirk *et alii* (1961, 1962).

**The Transferrins**.—Although actual figures are not available, Kirk and Lai (1961) confirm that within the Lewis system the incidence of Tf$^a$ decreases markedly as one moves from the centre to the periphery.

**DISCUSSION**

We have therefore a number of genetic markers amongst the Aboriginal population which show significant differences between Cape York, Central Australian group of natives and those situated on the periphery of the continent. The various mechanisms that may account for this distribution are as follows.

**Isolation**

This is the most commonly quoted cause of the varying gene frequencies seen in the Central Australian natives; but the more closely the problem is studied, the more difficult it is to substantiate this deduction, as the laws of kinship, marriage and hospitality are designed to ensure genetic mixing, and the corroboree is a frequent source of genetic interchange. The rapid spread of such infectious diseases as smallpox prior to white colonization, and gonorrhoea after it, shows that isolation is more apparent than real, and, the presence of shell ornaments at Warburton (Corner, 1963) shows that the Central Australian Aborigines had contact with coastal natives. However, there is a greater degree of isolation between certain groups of the Central Australian natives. For example, as McCarty (1959) has emphasized, this situation exists between the natives of the Warburton Ranges and those of Cundeele, as they are separated by a virtually impassable desert, and the only common meeting ground is the nearest point of this area where it approaches the peripheral group.

**Random Genetic Drift**

Blood groups and serum factors are particularly valuable as genetic markers, in that they fulfill many of Boyd's postulates (1940)—that is, they are independently inherited, they are subject to a low rate of mutation and they are not influenced by environment. Although Mourant (1950) and Kirk (1961) have shown a marked interdependence between the ABO and Rh systems, this does not
apply to the Australian Aborigines, who are all Rh-positive. Figure 1 shows the gradient in gene frequency between the peripheral and central Aborigines as exhibited in the ABO, Rh and Mn blood groups, and in the haptoglobin, Gc and Gm serum factors. These six independently inherited characteristics have a highly significant correlation, which is in direct opposition to the theory of random genetic drift.

### Environmental Factors

These could in no way account for the differences in the frequency of the genetic markers at present under consideration, according to Boyd (1949).

---

**Figure 1: Gene frequency gradients.**

The Australian Aborigine is Not of Unracial Origin

If this is true, then, according to the initial postulate, the gene frequency of the various factors will show a gradient between the peripheral and central natives, the degree of which will depend upon the initial genetic differences between the primary and subsequent immigrants and upon the degree to which primary immigrants have been diluted by the subsequent race.

It is apparent from Figure 1 that the gradient of the gene frequencies between the peripheral and central natives corresponds almost directly to a dilution effect. Moreover, this hypothesis is very strongly supported by the distribution of the E blood group. The fact that the E blood group is practically restricted to the centre of Australia suggests that it could have occurred in this locality by only one of two mechanisms—that is, either it has occurred by mutation, or it has been present from the preceding race. The very rapid diminution in its frequency towards the periphery on the one hand, and the similar distribution between the Warburton and Cundelee Aborigines of Western Australia (who are socially isolated on the other), would make the theory of mutation most unlikely, unless one postulated that it arose spontaneously in both areas at a similar time. Gmab and Gsab show an inverse ciliary distribution and support the hypothesis.

### CONCLUSION

The only theory that at present fits the known distribution of certain physical characteristics, blood groups and serum factors amongst Australian Aborigines is that they are derived from at least two successive waves of immigrants, who were genetically dissimilar.

**REFERENCES**


Corner, S. (1961), Personal communication.


Vos, G. H. (1964), Personal communication.


The Obstetrical Performance of Urban Aborigines

John Morrison

King Edward Memorial Hospital, Bagot Road, Subiaco, Western Australia

Volume 8, Number 2, May, 1968, pages 95 to 98
The Obstetrical Performance of Urban Aborigines

John Morrison*

King Edward Memorial Hospital, Bagot Road, Subiaco, Western Australia

Sir John Cleland commented in 1966 that there are virtually no records available of aboriginal obstetrical performance, except for a single case he himself described in 1927. This paper presents a retrospective analysis of 100 consecutive deliveries of aboriginal patients in King Edward Memorial Hospital in a period of 14 months, and compares their morbidity with the over-all incidence in clinic patients.

Material

The aboriginal patients were under the care of the Native Welfare Department of Western Australia and were derived from the same area as the caucasian clinic patients. All were detribalised and lived in urban areas; the majority lived close to Perth and were part-caste. The caucasian clinic patients were booked on the basis of either abnormal obstetric factors or low income, and in themselves represented a high risk group. The terms used are those defined by the Royal College of Obstetricians and Gynaecologists in their standard tables.

Analysis

General Data

There were 26 primiparae and 74 multiparae in the series, of which 11 primiparae and 32 multiparae were unbooked patients. Eight of the primiparae and 69 of the multiparae were married or living in a stable de facto relationship. The mean ages at delivery are shown in table I.

Previous Obstetrical History

The previous obstetrical performance is shown in table 2. Of the multiparous patients with previous Caesarean section, only 1 patient had a repeat operation. The previous stillbirth rate was 35 and the perinatal mortality was 81. The abortion rate was 6.5%.

Antenatal Period

The high incidence of anaemia and infection of both genital and urinary tracts is shown in table 3. The true incidence is probably higher, as a large number of patients were unbooked. The 3 patients with cardiac disease belonged to Grade 1, Grade 2 and Grade 4, respectively. There were 2 patients with diabetes mellitus and in both the condition was moderately severe. The incidence of toxaemia was similar in the 2 groups of patients, but in the aborigines the type of the toxaemia could not be classified due to their late booking. The occurrence of antepartum haemorrhage was twice as common in aboriginal as in caucasian patients, and a similar ratio was seen in twin pregnancy.

Labour and Puerperium

The principal complications of labour and the puerperium are shown in table 4. Surgical induction of labour was somewhat less common in the aboriginal group, the main indication being toxaemia. Of the 4 breech presentations, 1 required Caesarean section for disproportion. The over-all Caesarean section rate was 13%, a relatively high figure, the major causes being disproportion (10%) and deep transverse arrest or persistent occipito-posterior position (7%). The patient with prolapsed cord was delivered vaginally as the foetus was dead. Forceps or vacuum extraction was performed in 14% of cases, the principal indication being foetal distress, frequently associated with either deep transverse arrest or persistent occipito-posterior position. Craniotomy was performed on one occasion in a primipara who was admitted in obstructed labour at 8 cm. dilatation, the foetus having already died. Both the post partum haemorrhage and manual removal rates were significantly

*Present Address: “Shepley”, Blindley Heath, Near Lingfield, Surrey, U.K.
Table 1. Age and Parity — Aboriginal Patients

<table>
<thead>
<tr>
<th>Parity</th>
<th>1</th>
<th>2-4</th>
<th>5-7</th>
<th>8-10</th>
<th>11-13</th>
<th>14-16</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age in years</td>
<td>19.3</td>
<td>22.6</td>
<td>28.2</td>
<td>31.2</td>
<td>38.5</td>
<td>40.3</td>
</tr>
<tr>
<td>No. of patients</td>
<td>26</td>
<td>33</td>
<td>24</td>
<td>7</td>
<td>6</td>
<td>4</td>
</tr>
</tbody>
</table>

Table 2. Previous Obstetric Performance — Aboriginal Patients

<table>
<thead>
<tr>
<th></th>
<th>Primiparae</th>
<th>Multiparae</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Booked</td>
<td>Non-booked</td>
</tr>
<tr>
<td>Number of patients</td>
<td>24</td>
<td>42</td>
</tr>
<tr>
<td>Abortions</td>
<td>3</td>
<td>12</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Stillbirths</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Viable births</td>
<td>0</td>
<td>166</td>
</tr>
<tr>
<td>Neonatal deaths</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Caesarean section</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Breech delivery</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Forceps or vacuum extraction</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 3. Complications of the Antenatal Period

<table>
<thead>
<tr>
<th></th>
<th>Primiparae*</th>
<th>Multiparae*</th>
<th>% incidence Aborigines</th>
<th>% incidence K.E.M.H.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B.</td>
<td>N.B.</td>
<td>B.</td>
<td>N.B.</td>
</tr>
<tr>
<td>Anaemia</td>
<td>4</td>
<td>0</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>Moniliasis</td>
<td>6</td>
<td>2</td>
<td>17</td>
<td>7</td>
</tr>
<tr>
<td>Trichomoniasis</td>
<td>3</td>
<td>2</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>Gonorrhoein</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Urinary tract infection</td>
<td>4</td>
<td>0</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Rh incompatibility</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Pre-eclamptic toxaemia</td>
<td>4</td>
<td>0</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Essential hypertension</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Unspeficd toxaemia</td>
<td>5</td>
<td>5</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Twin pregnancy</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Placenta praevia</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Abruptio placenta</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>A.P.H. of doubtful origin</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

*Aboriginal Patients, B—Booked; N.B.—Non-booked.
### Table 4. Complications of Labour and Puerperium

<table>
<thead>
<tr>
<th></th>
<th>Primiparae</th>
<th>Multiparae</th>
<th>% Incidence Aborigines</th>
<th>% Incidence K.E.M.H.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B.</td>
<td>N.B.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Artificial rupture of membranes</td>
<td>4</td>
<td>3</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Prolapsed cord</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Prolonged labour</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Disproportion</td>
<td>1</td>
<td>4</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Deep transverse arrest and Persistent occipito-posterior</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Breech delivery</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Forceps or vacuum extraction</td>
<td>3</td>
<td>3</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>L.U.S.C.S.</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>Postpartum haemorrhage</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Manual removal</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Puerperal pyrexia—genital</td>
<td>3</td>
<td>4</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Puerperal pyrexia—extra-genital</td>
<td>1</td>
<td>3</td>
<td>4</td>
<td>7</td>
</tr>
</tbody>
</table>

### Table 5. Mean Duration of Labour — Aboriginal Patients

<table>
<thead>
<tr>
<th></th>
<th>Primiparae</th>
<th>Multiparae</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B.</td>
<td>N.B.</td>
<td>B.</td>
<td>N.B.</td>
</tr>
<tr>
<td>Number of patients</td>
<td>14</td>
<td>6</td>
<td>38</td>
<td>26</td>
</tr>
<tr>
<td>1st stage</td>
<td></td>
<td></td>
<td>13 hr. 14 min.</td>
<td>14 hr. 45 min.</td>
</tr>
<tr>
<td>2nd stage</td>
<td></td>
<td></td>
<td>53 min.</td>
<td>52 min.</td>
</tr>
<tr>
<td>3rd stage</td>
<td></td>
<td></td>
<td>12 min.</td>
<td>6 min.</td>
</tr>
</tbody>
</table>

### Table 6. Foetal Outcome — Aboriginal Patients

<table>
<thead>
<tr>
<th></th>
<th>Primiparae</th>
<th>Multiparae</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B.</td>
<td>N.B.</td>
<td>B.</td>
<td>N.B.</td>
</tr>
<tr>
<td>No. of patients</td>
<td>15</td>
<td>9</td>
<td>42</td>
<td>34</td>
</tr>
<tr>
<td>Average birth-weight of singletons (g.)</td>
<td>3415</td>
<td>2639</td>
<td>3463</td>
<td>3246</td>
</tr>
<tr>
<td>Average birth-weight of twins</td>
<td>—</td>
<td>—</td>
<td>2594</td>
<td>—</td>
</tr>
<tr>
<td>No. of infants &lt; 2500 g.</td>
<td>0</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>No. of infants &gt; 4000 g.</td>
<td>2</td>
<td>0</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>No. of infants with foetal distress</td>
<td>1</td>
<td>1</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>No. of infants with major congenital abnormalities</td>
<td>1</td>
<td>—</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>No. of stillbirths</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>No. of neonatal deaths</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>
higher than in the caucasian group. The mean duration of the various stages of labour in aboriginal patients delivered vaginally is shown in table 5.

The outstanding complication of the puerperium was infection; this was in the genital tract in 11% and outside the genital tract (principally the urinary tract) in 15%. These rates were significantly higher than in the general clinic group.

**Foetal Outcome**

Table 6 gives the average birth-weight for single and multiple pregnancies. Twelve of the infants weighed less than 2500 g.: Of these, 2 were stillborn (placenta praevia (1) and accidental haemorrhage (1)) and 1 died in the neonatal period (diabetic mother with pre-eclampsia, labour induced prematurely). Only 1 infant in this group was associated with a multiple pregnancy. The cause of the prematurity could not be discovered in 5 of the cases. A further infant was delivered at term and was obviously dysmature, while the remaining one was associated with premature rupture of the membranes and intra-uterine infection. Three of the infants had major congenital abnormalities. Of the 10 infants weighing over 4000 g. the closest association was with high parity. Foetal distress, diagnosed on the basis of foetal heart rate changes and/or meconium staining of the liquor was present in 22%. Of the 5 stillbirths in the series, 2 occurred in primiparae, one being associated with essential hypertension, anaemia and post-maturity, while the other resulted from disproportion and obstructed labour. The 3 stillbirths in multiparae were associated with placenta praevia, accidental haemorrhage and "cause unknown", respectively. Of the 2 neonatal deaths, both probably resulted from intra-uterine infection. The stillbirth rate in this series of 100 patients was therefore 48.6, while the perinatal mortality rate was 67.9.

**Comment**

In Western Australia aborigines form some 3% of the population. Kirk et al. (1964) have estimated that there are some 19,000 full-blood aborigines in Australia, while the part-caste population is probably double this. They have emphasised that the combination of low abortion and high fecundity rate, even though accompanied by high perinatal mortality, must be associated over the next few generations with a rapid increase in the aboriginal population. The present study also demonstrates that the detribalised aborigine constitutes a particularly high risk in obstetrics, with a perinatal mortality twice that of social class 4 amongst the caucasian population.

**Summary**

A consecutive series of 100 aboriginal patients, delivered at the King Edward Memorial Hospital, Perth, has been studied and compared with a caucasian social class 4 group attending the same hospital.

It is evident from the study that the detribalised aborigine constitutes a high risk patient in relation to reproduction.

**References**


Appendix B.

Publications referring to the Western Desert Aborigines, in which the help of Dr. John Morrison is formally acknowledged.


3. "The Distribution of the Gamma Globulin Types Gm(a), Gm(b), Gm(x) and Gm-like in South and Southeast Asia and Australia." G.H. Vos, R.L. Kirk and A.G. Steinberg, (1963), Amer. J. Hum. Gen. 15, 44.
UNIVERSITY OF GLASGOW

FORM OF APPLICATION FOR DEGREE OF M.D.

"A Cultural, Physical and Pathological Study of the Western Desert Aborigines of Australia."

TITLE OF THESIS:

SURNAME: Morrison
OTHER NAMES: John

ADDRESS: 12 Moriac Street, Moorooka, Qld., Australia. 4105

DATE OF BIRTH 17.2.33 DATE OF GRADUATION AS M.B., Ch.B. (Glasgow) 1956

OTHER QUALIFICATIONS AWARDING INSTITUTION DATE OF AWARD
F.R.C.S. R.C.S. Ed. 1968

MEDICAL APPOINTMENTS HELD SINCE GRADUATION

Designation of Post where held from to
See attached list. ............................................. ...........................

NAME AND ADDRESS OF GENERAL PRACTICE, HOSPITAL, DEPARTMENT, LABORATORY OR OTHER INSTITUTION WHERE WORK FOR THIS THESIS WAS UNDERTAKEN

General practice, Kalgoorlie, Western Australia.

General practice, Leonora, Western Australia.

DECLARATION

I declare that the work has been done and the thesis composed by myself, and that the books and papers cited were all consulted by me personally, unless it is otherwise stated.

COLLABORATIVE WORK

where material based on work undertaken in collaboration with others is included in the thesis a further and separate statement must be submitted clearly defining the candidate's individual contribution.

DATE: 18/12/72 SIGNED:

CERTIFICATION

I hereby certify that the above named candidate for the degree of M.D. has been engaged since graduation for at least one year either in scientific work bearing directly on his profession or in the practice of Medicine or Surgery.

PERIOD CERTIFIED: 1957-1973

SIGNED: O.V. Morrison 7/8/5.

DATE: 18. 12. 73.
ADDRESS: 12, Arionie St, Moorooka Ht., Queensland, Australia.
POSITION: Medical Practitioner.
<table>
<thead>
<tr>
<th>Designation of Post</th>
<th>where held</th>
<th>from</th>
<th>to</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.M.O. Medicine</td>
<td>Glasgow Royal Infirmary</td>
<td>1956</td>
<td>1957</td>
</tr>
<tr>
<td>J.M.O. Surgery</td>
<td>Glasgow Royal Infirmary</td>
<td>1956</td>
<td>1957</td>
</tr>
<tr>
<td>S.H.O. Gynaecology</td>
<td>Dryburn Hospital, Durham</td>
<td>1957</td>
<td>1958</td>
</tr>
<tr>
<td>S.H.O. Obstetrics</td>
<td>Dryburn Hospital, Durham</td>
<td>1957</td>
<td>1958</td>
</tr>
<tr>
<td>General Practitioner</td>
<td>Kalgoorlie, West. Australia</td>
<td>1958</td>
<td>1960</td>
</tr>
<tr>
<td>General Practitioner</td>
<td>Leonora, Western Australia</td>
<td>1960</td>
<td>1963</td>
</tr>
<tr>
<td>Demonstrator, Anatomy</td>
<td>University of W. Australia</td>
<td>1963</td>
<td>1964</td>
</tr>
<tr>
<td>Lecturer, Anatomy</td>
<td>University of W. Australia</td>
<td>1964</td>
<td>1965</td>
</tr>
<tr>
<td>Registrar, Surgery</td>
<td>Royal Perth Hospital, W.A.</td>
<td>1965</td>
<td>1966</td>
</tr>
<tr>
<td>Registrar, O. &amp; G.</td>
<td>K.E.M.H., Subiaco, W. A.</td>
<td>1966</td>
<td>1967</td>
</tr>
<tr>
<td>Lecturer, O. &amp; G.</td>
<td>U.C.H., University of London</td>
<td>1968</td>
<td>1969</td>
</tr>
<tr>
<td>Senior Lecturer, O. &amp; G.</td>
<td>University of Queensland</td>
<td>1969</td>
<td>1973</td>
</tr>
<tr>
<td>Reader, O. &amp; G.</td>
<td>University of Queensland</td>
<td>1973</td>
<td>present</td>
</tr>
</tbody>
</table>
"A Cultural, Physical and Pathological Study of the Western Desert Aborigines of Australia."

Summary of Thesis presented for the Degree of Doctor of Medicine, University of Glasgow,

by John Morrison, M.B., Ch.B.
This study of the Western Desert aborigines of Australia was undertaken within the confines of general practice, and is subject to both the limitations and the opportunities that this entails. The aborigines of this region have been considered from three aspects: the cultural, the physical and the pathological, because it is only against the background of these three integrated facets that adequate medical therapy can be given.

Within each of the three major aspects, variations between the norms that pertain to the Western Desert aborigine and the differences and similarities between them and other aboriginal and the white inhabitants of the continent are noted.

The culture of the Western Desert aborigine has been considered from the aspect of family life, the environment, diet, and the customs surrounding birth, initiation, infanticide, geriatricide, marriage, and the arts, the language, the psychology of the people. Most of these aspects have considerable ceremony attached to them, and are surrounded by a double mythology, that of the aborigines themselves and that of the anthropologists who have studied them. In most cases there is a simple, rational explanation for the various customs, nearly all
of which are a form of preventive or social medicine and when considered against the background of their environment prior to colonization it is obvious that such customs as infanticide and geriatricide were evolved from necessity. Cannibalism has never been established in its true sense in Australia, and is an erroneous concept, as also is the belief in the widely reported practice of abortion. In my experience, the widespread belief that the aborigine does not relate sexual intercourse with conception is also erroneous, and supportive evidence that circumcision and subincision have been environmentally determined is also given. The particular medical implications of their diet, the aboriginal pattern of aggression, the complex marriage and kinship relationships and their psychology are also discussed. The discussion of the effect of Western civilization on the culture of the aborigines in general, and the Western Desert aborigines in particular, is traced historically with details of the main groups of settlers which affected them, the majority of these influences being adverse, by upsetting the balance of their environment and decimating the aboriginal population. The more recent effects of increased government aid to aborigines and the extension of the medical services to include the majority of aborigines has now been shown to have the opposite effect, and evidence of a large population explosion with the
magnification of the problems that exist at present, is to be expected in the future.

Their physical structure has been considered in detail, both from the author's work and from the available literature; because clinical measurements are of little importance unless the normal parameters of the population under consideration is known. The details include the normal growth curves for height and weight, the mean blood pressures, blood cell counts, haemoglobin estimations and such biochemical estimations as are known. Descriptions of the normal adult male and female with their range of body characteristics is given, and the pattern of growth for the child detailed. In the discussion of the gene frequency variations between the Western Desert aborigines and peripheral groups of aborigines, there is evidence that the Western Desert aborigines are derived from at least two distinct racial groups, giving for the first time support to the theory of a hybrid origin of the Australian aborigine. In the comparison between the aboriginal and Caucasian racial groups, attention is drawn to differences that exist between them as regards accepted normal physical, clinical and biochemical estimations, and it is concluded that true racial as opposed to tribal environmental factors may be responsible for some of these differences. Evidence is given that
the aborigines probably possess a more efficient iron transfer mechanism in the gut wall than the white population, and it is also postulated that the apparently abnormal reactions shown by the Western Desert aborigines to the Casoni test may be due to some variation in the P blood group similar to the variations within the E subgroup of the Rh factor which have already been shown to exist.

A survey of the diseases that may affect the aborigines in general and the Western Desert natives in particular has been made, using both personal observation and the available literature. A discussion of the epidemiology of these diseases between the various groups of aborigines and the white population shows that marked variations occur and particular attention is drawn to the lack of disease of collagen, immunity, hypersensitivity or stress in the native population.

This thesis has been prepared with several objectives. Firstly to provide a comprehensive picture, where none has previously existed of the Western Desert aborigines, and to present a comparative study of this group with other aboriginal tribes, and also with the Caucasian race, and thus, it is hoped, will be of some value to other medical practitioners dealing with aborigines and to researchers interested in this group of people.
Secondly, to present the author's experiences and researches on this particular tribal group and to correlate them with the available literature on the subject. Thirdly, to present the author's theories and conclusions on the various aspects studied and to emphasise the opportunities available for more elegant and intensive investigation of particular facets, many of which could be of fundamental importance. These include such aspects as the optimum method of assimilation of the aborigine in view of their cultural background and the present effect that Western civilization has had on it. Of the importance of the effect of assimilation on their physical characteristics and the eventual definition of parameters of physical normality when assimilation has occurred.

Clinical investigation of the epidemiology of disease, the apparent lack of inborn errors of metabolism, the low or non-existent incidence of disease of hyper-sensitivity or auto-immunity, appendicitis and peptic ulceration, all require follow-up, as does the effect of assimilation and Caucasian nutrition in the aetiology of cholelithiasis and diabetes mellitus in the aborigine.

It is evident that the aborigine is considerably disadvantaged in present Australian society, and that a multi-factorial approach, of which medical attention is only one aspect, is required if he is to achieve a standard
of living similar to the white population. The most pressing requirements are for a broad based educational policy which will adequately bridge the gap between their traditional culture and Western civilization, and which emphasises the problems posed by the adoption of a Western form of living and nutrition, coupled with an active social welfare and public health campaign to ensure adequate nutritional and hygienic standards, and which retains as far as possible some of their traditions.

This policy can only be successful if its proponents have both the knowledge of the needs of the aborigines and the humanity with which to implement it, and it is in the hope of improving both these aspects that this thesis is presented.