ABORIGINAL INFANT AND TODDLER MORTALITY AND MORBIDITY IN CENTRAL AUSTRALIA 1965 - 1969

A Thesis submitted for the Degree of Doctor of Medicine by

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TABLE OF CONTENTS

Page

Page

Page

INDEX OF TABLES

Page

Page

Page

INDEX OF FIGURES

Page

SUMMARY

The thesis describes, in some detail, the historical, social and environmental factors which have led to the present poor state of Aboriginal child health. High mortality and morbidity rates, amongst infants and secotrants in Central Australia, during the five year period from 1965 to 1969, are discussed and many relevant data presented.

The most common causes of sickness and death in this group are Diarrhoeal disease and Respiratory infection. Aetiologies of these conditions and therapeutic difficulties are discussed. Geographical variations in morbidity are demonstrated, and an investigation into the relationship between family size, or overcrowding, and infection rate is described. The results of extensive anthropometric, audiometric and chest X-ray surveys indicating excessively high levels of abnormality are reported.

The role of marasmic malnutrition in potentiating common infectious disease is stressed, and data related to duration of hospital stay and mortality are set out to this end.

Malnutrition is discussed from the standpoints of lethal significance, classification and diagnosis, incidence in Central Australian children, effect on physical growth and educability and, finally, prevention.

Malnourished children are shown to stay longer in hospital and die more readily than their well-fed counterparts. The most satisfactory method of diagnosing and prognosing the marasmic type of malnutrition, which occurs commonly in these children, is found to be nutritional anthropometry. Other screening methods have been investigated, but found unsuitable for use in remote areas, or inappropriate in Marasmus, as distinct from other forms of protein-calorie disturbance. Cytology of buccal smears and skeletal radiography are two such projects, and the findings briefly reported.

Several specific nutritional deficiencies occur. Iron deficiency anaemia, Ascorbic acid desaturation, Magnesium deficiency and hypoalbuminaemia are discussed and their significance delineated. Immuno-globulins G and M are regularly above accepted normal levels, and the trends seem related more to age than any specific disease.

A high incidence of growth failure is found in the paediatric Aboriginal community. This is clearly nutritional in origin, rather than genetic, and varies considerably from place to place. A relationship exists between geographical variations in incidence of malnutrition, mortality, morbidity and environmental conditions.

Two problems are recorded, which are highly significant,

in view of the Aboriginals' desperate need for education. These are a rising incidence of severe deafness from chronic otitis media and the reduced educability of some children due to malnutrition in infancy.

Finally the main problems are opposed and some solutions suggested which are generally both inexpensive and relatively simple to instigate within the present Administrative structure.

DECLARATION OF ORIGINALITY

This thesis contains no material which has been accepted for the award of any other degree or diploma in any University, and, to the best of my belief, it contains no material previously published or written by another person, except where due reference is made in the text.

(D. K. Kirke)

ADVANCES MADE TO MEDICAL KNOWLEDGE AND PRACTICE

A comprehensive assessment of the significant factors responsible for mortality and morbidity in Central Australian Aboriginal children has not previously been available. In the past it was recognised that these children died relatively more frequently than their non-Aboriginal counterparts, but reliable statistical analyses of age specific mortalities and causes of death had not been produced.

The high incidence of malnutrition and its potentially lethal interaction with infectious disease are much better appreciated as a result of investigations reported in this thesis. Consequently treatment of hospital patients, and children in remote areas, has been markedly improved. The 1969 infant mortality rate was one-third of the 1964 level.

The extent to which unhygienic practices, poor economy, faulty dietary knowledge and overcrowded living conditions aggravate child health problems in this area is exposed.

The magnitude of the medical problem, in both its social and biological aspects, is shown to be far greater than was previously suspected. Deafness from chronic ear infection, and reduced educability due to infantile mal-

nutrition indicate that urgent measures are necessary if Aboriginal child health is to be improved by education.

Sufficient information is presented to form the basis of a far-reaching health programme. Several projects, which could be undertaken promptly and at minimal expense, are suggested.

ACKNOWLEDGEMENTS

A great number of nursing sisters, in both the N.T.M.S. and the Welfare Branch, have given willing assistance in the collection of data over the past five years. Without their help the task could not have been contemplated.

Others whose co-operation has been greatly appreciated are the school teachers, who took an enthusiastic part in the study of intellectual ability, and the people living in remote places who gave freely of their time and hospitality.

Professor George Maxwell of the Department of Child Health, at the University of Adelaide, has supervised the preparation of this thesis and given a great deal of helpful advice and, above all, encouragement. To him my special thanks are due.

ABBREVIATIONS

I.M.R.	Infant Mortality Rate.
N.T.M.S.	Northern Territory Medical Service (an organ of the Commonwealth Dept. of Health.
R.F.D.S.	Royal Flying Doctor Service.
T.M.R.	Toddler Mortality Rate.
U.N.E.S.C.O.	United Nations Educational Scientific and Cultural Organisation.

A Pitjantjatjara boy in his home environment at Musgrave Park, S.A.

PART I INTRODUCTION

HISTORICAL REVIEW

There can be no doubt that Aboriginal infant mortality has always been high. We have little evidence to show that any form of deliberate contraception was practised, but families were small. Infanticide was, and in a few areas to some extent still is, a means of population control. In her original nomadic state an Aboriginal mother could not afford to have more than one child-in-arms at any time since she was also expected to transport the various digging and carrying utensils, and forage for food. Thus a newborn child was often destroyed as its next older sibling still required to be breast fed and carried when the family moved from one camp to another. Eyre (1845) considered that it was very unusual to see a Native woman at Moorundi on the River Murray rear more than two children, and Spencer and Gillen (1899) found families of more than four children rare in the Central Australian Aranta Tribe, two or three being the general rule. Nowadays families of six and seven children are by no means uncommon, although frequently there are gaps which presumably correspond to deceased children, rather than deliberate family planning.

It is not known with certainty what diseases affected these people before the First Fleet landed in 1788,

but probably there were factors, other than infanticide, limiting family size and causing Byre to comment. Certainly not long after European contact diseases, such as Small Pox and Measles, took an enormous toll of Aboriginal lives, and in some areas practically exterminated whole communities (Sturt, 1833; Cleland, 1966). This was particularly so in the more heavily populated areas along the rivers such as the Murrumbidgee, the Darling and the Murray. Sturt (1833) mentioned all three of these rivers and described a cutaneous disease resembling Small Pox, a leprosy-like condition, starvation and Syphilis (probably the endemic form, or Yaws). There is every reason to believe that at this time adults often died prematurely and many children died in infancy. One theory is that Small Pox (and possibly other infections) was periodically introduced to Australia by Malays landing on the North Coast, before 1788. The first Small Pox epidemic reported, in 1789, quite probably had no relationship to the arrival of the First Fleet.

Had the Aboriginals had to contend with tropical diseases, such as malaria and some of the intestinal helminthic infestations which have affected many other primitive races, they may not have survived at all.

Estimations of total Aboriginal population in the very early days of European settlement vary but 250,000 -

300,000 is a reasonably widely held estimate (Couper Black, 1966). This total was presumably limited by naturally occurring food and water, and by other factors related to nomadic existence. It is often said that the Aboriginal lived in harmony with the country, neither one changing the other (Cleland, 1966). No doubt, from time to time, this ecological equilibrium was temporarily upset by droughts, floods and fires which limited population growth, as possibly did intertribal feuds, although there is considerable doubt as to whether this was significant.

The gradual spread of white men over the Continent, both explorers and the early pastoralists, helped to spread infectious disease to totally susceptible groups of natives. Open warfare often supervened when the black man tried to prevent the white man penetrating into tribal country and there are many examples of wholesale slaughter of Aboriginals, women and children included, as retribution for some alleged misdemeanour on the part of the black man. As recently as 1928, thirty-one Aboriginals were shot by a "punitive party" because some of the Aboriginals had murdered a dingo trapper who had interfered with their women. They, after all, had only obeyed their tribal law. Chaining of Aboriginals was another common practice which was finally made illegal only thirty years ago.

For obvious reasons much of this sort of historical fact is not written in any official publication, but there are still many stories told by both Aboriginals and white men, which are horrifying even if only half are true.

In 1869 John McDouall Stuart planted the British flag on Central Mount Stuart and proclaimed, "May it be a sign to the Native that the dawn of liberty, civilisation and Christianity is about to break on him". Stuart himself had encountered many hostile natives on several of his expeditions and yet he apparently felt that rapid assimmilation of black and white would ensue. There are still Aboriginals alive who remember the "punitive parties" and who have witnessed relatives being shot. Obviously Stuart's grand-sounding proclammation was rather premature.

The spread of disease, the often deliberate wholesale slaughter and the displacement of the Aboriginal from his spiritual home and hunting grounds led to a rapid decline in population. So obvious was the logical outcome of this decline that, forty years ago, extinction of the race was confidently predicted. In 1928, Professor J.B. Cleland's prediction was that "the pure-blooded Australian Aborigine is fast dying out" and "only a few years will see in all probability, the complete disappearance of pure-blooded natives". His prediction was only too true of Tasmania

where there are no Aborigines left. At this time the entire full blood population was estimated to be 25,000 although there may have been more people living in the Western Desert than was generally realised. In any case in less than 150 years the Aboriginal population was reduced to approximately one tenth.

POPULATION

The total full-blood Aboriginal population in the Commonwealth is approximately 40,000, and is increasing at a rate of three percent per annum. There are also about 80,000 part-Aboriginal Australians. It is not certain when the population decline ceased and the increase began, but it was probably soon after the Second World War. Welfare services and Medical attention, in particular, Penicillin, became more widely available and acceptable, and the social conscience of a few people was roused. Efforts were made by both blacks and whites to save the lives of Aboriginals who previously would have been allowed to die.

If the population was 25,000 in 1949, a three percent per annum increase would mean approximately 40,000 in 1969. At the moment, the population is increasing at that rate, but whether it has done so for 20 years, or whether the population never fell as low as 25,000 or whether the increase started, say, 30 years ago cannot be stated unequivocally.

Geographical Distribution.

20,000 full bloods live in the Northern Territory, 12,500 in the Northern Division and 7,500 in the Southern Division. There are no full blood Aboriginals in Tasmania, possibly six or ten in Victoria, one hundred in New South Wales, and the remainder distributed over Western and South Australia and Queensland in the approximate ratio of 3:1:4.

The area served by the Northern Territory Medical Service includes the far North of South Australia, which involves a further 1,000 full bloods.

Distribution by Domicile.

The population in Central Australia is still rather unstable in that small groups tend to move from one place to another, but this tendency is becoming less marked. Generally speaking there are four types of domiciliary area, namely -

1. Welfare Branch Settlements in the N.T. and Aboriginal Affairs Department Reserves in South Australia.
2. Church Missions
3. Pastoral Properties (or Cattle Stations), and
4. on the fringes of townships, railway sidings and so on.

This latter group, of fringe dwellers, is so small as to be scarcely significant, except in the eyes of tourists

who usually see only this group. The other groups, on Settlements (or Reserves), Stations and Missions are numerically in the ratio of approximately three to two to one respectively, each unit being less than 1,500.

In the study area there are ten Settlements and Reserves:- Amoonguna, Areyonga, Docker River, Haast's Bluff, Jay Creek, Papunya, Warrabri, Yuendumu, Amata (or Musgrave Park) and Indulkana. The last two are in South Australia. These communities vary in size from 80 to 1,000 people and are marked on the map (Figure 1:1) as squares.

Only three Missions exist in the area and these are Hermannsburg (Lutheran) and Santa Teresa (Roman Catholic) in the Northern Territory, and Ernabella (Presbyterian) in South Australia, and their population varies from 300 to 550. They are marked as crosses on the map.

The cattle stations are too many to ennumerate, but several carry a relatively large Aboriginal population, which amounts to 200 people in two or three cases and quite commonly more than fifty. The stations appear on the map as shaded circles.

The towns and other small communities where the fourth and smallest group, the fringe dwellers, lives are marked as hollow circles.

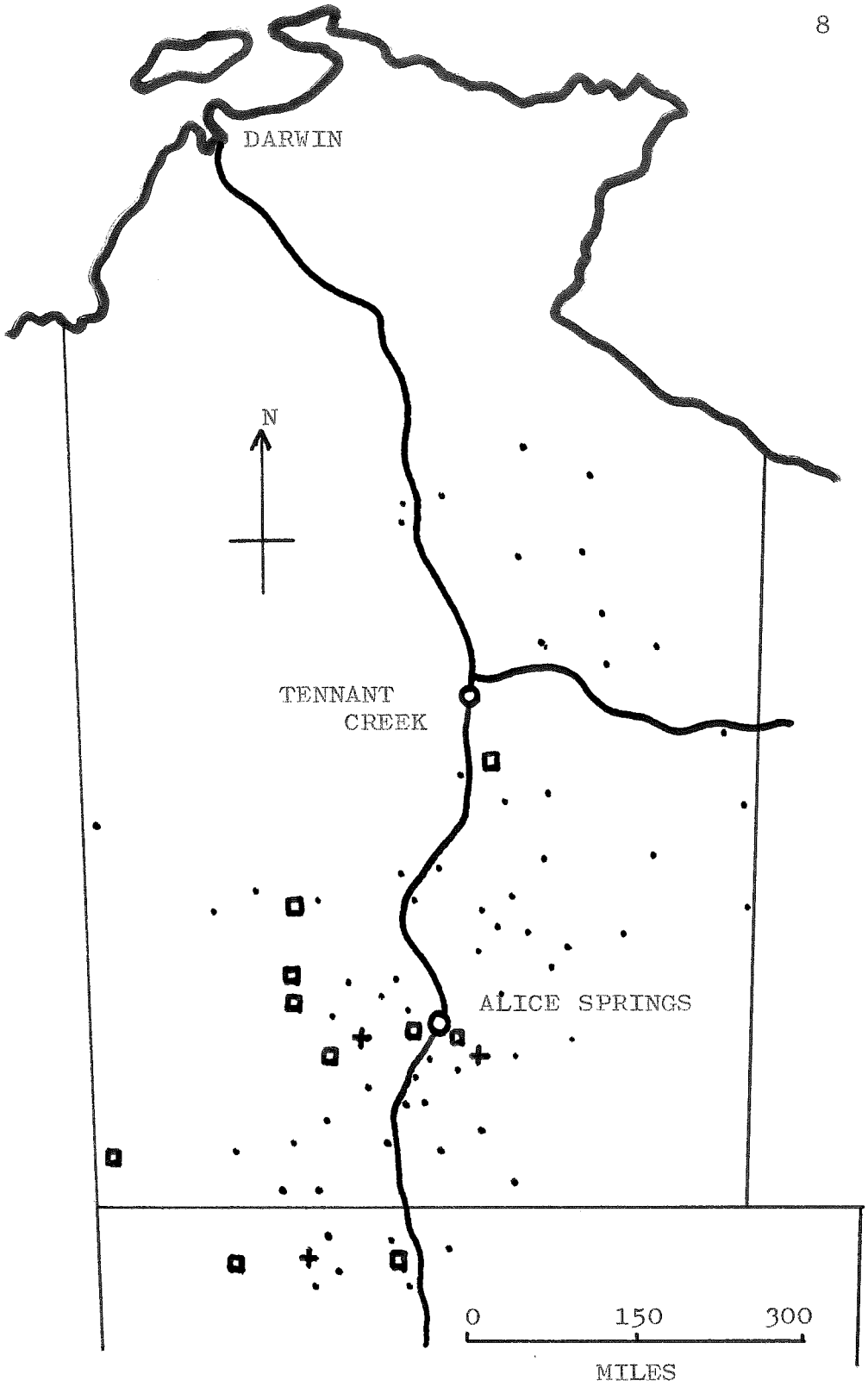


FIGURE I:1 CENTRAL AUSTRALIA - Missions (✚), Settlements (◘) and Cattle Stations (•).

Tribal Distribution.

Basically there are eight tribal entities in the group of people under consideration. Some intermixing and intermarriage between some of these subdivisions occurs, particularly in the Northern areas, but, generally, tribal marriage laws still hold. In some situations, for example in large Welfare Settlements where several tribal groups live in the same community, quite often there is friction, and even violence, between the factions because of a dispute related to marriage laws. The eight tribes and their approximate geographical locations correspond to the following groups :-

1. Wailbri
2. Alyuwarra
3. Kaiditj
4. Anmatjira
5. Aranta
6. Luritja
7. Pintupi
8. Pitjantjatjara

Of these eight, four take precedence by virtue of numbers and distinct language. The Pitjantjatjara and Pintupi are related members of the Western Desert group of tribes and speak a not dissimilar language. The other two main groups, namely Aranta and Wailbri seem to be distinct entities.

The Pintupi is the group which has had least exposure to white man's ways and some of these desert people have been in contact for little more than five years, which is

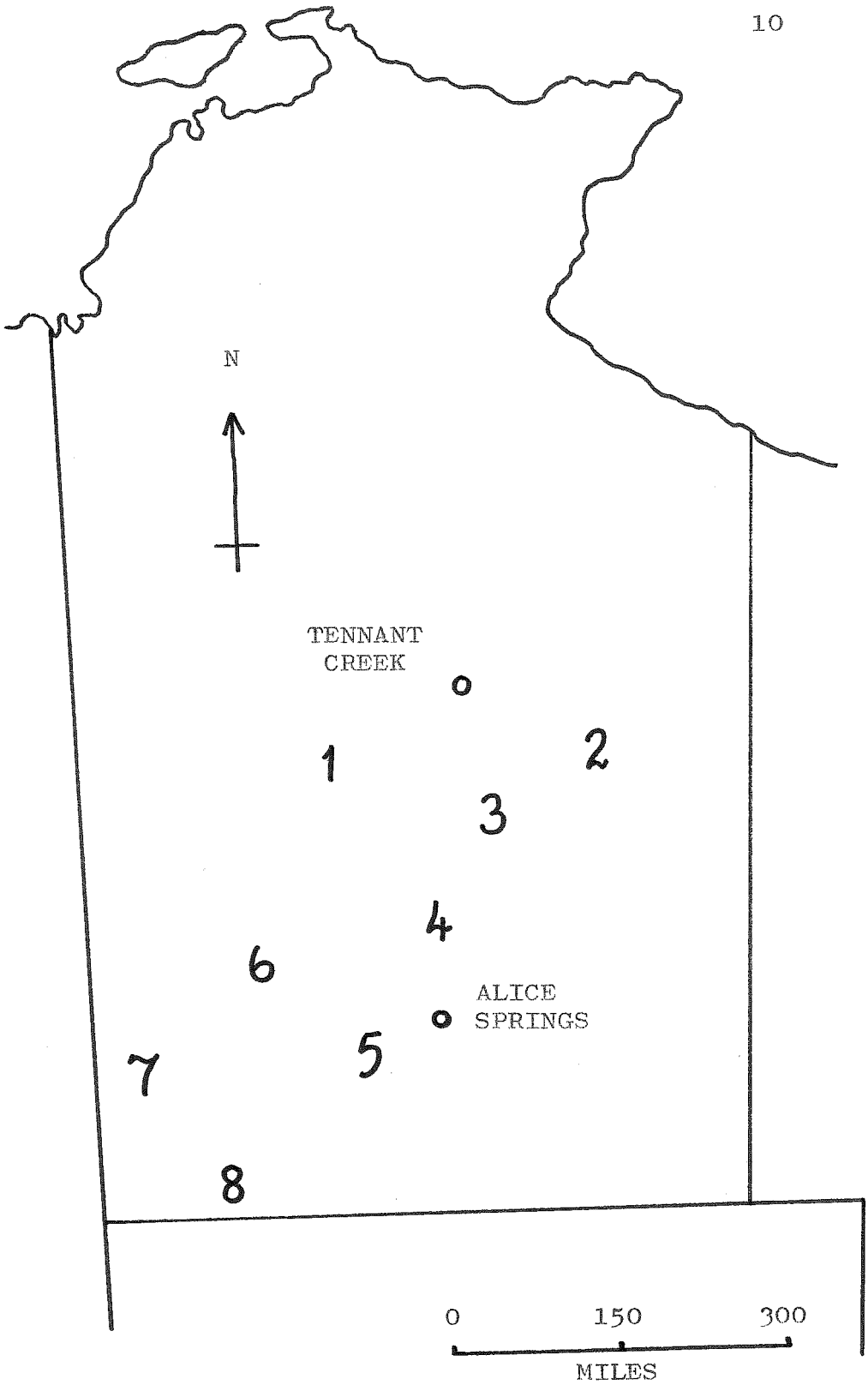


FIGURE I:2 CENTRAL AUSTRALIA - Tribal Areas.

in contrast to, say, the Aranta people at Hermannsburg, who have lived in relatively close association with white people for more than a century. The apparent differences between two such groups depend more upon environmental than tribal considerations. Example of this are the high incidences of mature-onset diabetes and coronary vascular disease in the more sophisticated Aranta at Hermannsburg, and the virtual absence of these diseases amongst the Pintupi.

Although there is still argument it seems reasonable to suppose that the tribes in Central Australia had a common origin and would probably, even 20,000 years later, have a similar genetic potential and produce similar pheno-types if their environments were standardised for long enough.

Age of Population.

The Aboriginal population is, not surprisingly, younger than the Australian community as a whole. This trend will accentuate for a few years since the birth rate is very high and the childhood mortality falling. Table I:1 contrasts the age distribution of the Aboriginals in Central Australia with that of the white population in the Northern Territory.

TABLE I:1 Age Distribution.

AGE	ABORIGINAL	WHITE (N.T.)
Under 5 years	19.3%	12.9%
5 - 14 "	27.0	18.0
15 - 45 "	37.9	53.4
Over 45 "	15.8	15.7

Almost 50% of the full blood Aboriginals are under 15 years old despite an astronomical infant and early childhood mortality for as far back as records are available. This phenomenon is explained by a high birth rate of 45 live births per 1,000 Aboriginal population. The white N.T. birth rate is 26. The big differential occurs despite a smaller percentage of the Aboriginal population being of the reproductive age, although most aboriginal women in this age range in fact do marry and reproduce which is not so of the whites. If the childhood mortality continues to fall and even if, which is likely, it does not fall below, say, two or three times the white Australian level for many years, the full blood population will increase very rapidly.

At the present rate of increase, of three per cent per annum, a population of 8,500 will double in 24 years and treble in 38 years, and, if the same rate applies

to all the full bloods in Australia, in 100 years there might be three quarters of a million Australian Aboriginals.

Some attempts at family limitation, mainly by means of the Intra Uterine Contraceptive Device have been made, but so far these attempts have had limited success. Obviously before such a programme can be made acceptable the need for limitation of family size must be felt by the Aboriginal and the concept of contraception carefully elucidated. It has been found in other primitive groups, with birth rates as high as that in our Aboriginals, that well organised birth control schemes have lowered the birth rate by 30% or more.

TABLE I.2 Effects of Birth Control Schemes in Other Countries.

	LIVE BIRTHS PER 1,000 OF POPULATION	
	1956	1966
Taiwan	44.8	32.4
Singapore	48.2	29.8
Hongkong	39.7	24.9
Puerto Rico	34.8	28.3

Table I.2 shows the degrees of success of some family planning endeavours in other countries. The 1956 figures are prior to introduction of the schemes, and in 1966 they

had been in progress for several years. (Victor-Bostrom Fund report, 1968).

Distribution of The Sexes

Since the first of January 1965, 363 full blood Aboriginal babies have been born in the Alice Springs Hospital. Of these 182 were male and 181 were female.

At the time of the 1966 Census, for the entire Northern Territory Aboriginal population, the Male : Female ratio was 1.024 : 1. (N.T. Statistical Summary, 1968). It is interesting that more male than female children have been admitted to the Alice Springs Hospital during the last four years, the ratio being 5:4 in favour of boys, whereas, the deaths in this group over the same period favoured girls by 8:7.

ADMINISTRATION AND MEDICAL SERVICES

The situation in the Northern Territory is rather complex. The Welfare Branch of the N.T. Administration is directly responsible for provision of facilities such as housing, water supply and sanitation on Settlements but only indirectly responsible for such things on Missions and Pastoral Properties. Although the Welfare Branch administers several hospitals on Settlements and employs nurses to staff them, there is no medical officer on the Welfare establishment. The Commonwealth Department of Health, through its Northern Territory Medical Service, provides

base hospitals in Darwin, Katherine, Tennant Creek and Alice Springs, and Rural Health services based in Darwin and Alice Springs. These services include routine aerial medical visits to outlying areas including Settlements, Missions and Cattle Stations. In the Alice Springs area this means that the places with relatively large populations such as Missions and Settlements are visited by an aerial medical officer every three weeks, and the smaller places on a six weekly basis if they request a visit. Besides the Aerial Medical Service there are other groups, one doctor and three sisters in Alice Springs and a larger group in Darwin, which undertake infant welfare and a variety of other tasks such as leprosy control, audiometry of school children, vaccinations, data collection for research projects, anthropometry and growth studies in the more remote areas.

Advice on medical matters can be sought by people in rural situations during fixed radio schedules each day. Evacuation to hospital of severely ill patients can be arranged in the same way.

The Northern Territory Medical Service carries over into the far North of S.A. but the Welfare Branch has no jurisdiction over the border, its task there being performed by the S.A. Department of Aboriginal Affairs.

This diversity of administrative control, especially

since Dept. of Health staff members are only in an advisory capacity when visiting Settlements etc., leads to many problems, which are related to childhood morbidity. Thus emphasis is often put on problems unrelated to community health, and suggestions made by health workers misinterpreted.

The situation has recently been further complicated by legislation giving Aboriginals full civil rights, including voting power, and an award wage. Whether or not these factors will lead to more rapid assimilation is hard to say, but they certainly have brought to bear pressures which again may have an unfortunate influence on morbidity.

Immunisation.

For some years it has been Health Department policy to immunise the entire Aboriginal population against Diphtheria, Whooping Cough and Tetanus, and also Poliomyelitis, according to the Commonwealth Serum Laboratories recommendations. Although a few children are missed the number is low and at the moment probably a higher proportion of the Aboriginals than of the white population are immunised against these diseases.

In 1968 the Sabin oral poliomyelitis vaccine was introduced and since then epidemics of measles and influenza have been aborted by using, on a wide scale,

"Koplivac" vaccine and a specific Influenza vaccine.

Florid cases of Tetanus, Whooping Cough and Diphtheria are rarely diagnosed.

The people are given Mantoux tests and periodically X-ray surveys are carried out. Tuberculosis is reasonably well under control, although perhaps half a dozen cases are notified each year.

WAY OF LIFE

The socioeconomic conditions under which the Central Australian Aboriginals live are uniformly very poor. The recently introduced award wage has meant that the Aboriginal has a much greater earning power than previously but so far there has been no apparent improvement in his overall situation, and in some ways there has been a deterioration.

Earning Power and Employment Opportunities.

There are very few skilled Aboriginals in this area, although many of those who have been brought up on Cattle Stations know the ways of stock and can sometimes find employment as stockmen. Others may have a smattering of mechanical knowledge, and a few have undergone more or less formal training as clerks, nurse aides, cooks and so on.

The bigger centres of population can use a fair bit

of unskilled labour in municipal duties, for example, on the hygiene squad, gardening, cleaning, driving, etc.

The situation, in most places, is one of economic starvation. There just is not enough adequate employment for the present work force, and there is nothing to suggest that this will improve as the work force increases rapidly. Jobs are manufactured in order to employ more but the result is to underline the economic gap. Several normally healthy and intelligent young men may spend every day raking a dusty area outside some public building, say, a canteen. Obviously this is the sort of employment, for which even the minimum "training allowance" of $25 per week, can only be offered with a Government subsidy. Private cattle station managers, not being able to afford the new wage scale have had no alternative, but to send many aboriginal families away to Settlements and Missions. This has resulted in increasing overcrowding on these bigger places, with all the attendent risks of crossinfection, exhaustion of facilities such as water supply, sanitation, housing, food supply etc. and the less obvious problems associated with the mixing of groups from different places and possibly with different codes of behaviour.

This economic gap brings to mind the words of J.F. Kennedy when he spoke in 1960 of a similar situation in Africa. "The gap in living standards and income level

and hope for the future .... presents our most critical challenge today, and it is to this gap we have responded most sporadically, most timidly and most inadequately". (Kretchmer, 1969).

The Government policy, particularly on Settlements, is to employ as many Aboriginals as possible. In some places up to 20% of the population is on the payroll and another few percent receive one or other type of pension. Child endowment and unemployment benefits also add to the increased spending power of the Aboriginals. Usually subsidised wages are provided for about one seventh of the population.

This discussion of economics may seem unrelated to childhood morbidity but as Virchow once said "medicine is as much a social as a biological science". In the present context this recent change in legislation has meant, that whereas, previously the Aboriginals were given food handouts and on most big places fed on the mess hall principle (this particularly applying to preschool and school children), now they are expected to buy their own food. This implies that they should have some knowledge of food values and, more especially, if infantile malnutrition is to be avoided, the principles of infant feeding and educational diets. Unfortunately this knowledge is lacking, and although appropriate types of food are often available

through Settlement canteens and Station stores etc. they are often passed over in favour of articles of little or no nutritional value such as soft drink, sweets and biscuits, because of ignorance.

Even if some of the people were by chance or training discerning shoppers, a minimum of $25 and maximum of $36 weekly would provide only a meagre menu. Very few European families could be expected to improve their circumstances on such a wage, especially in remote areas where the cost of living is very high.

Cooking, of course, is done on an open wood fire, with possibly two billy cans made from evaporated milk powder tins and holding about four pints of water, and one large dixie which might be used for making stew, washing clothes, carting water for cooking, washing and drinking and even as a chamber pot for an old man at night. A consideration of these facilities would limit the sorts of food purchased.

In the past, the ration issue of beef, flour, tea, sugar and tobacco has been supplemented with "bush tucker" in the form of certain seeds, yams or sweet potatoes, small reptiles and animals, kangaroo meat, insects and so on. It is even said that certain parts of lizards were given to weanling infants. This dietary supplementation still occurs but in large communities such as Papunya and Yuendumu which

have been in existance for a few years, not only is there practically no firewood for a considerable radius, but also very little "bush tucker" within a reasonable distance. Occasional Aboriginals who own motor vehicles and rifles may shoot a few kangaroos, but generally speaking the people are practically dependent on white man's foods. In a few situations, on Cattle Stations usually, where the Aboriginals have been left much to their own devices, they still depend very largely on food gathered from the surrounding country. It is interesting that the incidence of malnutrition in such places is as high or higher than anywhere else. Figures showing this phenomenon appear in the section on morbidity.

Tribal Breakdown and Cultural Blocks.

The extent to which the originally powerful tribal organisation has broken down varies widely in different places. The tribal elders, once supreme, appear to have lost some control over the young adults, but the formal initiation rites continue in several areas and corroborees still form a significant part in the life of many groups. The Pintupi and the Pitjantjatjara tribes have probably stayed more intact in this respect than the others.

This is only important from the health point of view in that various cultural blocks, often associated with

language barriers, make any sort of education programme exceedingly difficult. The gap between our scientific understanding of disease and the spiritual outlook of the Aboriginal is immense. It is quite probable in many simple situations that there is basic misunderstanding leading to misplaced harsh judgements. An example of this might occur when a child, who is dangerously malnourished by our standards, is active and happy, and therefore well, by Aboriginal standards. We want to evacuate this child to hospital, but his parents can comprehend no good reason for doing so. Likewise to keep a patient in hospital, or even on any sort of treatment, for a radiological lesion, creates difficulties in maintaining rapport, despite time consuming explanations and demonstrations. It is well known that children get better in hospital, having left the evil spirit behind, but relapse soon after homecoming, being once again in contact with the evil spirit.

The much vexed question of the importance of the tribal "witch doctor" or "medicine man" warrants some mention. These men still exist and practise in many places, amongst them surprisingly some of the more sophisticated areas.

Professor Elkin is prepared to believe that these men have supernormal powers, and can, or could, infact practise telepathy. His book "Aboriginal Men of High Degree" quotes

many well authenticated examples of clairvoyance, thought transference and prophesy.

Often a mother will take her ailing child to the witch doctor because she feels bound to do so and fears for her reputation, and may even fear retribution if she fails to go, but probably it is more likely that she has great respect for the medicine man's powers and consults him as a matter of course and from free choice.

In the Alice Springs area, over the last four years, about twenty children, ranging in age from a few days to twelve years have been seen with evidence of some sort of physical interference on them. The evidence has almost always taken the form of ecchymoses on the anterior chest near the xiphisternum or on the anterior abdominal wall. On one occasion in a neonate with meningitis they appeared over the bulging anterior fontanelle. These lesions are caused by suction with the mouth presumably in much the same way as "love bites" are produced.

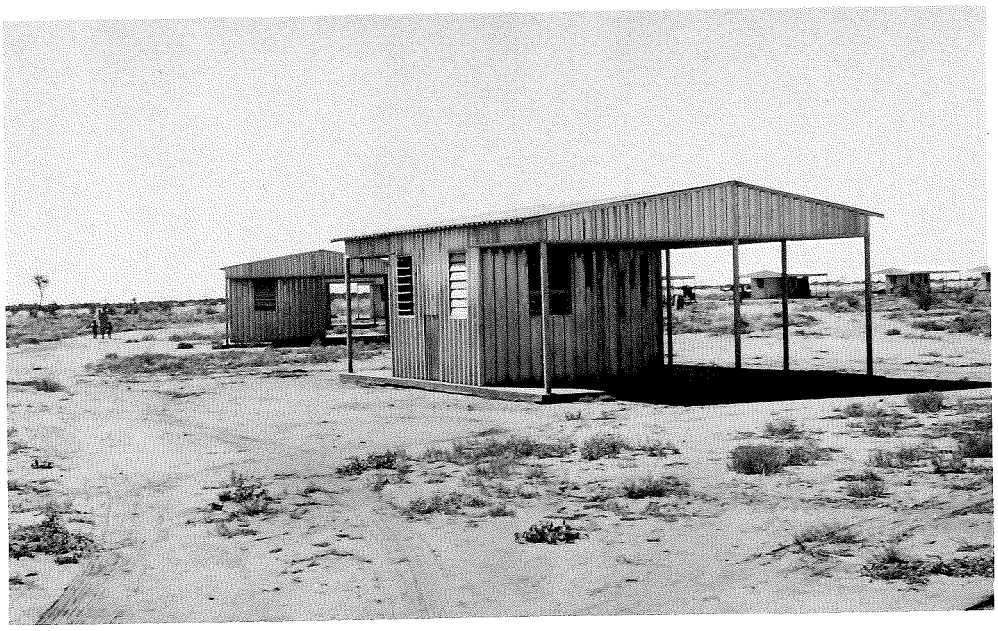
In fairness to the witch doctors responsible, it should be said that the patients with the marks on the thorax in fact had chest disease, and the abdominal bruises corresponded to gastroenteritis. On only one occasion was the distribution of the ecchymoses misleading. They appeared on the abdomen of Albert Namatjira's grandson who subsequently died of Staphylococcal pneumonia.

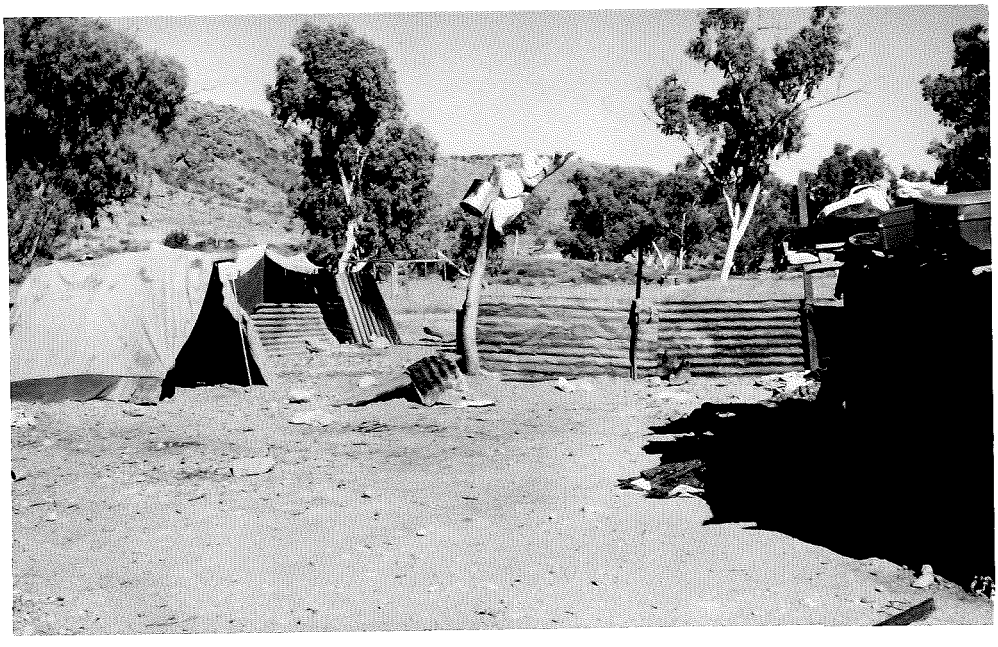
1. Humpy at Aileron Station, N.T.

2. Stone house at Hermannsburg Mission, N.T., with electricity and running water.

1. "Kingstrand" aluminium, single roomed, concrete floored house at Yuendumu settlement.

2. Camp area at Areyonga settlement. Note food and belongings out of the reach of dogs.





These ecchymoses had not caused any significant damage to deeper structures in the four cases autopsied.

The witch doctors are reputed to interfere with patients in other ways but no evidence thereof has been seen during this period under consideration. The main deleterious effect of such adherence to old customs is one of delayed presentation of desperately sick children to the sister in the Mission or Settlement Hospital, not uncommonly with fatal results.

Housing.

During their days as nomads the Aboriginals in Central Australia had no need for permanent or even relatively substantial dwellings. Very often a low, brush wind-break with a fire on the leeward side was sufficient. In the cold weather the structures were rather more sophisticated and had rooves. The average size varied a bit from group to group. Some would build a circular shelter or "wiltja", sometimes with a smoke hole in the centre of the roof, the diameter of the circle being from six to ten feet. Other groups favoured the lean-to principle with a central ridge, only three or four feet off the ground, and varying in length depending on the number of people to be housed. The overall floor area would rarely have been more than fifty or sixty square feet even for the largest families.

Some years ago the Government, in an attempt at re-housing, erected many dozens of stone, brick and later aluminium, single roomed, cement floored dwellings, some examples of which are shown in the photographs.

This took place mainly on the Settlements and still many of the people prefer to live in humpies. The reasons for this preference are fairly obvious when it is appreciated that the more formal type of dwelling usually contains no furniture, no fire place, no running water and is up to twenty degrees fahrenheit hotter in summer and twenty degrees colder in winter than the low lying humpy made of brush, old bits of galvanised iron, hessian and wire etc. which could be dismantled in toto and moved in a matter of a few minutes.

On two of the Missions stone and iron houses, often with two or three rooms, have been built, partly by the Aboriginals themselves, and occupied for many years. Reticulated water and electricity are currently in use at one place whereas in another, where the entire population of five hundred lives in humpies, there is one tap four hundred yards from the camp, serving everybody.

In many cases it is still the custom for a family group to move to another location if a member should die.

This makes permanent rehousing without re-education impossible.

So generally speaking the population, with a few exceptions, lives in grossly over-crowded, primitive conditions which fundamentally are quite similar to those seen in Glasgow in the late 1920's about the time of a slum rehousing programme. The failure of rehousing, without re-education, to lessen the respiratory infection rate is well demonstrated by Smith (1934) who followed approximately 1,000 people in a poor-class quarter and a similar number in a rehousing scheme in Glasgow. Of course the rehoused group had retained their slum habit of crowding into one bedroom at night and so continuing to cross infect each other. Thus the respiratory infection rate scarcely changed.

Almost all of the larger aggregations of Aboriginals, certainly on Settlements, have the use of communal ablution facilities and watering points. Some have no formal facilities for disposal of excreta, and therefore must move camp when the ground becomes fouled.

The water supplies on the various Settlements etc. are often of questionable safety for human consumption both from the bacteriological point of view and biochemically. Creek bed soaks, which are commonly used as watering points, after some weeks of use frequently contain uncountable

numbers of Coliform and B. Coli organisms per 100mls. This situation is the rule, not the exception.

One recent biochemical report on the water from a particular bore, around which some 200 Pintubis were living, states that the water is not suitable for domestic use because of excessively high Sulphate, Magnesium and Nitrate levels. Other reports from a variety of places indicate formidable levels of Potassium, Sodium, Calcium and Chloride.

Currently the two top priorities being considered by the Welfare Branch are water supply and housing. It would seem that educated motivation to want better housing and water supply might more reasonably be the primary aim.

Family Size.

This has been mentioned briefly before, mainly to show that, last century, the average family tended to be smaller than it is now. As Sir Frank Fenner said during the A.B.C. program "Insight" early in 1969, "no longer is the community limited by naturally occurring food and water", nor is it limited to the extent it was by the ravages of preventable and treatable diseases. Now instead of a woman expecting to lose at least one in every three children, she considers herself unlucky to lose one

at all. This change has been recent in Central Australia, certainly within the last five years, so it is pointless trying to assess average family size, since this is increasing all the time. In 1968 about 30% of families had five or more children living, and the probability of several more yet to come. There is just beginning the realisation by Aboriginal mothers that family size can be limited, artificially, and so far about 140 Intra Uterine Contraceptive Devices (of the Lippe's Loop type) have been inserted at the recipients' request. There has been a high failure rate and difficulty in following up cases, and as yet there has been no apparent effect on birth rate, as already indicated.

Apart from the obvious economic disadvantages of a large family there is a good deal of evidence to suggest that the infection rate amongst children rises with the number in the family. Dr. Smith's work in Glasgow in 1928 - '29 supports this, as does an investigation into the situation in Central Australia. This study is summarised, in the section on General Morbidity, under the heading, "Infection rates and Family Size".

EDUCATION

All the Settlements and Missions have schools as part of their establishment. In some places these schools achieve high attendance rates and have enthusiastic teachers and ample equipment. There are a few

Pre-schools in action and others planned. At the other end of the scale, in some areas, and even quite heavily populated ones, there are no schools at all, or at best, a single teacher with a caravan class-room. Some stations have schools and some do not.

Generally speaking probably 75% of children between six and twelve years of age undergo some basic schooling. The shortness of the school day and the length of school holidays mean that parental influence has every chance of making the school teachers' task extremely difficult, and often discouraging. Many problems are engendered because of poor teacher orientation, language barriers, rapid teacher turnover and isolation.

Many teachers, particularly in one or two teacher trailer schools in isolated areas, do much good work, and in some cases exceed their duty and take upon themselves responsibility for the health of the community and often even organise adult education classes.

As in most other things, pertaining to Aboriginals, the educational level is changing and any statement regarding it at this time may well be obsolete in five or ten years. There is much talk about adult education and health education, and within the next few years some broad, enlightened policies may be formed. At the moment attempts at adult education are sporadic and inadequate.

PHYSICAL ENVIRONMENT

The physical environment in rural Central Australia can be rigorous in the extreme. It is an arid zone and consequently the spectrum of disease seen is rather different to that further north in the Tropics.

Rainfall.

The average rainfall over the past seven or eight years has been about 8" per annum, but cannot be relied upon at any particular time of the year. For example, the rainfall at the Alice Springs Aerodrome in 1965 was 3.23" and in 1966 15.32". (N.T. Statistical Summary, 1968). One beneficial result of the dry climatic situation is that many pathogenic Parasites, such as hookworm, cannot complete their life cycle, because of desication, and consequently are only very rarely the cause of disease in this part of the country.

Temperature.

The temperature range from the hottest day in Summer to the coldest night in Winter may be over 100 degrees Fahrenheit. Quite often the ground temperature at night falls 10 degrees or more below freezing. Cold injury might be expected from the degree of frost in winter but has in fact rarely been diagnosed, except in premature babies who are more difficult to keep warm. An advantage of sleeping in a small enclosed space with several other

people, a few dogs and a fire is the warmth thereby engendered. It is possible however that cold air might be inhaled, penetrate sufficiently, and, aided by smoke from the ever-present fire, may cause partial or total inhibition of ciliary activity in the trachea and bronchi thus laying open the way for lung infection. This is an attractive theory especially in children with chronic purulent post-nasal discharge, but the incidence of severe lower tract respiratory infection is not very much higher in Winter than in Summer, except that in May and June, which corresponds to the onset of cold weather, there is usually a high incidence of laryngotracheobronchitis and bronchiolitis. The frequency of fluid losing diseases such as gastroenteritis and bronchiolitis amongst the Aboriginal children is excessively high, and when the ambient temperature is, say, 115 degrees Fahrenheit and the relative humidity below 20%, replacement of large volumes of fluid in a sick infant becomes well nigh impossible other than by the parenteral route. Dehydration, and consequent electrolytic imbalance, is a constant problem.

Dust.

It is common to see vast clouds of dust, mineral rather than proteinaceous and therefore probably non allergenic, covering whole areas. In the Summer, in dry years, these dust storms may be almost daily occurrences.

The relationships between dust and respiratory or eye infections and irritation are not known.

Flies.

During the warmer months flies, both bush and domestic or house flies, can be very numerous and certainly take a part in crossinfection. Several forms of conjunctivitis, significantly, are known locally as "fly eye" or "fly bite".

The Aboriginals tend to let flies crawl into their ears and eyes, and even noses, in great numbers without bothering to brush them away. The same trait is seen in white people who have been out of doors for long periods during the Summer. When working in the open often both hands are occupied and a fly-net is too hot so no alternative remains but to allow the flies free access.

An Aboriginal mother who does not bother about flies in her own eyes and ears is unlikely to worry about her baby's orifices. In some communities a very high percentage of children have discharging eyes and ears. No doubt flies help to make this a self perpetuating situation. Fly-blown ears, full of maggots, are not uncommon in preschool children. Quite recently a small epidemic of gonococcal conjunctivitis amongst children between six months and two years of age at a particular Station North of Alice Springs was almost certainly vectored by flies.

Professor Cleland (1966) mentions a similar outbreak in Central Australia.

Dogs.

Some controversy still exists as to whether the many hundreds of hungry and diseased dogs, invariably seen around an Aboriginal camp of any size, can be held responsible for disease. One dog at a Settlement, had reputedly been chained to a verandah post, living in its own excreta, for five years, at least, and certainly provided an extra attraction to flies. Dogs obviously keep their owners warm at night, but it seems in the past that not many of them assisted with hunting. The dogs keep the camp area free of food scraps, and probably keep at bay vermin, snakes and so on which may conceivably be harmful to the community. Several well authenticated reports of dogs consuming still born babies and, quite frequently, placentae, and an occasional much less reliable statement that dogs have interfered with and even killed and eaten babies born alive, have been heard from time to time. With the introduction of civil rights, and therefore civil responsibilities, for the Aboriginal, some legislation was promulgated that each Aboriginal could have only one unregistered dog. As a result many thousands of dogs were shot or poisoned and the wrath of many Aboriginals incurred. Despite this thinning of the dog population about two years ago, the numbers are very high.

During an upsurge in the number of cases of Shigellosis being reported, many of the dogs at Papunya Settlement were swabbed, but none proved to have pathogenic organisms in its rectum (Maxwell, 1967, personal communication). Thus the dogs have never really been inculpated, but it is difficult to believe that their skin sores, discharging eyes and small body parasites do not cause disease in humans, either directly or via flies.

Distance.

Remoteness has been mentioned previously, but its relationship to childhood mortality makes it worth stressing. This relationship has been found in other countries. In 1947, in China, Cheng found the infant mortality in the rural areas to be more than twice that amongst the urban people. Late presentation is one of the biggest problems facing the paediatrician in this community. It so often means that irreversible changes, say in the kidney or brain have occurred in the days, or even weeks, after the onset of the disease and before hospitalisation. Within a few weeks of each other, two children, one aged six months and the other three years were admitted to the Alice Springs Hospital with meningitis. Both patients came from remote areas and both had been unwell for two or three weeks before being admitted moribund. At autopsy the brains were porridge-like and thickly coated and

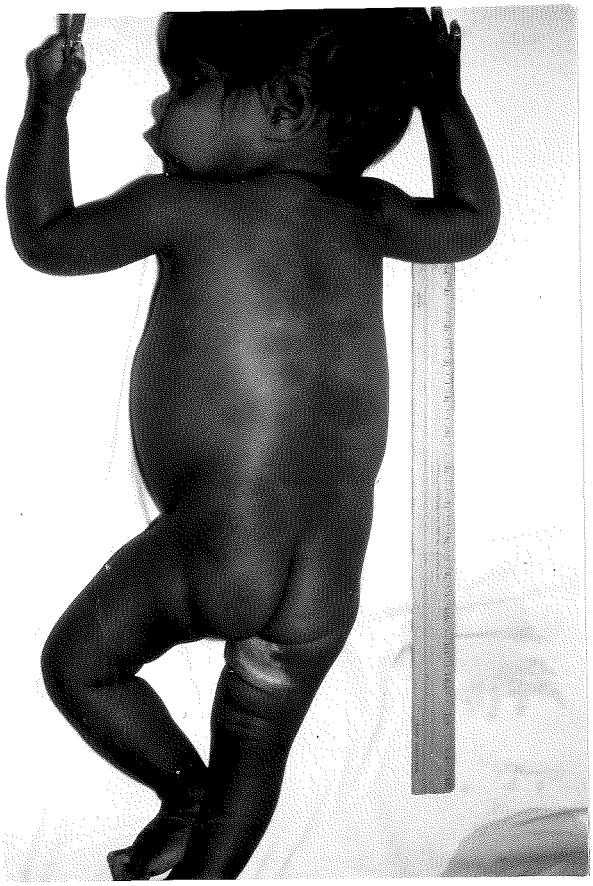
impregnated with an immense quantity of pus. This sort of story can be repeated over and over and whether the kidneys have failed and anuria supervened, or whether the bilateral, extensive, pyogenic pneumonia has destroyed whole lung lobes or whether brain damage has occurred it is usually because the individual arrived in hospital or sought medical advice too late.

Remoteness, of course, is not the only cause of late presentation. Another potent cause is failure to recognise disease in small children. It often happens, when an Aboriginal mother, who lives up to 400 miles from Alice Springs, presents her ailing baby to the Station manager's wife, that the incipient severity of the situation is not appreciated. Some Station manager's and resident owner's wives are trained nurses and others have acquired considerable experience in medical matters, but whether to call for an aerial evacuation, at great expense to the Royal Flying Doctor Service and the tax-payer, is a decision not to be taken lightly.

By the time the tribal medicine man has tried his remedies and the mother has convinced a white person, with wireless, that her baby is sick, and provided there is a serviceable airstrip nearby, or road transport available, even under the most fortunate of conditions one or two days might be forfeited.

1. Fair hair in a normal Pintupi child at Papunya Settlement, N.T.

2. Mongolian spots on a one year old Pitjantjatjara child from Indulkana Reserve, S.A.



During the 1968/69 year the R.F.D.S., Alice Springs section, evacuated 347 Aboriginal patients, a high proportion of them children, to the Alice Springs Hospital. A similar number were brought to hospital by Department of Health aircraft. In the same period there were 3,365 radio medical consultations regarding Aboriginals, again most of them, children. The Rev. John Flynn's "mantle of safety" has grown into an immense machine, but still Aboriginal children lose their lives because of remoteness.

GENETIC DIFFERENCES.

In July 1950, UNESCO made a statement regarding the subject of race. This statement, as expressed by Colin Simpson (1951) said :-

1. Racial discrimination has no biological basis.
2. There is no evidence that racial intermarriage produces biologically bad results.
3. The range of mental capacity in all races is much the same, and
4. Race is less a biological fact than a social myth.

What differences there might be between black and white Australians, apart from such things as skin colour, nasal configuration and blood group incidences, cannot be fully delineated until such time as the two groups have existed in identical environments for several generations.

This applies particularly to growth potential, both physical and mental. In common with other darkly pigmented races, Mongolian spots are commonly seen in Aboriginal infants.

With particular reference to high morbidity from recurrent infection a study of immune globulins has been undertaken and is reported in the section on Serum Proteins in Part III.

SUMMARY

The situation obtaining amongst the Central Australian Aboriginals is similar in most general respects to that in other socially depressed and technologically undeveloped racial groups. These primitive people, uneducated in the basic principles of static community life, live in what are usually grossly overcrowded and unhygienic conditions. They have no conception of contagious disease, believing still that evil spirits are responsible for sickness. Their earning power is low and their values immature, by our standards.

As with other such communities the birth rate is very high, which aggravates many of the other problems. The old cultures and social systems are breaking down, and not yet being replaced with anything worthwhile, thus causing a loss of identity and an aura of hopelessness.

Added to this is the dependence upon the white man for subsistence, since hunting grounds are given over to beef cattle and old skills are lost.

The children find themselves in an unenviable position where nutritional deficiencies and repeated infections are their lot.

PART II MORTALITY

In 1967, for the first time, Aboriginal infant mortality figures were included in the official Northern Territory Statistical Summary. By some chance the mortality during that year was the lowest ever recorded in the area. Despite this, the combined Aboriginal and white Australian infant mortality rate was more than three times as great as it had been the previous year for non-Aboriginals only.

Since this publication, but not altogether because of it, there has been a tremendous increase in the amount of published material relating to Aboriginal childrens' problems. Initially such articles appeared in Medical Journals, but the theme was quickly taken up by the lay press, and has consequently become quite contentious in the political world. A great many people are only too willing to argue vehemently, at the slightest provocation, about the rights of Aboriginals and their problems, without having any first-hand knowledge of the situation at all or being able to offer any concrete solutions. Since the topic of Aboriginal infant deaths has been so popular, these same infants have continued to die at the rate of one in ten. A death rate of this magnitude is totally unacceptable in a technologically highly developed country, and it clearly represents a gross infringement of the rights of Aboriginal mothers. Only two percent of non-Aboriginal Australian

infants die. Six years ago, in 1964, one in four Aboriginal babies born in Central Australia died before the age of twelve months, and one in three died before reaching two years. During 1966, at Papunya Settlement, forty-two babies were born alive, and, in that year, fourteen infants and toddlers died.

Death rates like these exceed by far the figures quoted for such places as Rural India and Basutoland, which are generally regarded as having extremely high infant mortality rates.

Since 1966 there has been, in Central Australia, considerable improvement in the infant and toddler death rates, but this has been due to the efforts of a few individuals, rather than a change in policy by any Government Department. Consequently it is quite possible that these astronomical mortalities are not yet things of the ugly past. Even with the recent improvement, the Aboriginal infant mortality rate is still as high as that of white Australians at the turn of last century, and is five times as great as the present figure for non-Aboriginal babies.

Most of the factors relating to the aetiology of high mortality will be discussed in the chapter on Morbidity. The sections concerned with Diarrhoeal disease, Respiratory infection and Malnutrition are of particular relevance,

since these three, singly, or in combination, are responsible for 80% of infant and toddler deaths.

Great difficulty has been experienced in obtaining accurate data regarding still births, prematurity rates and to a less extent neonatal deaths. It remains customary, in many areas, for Aboriginals to refuse to talk about unborn children and even to deny the presence of an obvious pregnancy. The Central Australian native is very reluctant to speak of the dead. Usually the name of a dead person is dropped from common usage and his dwelling-place vacated, sometimes for many months. It is no wonder that the women tend not to give their children aboriginal names until they are around two years old, and are therefore likely to survive.

Many Aboriginal babies are born in remote areas, with only native midwives in attendance, and may not be seen by a record keeper of any sort for many days or weeks. Thus it becomes impossible to discover, in some instances, if no child appears, whether a known pregnancy terminated in a still birth, or whether an early neonatal death occurred. Sometimes birth weight and age cannot even be estimated with any accuracy. Occasionally a birth and/or a death may be missed altogether, and consequently the data presented in the following section, especially those related to still births and neonatal mortality, are not claimed to

be entirely reliable. Some of the material appearing in this chapter was included in a paper, "Perinatal and Infant Mortality", read in Adelaide, during August, 1969, at a seminar on "Aboriginal Health".

PERINATAL MORTALITY

Perinatal Mortality Rate (P.M.R.) may be defined as the number of Still Births and Neonatal Deaths per 1,000 total births in the same period. The term "Neonatal" strictly means up to and including the 28th. day of life, but in this context it is broadly taken to mean the first month.

Records of still births prior to 1966 are so incomplete as to make them valueless, and Table II.1 includes only those data related to Perinatal Mortality from 1966 to 1969.

TABLE II.1 Perinatal Mortality (Aboriginal 1966-1969)

	1966	1967	1968	1969	TOTAL
Still Births	9	13	9	7	38
Neonatal Deaths	22	11	8	10	51
Total Births	320	333	363	356	1372
P.M.R.	97	72	46	48	65

Despite probable incompleteness of the data, it is fair to say that during these four years the Still Births have probably not varied very much. It is possible that

some of the deaths recorded, in 1966, as neonatal may have been Still Births, which would indicate a slight improvement in Still Birth rate, and little change in the neonatal mortality.

During the four years in question, an increasing proportion of Aboriginal women accepted antenatal care and were actually confined in hospital, so a trend towards lower Still Birth and Neonatal death rates could be expected. The Perinatal mortality shows such a trend, although the figures remain a good deal higher than the relevant white Australian ones. Table II:2 compares Aboriginal P.M.R. with that of the 1956 Australian level. All the figures are expressed in rates per 1,000 births.

TABLE II:2 Aboriginal and White Australian Perinatal Mortality Rates.

	ABORIG. (1966-69)	WHITE AUST. (1956)
Neonatal Death Rate	37	16
Still Birth Rate	28	15
P.M.R.	65	31

It is not surprising, having regard to the conditions surrounding the births of many Aboriginal babies, that Perinatal Mortality is at least twice that of their white counterparts. The logistic problems of providing adequate antenatal care in remote areas are enormous, even apart from the difficulties inherent in persuading Aboriginal

women to appreciate the need for and accept such a service.

NEONATAL, POSTNEONATAL AND INFANT MORTALITY

Neonatal mortality rate is the number of deaths of children in the first month of life per 1,000 live births in the same period, and postneonatal mortality is the number of deaths of children from one to eleven months of age per 1,000 live births in the same period. The sum of these two is the infant mortality rate (I.M.R.). The division at one month is important since often the main causes of death are different in the two groups.

Infant Mortality has long been accepted as a significant index of community health, both in the specific medical sense and within the broader meaning, including social and economic considerations. A falling infant mortality suggests that the community is becoming better educated to cope with the existing situation, or that living conditions, including medical facilities, in most cases, are improving.

It is the infantile deaths which have provoked the recent spate of newspaper articles, television documentaries and books, but in fact the post-neonatal and, as will be seen later, toddler mortalities are the biggest problems.

Table II.3 summarises Infant mortality, and its

components, for the years 1965 to 1969. The dramatic and sustained drop in deaths from 1967 onwards is clear.

TABLE II.3 Infant Mortality Rates (Aboriginal, 1965-1969).

	1965	1966	1967	1968	1969	TOTAL
Live Births	259	311	320	354	349	1593
Neonatal	39	71	34	22	29	35
Postneonatal	158	138	59	68	60	93
I.M.R.	197	209	93	90	89	128

Toddler mortality is part and parcel of the situation. It is mentioned here, briefly, so that more general discussion later can include both infants and one year olds.

TODDLER MORTALITY

Toddler, or Secotrant, Mortality Rate is the number of one year old children dying per 1,000 of the one year old population at risk in the middle of the same period.

Generally, in a sophisticated society, more deaths occur in the first month of life than during the rest of childhood put together. In the Aboriginals this adage is far from being true, and the main difference between Aboriginal and non-Aboriginal childhood deaths is in the toddler age group. Table II.4 shows the toddler deaths in Aboriginals during 1965-1969 and toddler mortality rates (T.M.R.) have been calculated using estimated mid-year populations at risk. Big fluctuations occur from year to

but the same overall improvement is seen here as in the I.M.R. It might be expected that a high I.M.R. one year would be followed by a low T.M.R. the next year. In other words, if weak babies die in large numbers in infancy then there might be relatively less weak one-year-olds to die during the next year. No such phenomenon is consistently apparent from the data, although the huge infant wastage in 1966 was followed by the lowest ever toddler death rate in 1967. An almost equally high Infant Mortality in 1965 was, however, succeeded in 1966 by the highest T.M.R. during the study period.

TABLE II.4 Toddler Mortality (Aboriginal, 1965-1969)

	1965	1966	1967	1968	1969	TOTAL
Population at Risk	200	195	245	286	320	1246
Deaths (12-23 Mos.)	14	26	3	9	7	59
T.M.R.	70	133	12	31	22	47

The fall in toddler deaths in 1967 was relatively of greater magnitude than the corresponding improvement in infant mortality at the same time.

COMPARATIVE MORTALITIES

Table II.5 puts the Aboriginal Mortalities, in the various age groups, into perspective with non-Aboriginal figures.

TABLE II.5 Aboriginal and non-Aboriginal Australian Infant and Toddler Mortality Rates

	ABORIGINAL (1965-1969)	WHITE AUSTRALIAN (1966)
Neonatal	35	13
Postneonatal	93	5
Infant (I.M.R.)	128	18
Toddler (T.M.R.)	47	2

The differences between these two sets of death rates are colossal. The differentials range from three-fold in neonates to twenty three-fold in toddlers! Numerically, of course, more Aboriginal infants, in the post-neonatal group, die than do children at any other age.

It is of interest to compare these infant mortality figures with those recorded for other groups living under less than adequate conditions. Table II.6 lists some of these, and it is clear that the Central Australian Aboriginal is indeed in an unfortunate situation.

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TABLE II.6 Infant Mortality Rates in Other Communities

		I.M.R.
Basutoland	(1955-1960)	181
Rural India	(1958-1959)	146
U.S. Indian	(1959)	57
N.Z. Maori	(1958)	53
Australia	(1901-1905)	97
N.T. (White)	(1901-1905)	149

These data were culled from Demography (1966) and Moodie (1969). Moodie raised the question that mortality rates were different in various places in the Northern Territory. He pointed out that less infants died on Missions than on Settlements and the present investigations support this claim. The mortality data so far presented has been restricted to the Southern Region of the Northern Territory, but, since this area contains only two rural Missions, for the purposes of the following discussion the far North of South Australia has been included. The places concerned, namely Ernabella, Amata and Indulkana, have precisely the same medical services and population types as other areas further to the North.

GEOGRAPHICAL VARIATIONS IN MORTALITY

Experience gained in the acute paediatric wards at Alice Springs, while providing a radio medical service

and during frequent visits to remote areas leaves no room for doubting that some places have higher morbidity and mortality than others. This phenomenon will be cited again in the discussion of various morbid conditions, and occurs even in separate cohorts of a single tribal group living under similar environmental conditions. Thus it is tempting to suspect that the relative adequacy of the medical attention provided at the various places may be largely culpable.

Table II.7 demonstrates the variable mortality between Church Missions, Cattle Stations and Government Settlements or Reserves. These figures support the general statement that less infants die on Missions, but on further splitting the data it appears that considerable variation occurs within each group.

TABLE II.7 Mortality on Missions, Settlements & Stations (1965-1969)

	LIVE BIRTHS	INFANT DEATHS	TODDLER DEATHS	I.M.R.
Missions	350	29	7	83
Settlements	872	129	27	148
Stations	508	74	30	146

It is abundantly clear that relatively many more one year olds from Stations perish than from Settlements, and more from Settlements than from Missions. The toddler

mortality rates for these three domiciliary groups are 75, 35 and 25 respectively. Thus Stations, which have, collectively, an I.M.R. as great as that on Settlements and a T.M.R. more than twice as high, represent the worst area in respect of early childhood deaths.

In Subsequent sections it will be explained that most Stations do not employ a trained nurse. The nursing sisters on Settlements gain a wealth of experience in handling children with diarrhoea and respiratory infection, but generally do not fully understand the problems associated with malnutrition and its diagnosis. These nurses unfortunately tend not to remain in remote areas for long periods. The sort of knowledge they gain is difficult to pass on, even if their successors arrive in time for a "hand-over", which is not common experience. Thus there may be a few regrettable occurrences while new staff learns the trade.

Probably the most tangible feature, affecting health, which distinguishes Missions from other places is the length of stay by staff members. Many nurses on Missions are virtually permanent, and consequently a good rapport builds up between them and their potential patients. They become experienced diagnosticians, especially with regard to ailing children, and assimmilate therapeutic skills from experience and from the rather informal and sporadic teaching beginning to be provided by the N.T.M.S.

There are other considerations which affect the relative healthiness of various communities. The absence of nocturnal frosts, for example, at Warrabri may lower the respiratory infection rate, and the close proximity of Amoonguna to Alice Springs allows for easy access to the base hospital facilities, but also to sources of alcohol, Veneraal Disease and other problems less common in remote areas.

Some evidence for the efficacy of a permanent, trained nurse in lowering infant mortality is shown in Table II.8. Four Stations with a resident nurse and four without have been compared. The eight stations are in the same general area North and North-West of Alice Springs and are populated basically by Aboriginals of identical tribal backgrounds. The doubled mortality in the absence of a nurse is clearly significant.

TABLE II.8 Mortality on Stations with and without Trained Nurses. (1965-1969)

	LIVE BIRTHS	INFANT DEATHS	I.M.R.
With Nurse	88	8	91
Without Nurse	93	17	183

Quite marked falls in I.M.R. occurred on some Settlements, during the five years studied, with the advent of long-stay nursing staff as distinct from those staying for only a few weeks or months.

Although mortality is lowest on Missions and highest on Stations, as has been mentioned, there are wide fluctuations within the broad groups. Table II.9 demonstrates this point further by showing the infant mortality rates for several Settlements and two Missions. This point has already been made with reference to Stations in Table II.8.

TABLE II.9 Mortalities on Individual Settlements and Missions (1965-1969)

	LIVE BIRTHS	INFANT DEATHS	TODDLER DEATHS	I.M.R.
Hermannsburg	135	7	2	52
Santa Teresa	103	14	1	136
Amoonguna	80	11	0	138
Areyonga	80	15	1	188
Jay Creek	44	2	2	45
Papunya	217	33	14	152
Warrabri	147	16	4	109
Yuendumu	166	26	4	157

Not only does the infant mortality vary but, to a less degree, so does the number of toddler deaths, probably for the same reasons.

INCIDENCE OF DEATH IN AGE GROUPS

The differences in mortality rates between Aboriginal and White Australian infants and toddlers are generally enormous. The proportions of childhood deaths occurring in various age periods are also very different in the Aboriginals. Table II.5 shows the difference in mortality rates, but the point is better made when they are expressed as percentages of the total number of deaths. Comparison of the mortalities in this way points to the post-neonatal infants and toddlers as needing the greatest attention from health workers.

The percentage distribution of infant and toddler deaths in age groups is shown in Table II.10.

TABLE II.10 Deaths in Age Groups as Percentages (1964-69)

	ABORIGINAL (1964-69)	NON-ABORIGINAL (1966)
Neonatal	21%	66%
1-5 months	29%	25%
6-11 months	27%	
12-17 months	17%	9%
18-23 months	6%	

During the last several years there has been as apparent swing in peak mortality towards the younger Aboriginal infants. This trend is not because more of them are

dying, but rather that much fewer of the six to seventeen month olds perish. In order to show this feature more clearly, recorded deaths from 1964 (N.T.M.S. records) are included in Table II.11. Some improvement in the first six months is seen, but it is in the older groups that the biggest change is apparent, particularly from 1967 onwards.

TABLE II.11 Deaths in Age Groups (1964-1969)

	1964	1965	1966	1967	1968	1969
Neonatal deaths	15	10	22	11	8	10
1-5 months	22	21	26	8	10	15
6-11 months	29	20	17	11	14	6
12-17 months	17	10	19	3	7	5
18-23 months	4	4	7	-	2	2

The reasons for the extreme wastage of life amongst the one to seventeen month children are the same as those making various morbid conditions common, and consequently will be discussed fully in Part III with the appropriate diseases. Suffice it is to say at this stage that there are no weird genetic or physiological reasons why so many Aboriginal infants and toddlers succumb. It is rather socio-economic poverty, illiteracy, lack of communication and malnutrition which potentiate common infections of the gut and lungs with lethal results. These issues can never appear in a statement of "medical" causes of death

such as appears in the subsequent section, but are none-the-less highly significant.

MEDICAL CAUSES OF DEATH

Several diagnoses, albeit often interrelated ones, usually appear on medical certificates of cause-of-death, and it can be difficult to decide which one is primary. In cases where two causes cannot be allotted in order of importance they must both be included, so that more causes of death are recorded than actual deaths.

During the period under consideration 268 deaths occurred in the infant and toddler age group and although a cause, or causes, of death are known for most of them, a few, who died in remote areas, were buried, with Coronial permission, without enough history being recorded for a retrospective diagnosis to be made. This circumstance was not uncommon in relation to early neonatal deaths. Table II.12 outlines the medical causes of death over the last five years. Malnutrition is included in cases where it clearly had a potentiating role in some other disease. No children were seen who died from pure starvation.

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TABLE II.12 Medical Causes of Infant and Toddler Deaths (1965-1969)

	NEONATAL	1-5 MOS.	6-11 MOS.	12-23 MOS.	0-23 MOS.
Respiratory Infections (B30, B31, B32)	17	45	34	23	119
Diarrhoeal Disease (B6, B36)	4	29	31	32	96
Other Infections (B10, B14, B17, B23, B43)	5	9	10	9	33
Congenital Defects & Immaturity (B41,B42,B44)	32	4	-	-	36
Malnutrition (B46)	2	9	23	27	61
Other Diseases (B18,B46)	8	5	3	2	18

The code numbers are taken from the abbreviated list of 50 causes based on the 1955 revision of the International Statistical classification of causes of death (Demography 1966). Malnutrition, despite its prevalence in two-thirds

of the worlds' population, was not accorded a number of its own in this list. Thus, for the purpose of this discussion, Malnutrition has been split off the last subdivision of B46, namely "all other diseases", and given its rightful position as a separate heading.

When it is considered that Malnutrition only occurred in association with another illness, usually Respiratory infection or Diarrhoea, the overwhelming predominence of deaths from infective diseases, in all age groups except neonates, is spectacular. In the entire group over 80% of deaths were caused by an infective illness with or without Malnutrition. If the neonatal deaths are excluded this figure rises to 95%. The vast majority of infective illnesses are either of the Respiratory or Alimentary tracts and, in the post-neonatal Infants and Secotrants, 83% of deaths are caused by one or other of these two. Diarrhoeal disease and gut infection are being used synonymously in this section. The slight inaccuracy of this assumption is discussed fully in the section on Diarrhoea.

The situation regarding neonatal deaths appears unlike that in other age groups because of immaturity and other diseases peculiar to the newborn period. Such conditions as severe congenital malformations, birth injuries, postnatal asphyxia, atelectasis, respiratory distress syndrome as well as prematurity, tend to mask the relatively high

incidence of death from infective disease.

The disease subdivisions, except that of immaturity, are taken individually and described in detail under Morbidity.

PREMATURITY

Mean birth weight shows great variation from place to place. This occurs even amongst separate communities of similar tribal background. For example the full-blood Aranta babies at Hermannsburg have an average birth weight of 3.5Kg., whereas at Santa Teresa, also amongst Aranta people, the figure is below 3Kg. Warrabri and Papunya have mean birth weights of 2.9 and 2.7Kg. respectively. This phenomenon suggests that any attempt to assess prematurity rates, based on the arbitrary weight of 2.5Kg., would be almost meaningless. According to this criterion 21% of live aboriginal babies born during 1965-1969 were of low birth weight. In this period, 1,593 live births were recorded, and reliable birth weights are available for 1,387 of which 291 were less than 2.5Kg. The incidence of low birth weight in non-Aboriginal Australians is around 5%.

Many of the low birth weight Aboriginal babies were in fact, mature in that, weight aside, they had none of the physical signs or behaviour patterns associated with

prematurity. Only 21 of the low birth weight babies could be considered to have died from immaturity, although, like malnutrition in the older children, it may have potentiated infections which led to further deaths in this group. Thus at least 34% of neonatal deaths were due to prematurity, but only 7% of the low birth weight babies in fact died in the first postnatal month.

It is significant that the highest incidences of low birth weight occur at the places which also have high mortality and morbidity. Clearly poor environment has a great deal to do with the production of these high rates. Table II.13 shows the birth weights for the places mentioned above in relation to their I.M.R. over the five year period.

TABLE II.13 Birth Weight related to Mortality Rates (1965-1969)

	MEAN BIRTH WEIGHT (Kg.)	I.M.R.
Hermannsburg	3.5	52
Santa Teresa	3.0	136
Warrabri	2.9	109
Papunya	2.7	152

Very little can be said of Still births since adequate data have been impossible to obtain. The figures currently available for the period under consideration suggest a

rate of something well over 20 Still births per 1,000 births. The non-Aboriginal figure in 1955 was 16 per 1,000.

The factors responsible for production of high numbers of Still births and low birth weight infants are likely to be identical, and include absence of antenatal care, maternal malnutrition and so on.

MORTALITY AND MALNUTRITION

It is clear that an enormous number of Aboriginal children die from apparently cureable, if not preventable, illnesses. The potentiating action of malnutrition on these infective diseases is all important, and its significance in Central Australia is discussed fully elsewhere.

A recent World Health Organisation publication (Scrimshaw, Taylor and Gordon, 1968) summarises present overseas knowledge on the interaction of nutrition and infection. There are some aspects of the problem which are not mentioned, and may indeed be peculiar to Australia.

Health workers, trained to practise in situations where malnutrition is expected to be a problem, no doubt know well that the severity of even the simplest illness may be increased, almost beyond recognition, in the

presence of many nutritional disorders. The treatment of many common conditions, such as infantile diarrhoea, becomes almost unbelievably complex and difficult. Unless these difficulties are well understood the simple diseases can easily be fatal ones. Medical training in Australia has lacked adequate reference to early childhood malnutrition and its attendant connotations, with the possible exception of a few specific deficiencies such as Rickets and Scurvy. Consequently doctors and many nurses, in Central Australia lack what should be prerequisite knowledge in this vital field.

Aggravation of this situation becomes apparent when ratios of staff to patients in hospital are considered. Although this precise problem is not directly related to malnutrition it is connected with mortality and bears mention at this point. During 1969, for several consecutive months, one dedicated medical officer was responsible for the overall inpatient care of over 100 paediatric patients. Obviously this is quite ludicrous, no matter how experienced and well trained the doctor is, when consideration is given to the monumental problems involved in treating severely ill, malnourished infants and toddlers. At the Adelaide Children's Hospital (Inc.) there is one Resident Medical Officer or Registrar to every 10 to 12 medical inpatients. In addition, of course, there are many visiting medical staff to give

advice and moral support, and myriads of paramedical people, such as physiotherapists, laboratory technicians and radiographers, as well as specialists to report on X-rays, pathological specimens, E.C.G.'s etc. Such facilities are either non-existant or totally inadequate in Alice Springs.

Prior to 1967 there had never been a medical officer qualified or experienced in paediatrics in the Alice Springs Hospital. Undoubtedly the biggest single reason why infant mortality fell so abruptly in 1967, was the advent of such a man to take control of inpatient care.

In order that hospital paediatric inpatient mortality might continue to fall several possible courses of action arise. They include provision of more medical and nursing staff, trained in the care of malnourished children, up-grading of the paramedical facilities and a very extensive rural health programme to involve case-finding, education of mothers with respect to basic hygiene and infant nutrition, and teaching of people, living in remote areas, to recognise serious illnesses, such as Malnutrition, early and thus prevent the problems of late presentation.

FALLING MORTALITY

The infant mortality in 1969 was one third of the

1964 level, and although in the period between those years, there were fluctuations, there have been marked falls in neonatal, post-neonatal and toddler mortalities. The main reason for this fall was the presence of a competent paediatrician in the Alice Springs Hospital but other factors contributed.

During the last five years there has developed an awareness by both medical and lay people, of the pressing problems such as treatment of diarrhoea and pneumonia. An expectation of higher standards of medical care appeared in rural areas due to more regular and frequent visits by public health sisters and medical officers. These visits clearly had a direct beneficial effect on mortality, as well as the indirect one mentioned.

A direct benefit from investigations made for inclusion in this thesis arose from the realisation of the importance of Malnutrition. Inpatient and outpatient care, and preventive measures in rural areas improved with the increasing knowledge regarding particular aspects of the local syndrome provided by some of the studies reported in this paper.

Some of the pre-Listerian practices common on Settlements were replaced by more hygienic methods. For example, the introduction of "Milton" antiseptic for sterilising babies' bottles and other feeding equipment

coincided with a dramatic fall in mortality at both Yuendumu and Papunya. Unfortunately the very nature of nurses' work in isolated places tends to produce low standards and, unless adequate support can be offered, it is only those women of exceptionally high moral fibre and ability who can maintain the standards of their training schools.

Increasingly frequent advisory visits to remote areas by a rural health medical officer and a trained health inspector, as well as the teams of public health nurses, have probably had a gradual effect on the overall picture, which is difficult to measure. Another possibility is that the end of an eight year drought, in 1966, brought about, in 1967, more abundant natural food, less dust and better morale which could all, conceivably, have a salutory effect on mortality. Figure II.1 summarises I.M.R. trends over the study period.

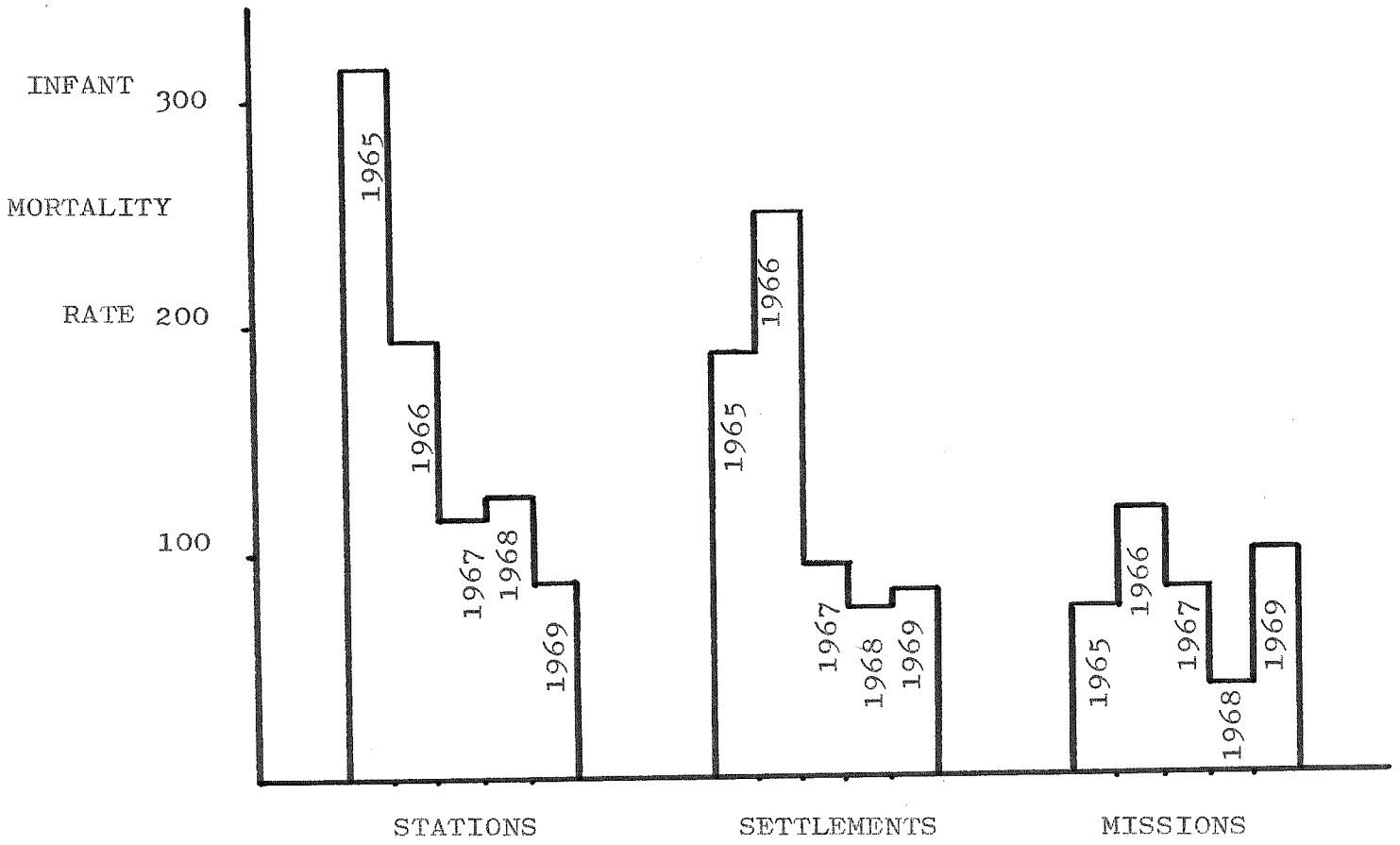

FIGURE II:1 Annual Infant Mortality Rates on Stations, Settlements and Missions.

PART III MORBIDITY

The recent fall in Aboriginal Infant and Toddler mortality means that morbidity rather than mortality, has become the index of public health in this group. The statistical services related to the study of morbidity in Central Australia are quite primitive, although a recently introduced system in Health Department hospitals will enable many data to be stored on computer cards and consequently, in years to come, be used to investigate disease trends and so on. There is still, however, a great mass of relatively minor but highly significant illness, dealt with by nursing staff and others in the rural areas, which is never recorded on the hospital system.

Even communicable disease is only sporadically reported, and it is these diseases which are responsible, in large part, for the high morbidity, and incidentally, mortality. In the technically highly developed parts of Australia the information services are changing their emphasis from the once common communicable diseases to such things as cancer, cardiovascular and renal diseas, and rheumatic problems. The Aboriginals in Central Australia are in an underdeveloped situation as evidenced by their high sickness and death rates as well as the economic factors discussed in the first part of this thesis.

Consequently it is the disease pattern affecting developing groups that is of primary importance at present, although the more sophisticated illnesses are beginning to be apparent among these people.

The data related to morbidity in the following pages, for the reasons suggested, are not claimed to be strictly accurate, especially those applicable to patients not hospitalised. Epidemics of such diseases as measles and gastroenteritis cause yearly morbidity statistics to fluctuate quite widely. During October 1968, over 40 cases of diarrhoeal disease from three places were hospitalised in Alice Springs, whereas, during 1966, there were many admissions due to measles and its sequelae.

In some areas where little or no local medical service exists much of the sickness amongst children goes unseen and unrecorded. Even where there are adequate nursing staff and hospitals, whether a mother will present her ailing child or not will depend on many factors. When the records are relatively well kept a good deal depends on the individual nurse as to diagnosis and classification. In view of these considerations, the comments on morbidity in rural areas will of necessity be general.

Throughout the ensuing discussion the statistics for the Aboriginal children where feasible will be compared with those for Europeans living in the same area.

The total population in this area is roughly the same for both groups, making for some interesting comparisons.

The attitudes towards active case finding vary enormously. Some bush nurses feel it to be their duty to go into camps looking for sick children and others believe that responsibility for presentation of patients rests with the mothers. A few of the nurses in charge of bush hospitals and medical rooms are prepared to treat quite seriously ill patients in their own environment with or without admission to the local "ward", asking daily for medical advice over the radio. Others prefer to have their patients evacuated as soon as there is the slightest suggestion of a dangerous illness. Recently one very competent nursing sister on a Mission treated at home, quite successfully, a 15 month old child with severe gastroenteritis and dehydration. Evacuation was impossible for at least 18 hours after the initial radio report and the baby's life was undoubtedly saved using the technique of repeated intraperitoneal Saline infusions.

Quite probably many lower respiratory tract infections, the extent and severity of which not being appreciated, are aborted by administration of parenteral antibiotics (usually Penicillin G.) and evacuation to hospital does not eventuate, although there may be some residual lung lesion which could produce recurrent and eventually

chronic disease.

The section following is a description of the sort of medical conditions confronting the nursing staff, and others, in rural areas and briefly the types of treatment available in those situations.

MORBIDITY IN RURAL AREAS

Up to 25% of the pre-school group on some Settlements and Missions are seen by a nursing sister each day. In the bigger Settlements, 50 children may be seen, up to 20 of those being infants.

Many of the children are seen routinely for treatment of chronic suppurative otitis media, impetigo, minor traumata and so on. In general it is infections of the upper and lower respiratory tract and bowel that produce most of the morbidity. The infective nature of these diseases means that one of the biggest enemies is crossinfection. In a camp situation, and without facilities for isolation, the management of even a small outbreak of, say, gastroenteritis is extraordinarily difficult.

Trauma in under five year olds is relatively rare, and is, in any case, taken very lightly by their parents. It is, however, not uncommon to see several cases of burns at the beginning of Winter due to sleeping too

close to a fire at night.

Malnutrition is probably on a par with Respiratory and Bowel infection as a cause of morbidity. In its early stages malnutrition is a sign rather than a symptom and has to be looked for routinely. It's significance as a synergistic factor with common infectious diseases has only recently become widely appreciated in this country, and cannot be overstated. Now a good deal of effort on the part of bush nurses goes in keeping longitudinal weight records and conducting infant welfare or "well-baby" clinics.

One of the most difficult tasks facing nurses in remote areas is to pick from the many children with relatively mild disease the few who need special care and possibly evacuation to the base hospital. Many bush hospitals have neither facilities nor sufficient staff to cope with inpatients, and the difficulties in communication with parents make outpatient management of sick children a nerve wracking business. Consultation with doctors over two-way radios, or radio telephones, can relieve the bush nurse of some responsibility and give her moral support, although it is upon her observations that the doctor must rely in assessing appropriate courses of action. The nurse not only has paediatric problems such as she would rarely have encount-

ered during her training, but also the whole gamut of adult medicine ranging from complicated obstetrics to diabetic coma and occasionally severe injury. Her armamentarium of therapeutic agents is limited, although to some extent she has a choice which is vetted by Department of Health authorities.

The cattle stations usually have a "Flying Doctor Kit" containing very basic medicaments, which, depending on the population and the qualifications of the person using the kit, may be supplemented to broaden their application. The Missions and Settlements are allowed to keep a relatively extensive stock of drugs, since with several different medical officers giving advice over the radio and prescribing treatment, even for similar illnesses different drugs are required. In fact the inconsistency of advice given over the radio is often confusing to people in the bush, many of whom naturally prefer to treat patients according to fixed rules of thumb.

In the paediatric field the most widely used drug of all is undoubtedly Penicillin "G". Other penicillins such as "V" and Benzathine are also commonly employed. Ampicillin has recently found favour in treating respiratory infections. Although it has been used for Shigellosis, its value is limited because of the emergence of resistant strains. Various brands of Tetracycline are available,

and in fact very widely used in adults, but their use in children under the age of seven years is not recommended.

Other medicaments in common usage in rural areas are Chloromycetin or Tetracycline eye ointment, Mycostatin suspension for monilial infection, Mepacrine for Giardiasis, a variety of solutions, powders and drops for chronic suppurative otitis media, decongestive nose drops, Mercurachrome and Gentian Violet for some skin lesions, several unctions such as "Furacin", "Centrimide" and "Ungvita" for others.

Occasionally there is need for a urinary antiseptic and now Naladixic Acid is most popular, although Sulphas and Nitrofurantoin are still used extensively.

Most of the so called antidiarrhoeal agents are not recommended for use in small children, and the tendency is to use a dietary regime according to an instruction sheet distributed by the N.T.M.S. Rural Health Section.

Benzyl Benzoate and D.D.T. emulsion are from time to time used in huge quantities for scabies and pediculosis capitis.

Antihistamines have a limited use in the age group under consideration, and usually the condition treated is the result of an insect bite.

The individual conditions making up the high morbidity rate are discussed separately later in this chapter.

MORBIDITY EXPRESSED BY HOSPITAL ADMISSIONS

In this section 770 consecutive admissions of pre-school age children to the acute Paediatric wards in the Alice Springs Hospital during 1968 are considered. Although figures for Aboriginal and European children are compared it must be borne in mind that the bulk of white patients are drawn from the urban areas whereas most of the Aboriginals are rural. It is also so that many of the Europeans can be treated as outpatients far sooner in the course of their disease than can the Aboriginals because of repatriation problems and differing environments.

Table III.1 shows the number of admissions during 1968 and relates them to the populations at risk.

TABLE III.1 Hospital Admissions (1968)

	ABORIGINAL	NON-ABORIGINAL
Approx. Population at risk (under 5 years)	1,600	1,200
Admissions	552	218

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The surprisingly high admission rate in the Europeans and Part-coloured group is accounted for largely by the tremendous incidence of respiratory infection in Alice Springs itself. This, in the urban community, leads to an elevated tonsillectomy and adenoidectomy rate, more febrile convulsions and so on. During 1968 Alice Springs had only one private medical practitioner and probably quite a few children presented, and were admitted, to hospital, who could have been managed at home by a visiting doctor.

Relative to the populations at risk about twice as many Aboriginals as European and part-coloured children were admitted, and roughly, on a day to day basis, 11½ times more beds were occupied by Aboriginals. The absolute daily bed occupancy for Aboriginals in 1968 was 43.9 and for the other group 3.8.

Since these figures were taken out, the average Aboriginal paediatric bed occupancy has steadily increased and is now (Dec. 1969) almost twice the 1968 mean. The reasons for this increase are manifold but rapidly increasing population and better case finding are high on the list as precipitating more admissions, and more experienced appreciation of the dangers of malnutrition often means longer hospitalisation. The more frequent diagnosis of chronic lung disease also is now

responsible for many admissions. One other factor of considerable importance is the improving rapport between hospital staff and Aboriginal parents. Previously, when so many children died, hospitalisation was regarded almost as a death sentence. Now that the mortality has fallen and hospital conditions have improved, parents are much more inclined to let their children be admitted and are often reasonably happy about long stays in hospital and even transfer to the Adelaide Children's Hospital, for more extensive medical attention.

Duration of Hospital Stay.

The time spent in hospital by Aboriginal children depends very largely on their nutritional status. Sometimes there are difficulties in repatriation to distant places and this may add a little to their hospital stay. The mean duration of hospitalisation for the Aboriginal children is 29 days and for the European and part-coloured group 6.4 days.

Table III.2 demonstrates the relationship between duration of stay in hospital and nutritional status.

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TABLE III.2 Duration of Hospital Stay and Nutritional Status (Aboriginal Children Under Five Years, 1968)

WEIGHT PERCENTILES	NO. OF CHILDREN	MEAN HOSPITAL STAY (Days)
Less than 3rd.	85 (53.1%)	39.9 (range 6-151)
3 - 24	40 (25%)	21.5 (" 3-56)
25 - 50	21 (13.1%)	11.5 (" 2-33)
More than 50	14 (8.8%)	9.3 (" 3-25)
TOTAL	160	29.0

It is clear that malnourished children spend more than twice the time in hospital than do their well nourished peers.

A comparison of durations of hospital stay for patients with diarrhoeal and respiratory disease each with and without malnutrition is theoretically not of great value since during a long stay in the acute paediatric wards any patient is very likely to have episodes of both respiratory infection and diarrhoea. However in Table III.3 fifty seven patients whose admission diagnosis was diarrhoea, without lung infection, and forty-nine with lung infection, and no diarrhoea, are considered and their weight percentiles related to the duration of hospital stay. These figures are significant.

TABLE III:3 Hospital Stay, Diarrhoea, Respiratory Infection and Malnutrition, (1966).

	NO. OF PATIENTS	PERCENTILE	HOSPITAL STAY
Diarrhoea	36	Less than 3	42.6
"	21	3 and over	14.2
Resp. Infect.	19	Less than 3	19.7
"	30	3 and over	11.5

Since these patients were admitted consecutively, except of course, for others with different diagnoses or Respiratory infection and diarrhoeal disease co-existing being interposed, it appears that there are considerable differences between the malnourished and "normal" groups.

During 1966 when these children were hospitalised, the diarrhoeal group spent much longer in hospital than those with respiratory disease. The mean stays for these two groups were 33.0 and 14.7 days respectively. More malnourished children than otherwise were admitted with diarrhoea, where-as the reverse was true of those with respiratory infection. Both malnourished groups stayed longer in hospital than the respective better nourished ones. This phenomenon was very much more marked in the children with diarrhoea. Over 60% of the patients admitted with diarrhoea and 40% of those with Respiratory

Infection were malnourished, and since only 16.5% of the population at risk are malnourished by the same criteria it is clear that both diseases are potentiated by malnutrition. This hypothesis is well supported by Scrimshaw, et al. (1968) and is discussed more fully in this chapter.

Age of Children Admitted to Hospital.

The incidence of hospitalisation amongst different age groups has changed slightly. During 1965 - 1967 the peak incidence was between nine and twelve months (Kirke, 1969) whereas in 1968 the peak was just over 12 months. This difference is possibly so small as not to be significant, but two factors which could explain it are that less infants are dying now and many of the survivors need hospitalisation during their second year, and also, with recent emphasis on infant feeding beginning to take effect, the onset of malnutrition is staved off for a few months.

Figure III:1 shows graphically the incidence of paediatric admissions by age. 19.6% of the Aboriginal admissions were under six months, 41.2% under 12 months and 65.8% under 18 months. These percentages are far higher than the comparable ones for the European and part-coloured community. Table III:4 shows how much difference there is between the two groups in rates of admission in the younger ages.

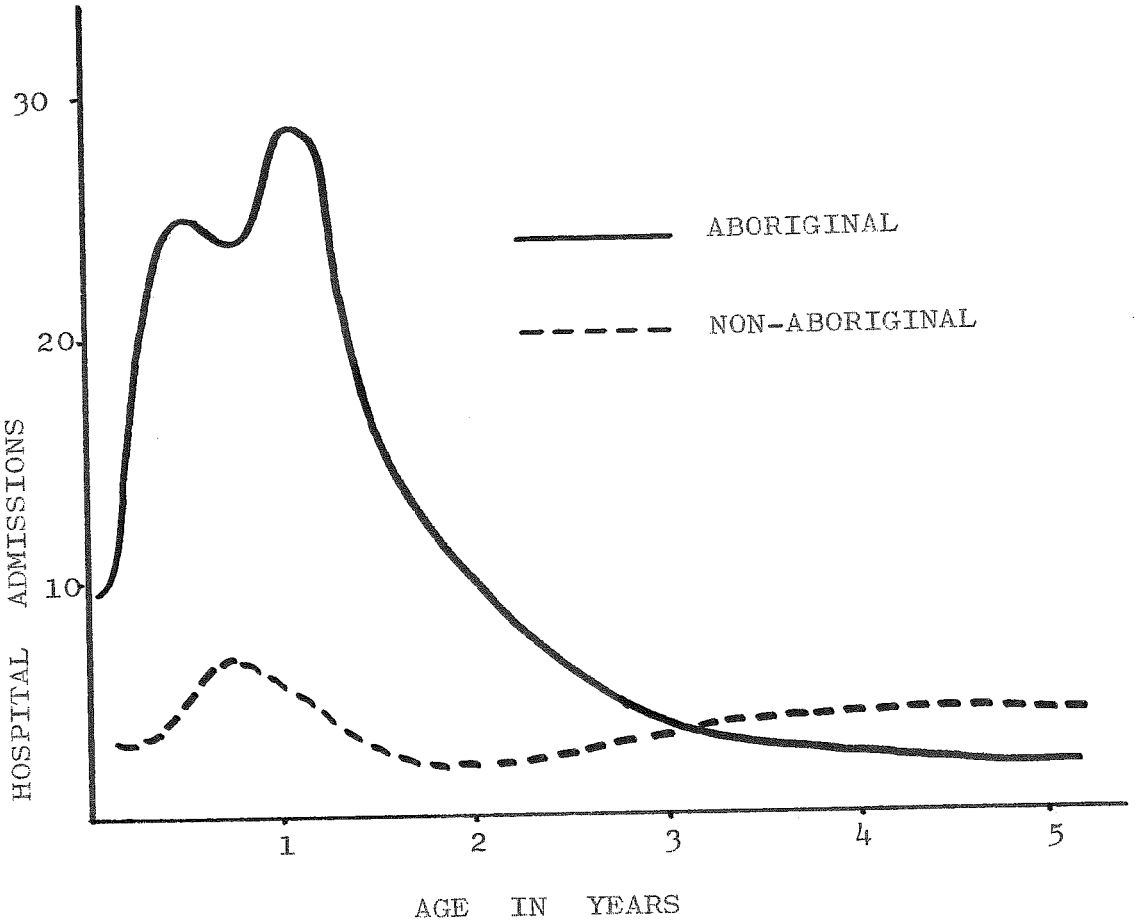


FIGURE III:1 Age distribution of paediatric admissions to the Alice Springs Hospital 1968.

TABLE III.4 Incidence of Admission by Age (1968)

	ABORIGINAL	OTHERS
Less than 6 months	19.6%	8.3%
6 - 11 months	21.6%	14.7%
12 - 17 months	24.6%	10.6%
18 - 23 months	10.1%	6.4%
2 - 3 years	11.0%	24.7%
3 - 4 years	6.9%	17.4%
4 - 5 years	6.2%	17.9%
TOTAL	552	218

Morbid Conditions Precipitating Hospitalisation.

The purpose of this section is to present briefly the incidence of the various illnesses, mainly in tabular form, and leave discussion of the individual diseases to the following sections.

TABLE III.5 shows the admission diagnoses, during 1968, of the same 770 patients mentioned previously. In some cases two co-incident conditions occurred and, if one could not be considered to take precedence over the other, both are included so that there are several more diagnoses than patients. For simplicity the number of cases of each illness is quoted as a percentage. In a similar way the disease pattern is presented for the various age groups

in figures III.6 to III.10. Although the primary object of this Thesis is to present the situation regarding Infant Morbidity and Mortality it is considered that morbidity in the whole pre-school group is worthy of comment.

TABLE III.5 Diagnosis on Admission to Hospital (1968) Under Five Years.

REASON FOR ADMISSION	ABORIGINAL	OTHERS
Diarrhoeal Disease	42.8%	25.4%
Respiratory Infection	25.0	35.2
Malnutrition	11.0	1.6
Other Infections	7.0	1.6
Trauma	4.0	9.6
Congenital	2.0	1.6
Social	1.2	0.8
Ingestion	1.0	4.0
Miscellaneous	6.0	20.2
TOTAL DIAGNOSES	584	254

In both racial groups the incidence of Respiratory Infection and Diarrhoea is very high. During October, 1968, 40 cases of Gastroenteritis were admitted as a result of epidemics on one Mission and two Settlements. Even without this sudden influx diarrhoea is still the most frequent cause of hospitalisation in the Aboriginals

whereas Respiratory Infection is more frequent in the white and part-coloured group.

The percentage of Aboriginals admitted for malnutrition is low compared to the number of inpatients found to have deviated significantly from a normal growth pattern. In fact over 50% were below the 3rd. percentile for weight in 1968, although only 11% were admitted because of malnutrition.

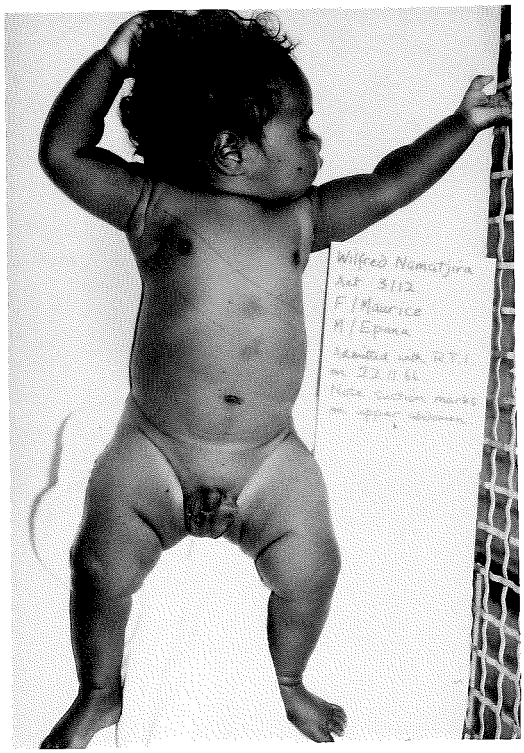
The heading "other Infections" includes meningitis, skin sepsis, abscesses, osteomyelitis, tetanus, conjunctivitis and so on. Trauma was largely made up of burns, foreign bodies and limb injuries, and the congenital anomalies consisted of talipes deformities, heart lesions, cleft palate and oculo-auriculo-vertebral dysplasia.

Admission for social reasons, such as incapacity of parents, awaiting interstate transfer or a breastfed baby admitted with its sick mother in order to continue breast feeding are fortunately uncommon. The range of toxic substances ingested can be quite wide since, in this area, many of the shrubs, such as Oleander, Bird-of-Paradise bush and some of the wild tomato varieties (Solanum Ellipticum), are quite toxic, and it being largely a pastoral area, materials such as kerosene, petrol, dieseline, turpentine, organic phosphate weed killers and many others are relativ-

1. Ecchymoses on upper abdomen produced by tribal medicine man.

2. Goldenhar's syndrome with hemifacial microsomia, conjunctival dermoid, orotragal raphe and a pre-auricular appendix.

3. One year old full-blood children, one normally grown and the other with marasmus.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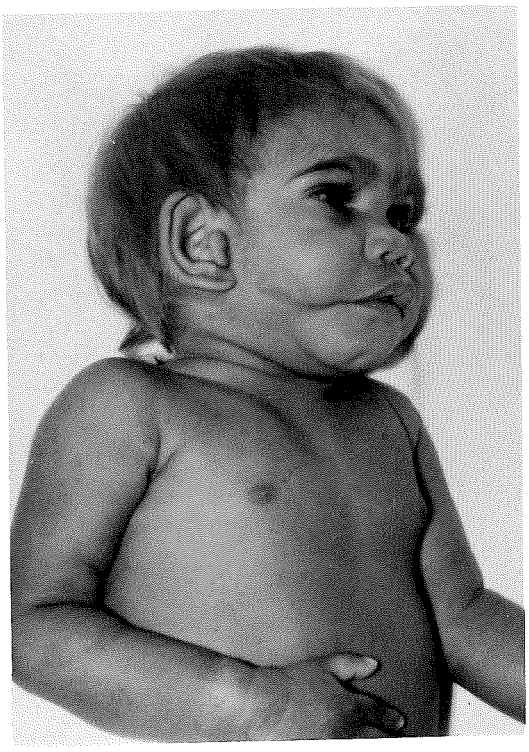

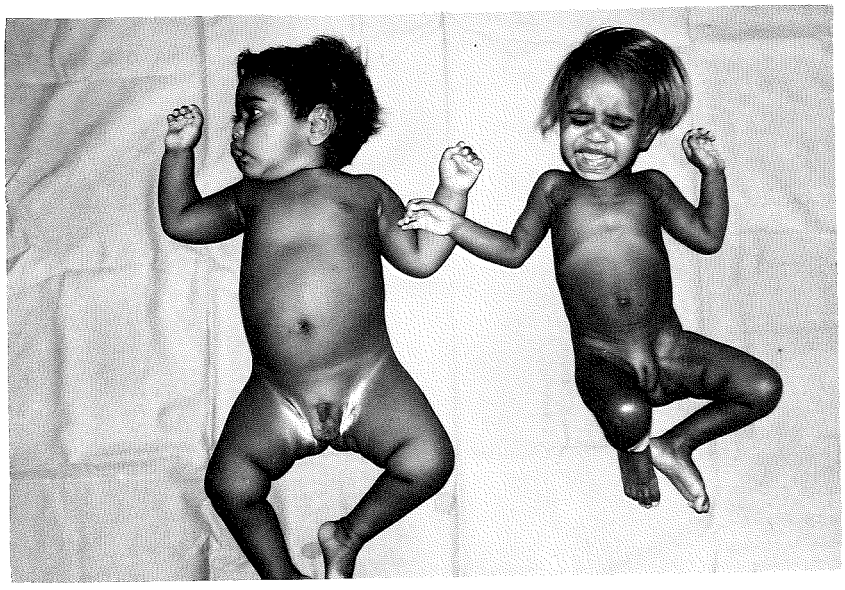

ely freely accessible. All of the above have been known to cause poisoning over the past few years, but in 1968 only Kerosene, turpentine, petrol, barbiturate and aspirin ingestion were seen. In the Aboriginal children it is almost always one of the petroleum products which does the damage.

Under the miscellaneous heading there is a heterogenous group of conditions. Corneal ulceration, pinhole urethral meatus, anaemia, renal calculous disease, dental problems, acute urinary retention, haematuria, prematurity, epilepsy, cystic lesions on lip and back and ulcerative stomatitis all occurred in the Aboriginal group. During 1968 no cases of acute nephritis were diagnosed in this age group but in 1966 two four year olds and one fourteen month old had the classical syndrome.

In the white and part coloured children the bulk of the miscellaneous conditions were taken up by tonsillectomy and adenoidectomy. There were several herniorrhaphies and plastic procedures for congenital lesions and these are included under the congenital heading. Rectal prolapse in siblings, hydrocele, rheumatic fever, ulcerative stomatitis and several convulsions, the cause of which not being fully identified, are also included in the miscellaneous group.

TABLE III.6 Disease Incidence Under Six Months

	ABORIGINAL	OTHERS
Diarrhoeal Disease	50	11
Respiratory Infection	33	6
Malnutrition	6	2
Other Infections	11	0
Trauma	1	2
Social	2	0
Ingestion	2	0
Miscellaneous	5	3
Congenital	2	0
NO. OF DIAGNOSES	112	24
NO. OF PATIENTS	108	18

.....

TABLE III.7 Disease Incidence 6 - 11 Months

	ABORIGINAL	OTHERS
Diarrhoeal Disease	63	17
Respiratory Infection	38	17
Malnutrition	16	2
Other Infections	4	2
Trauma	2	0
Congenital	3	2
Social	1	0
Ingestion	0	0
Miscellaneous	4	0
NO. OF DIAGNOSES	131	40
NO. OF PATIENTS	119	32

.....

TABLE III.8 Disease Incidence 12 - 17 Months

	ABORIGINAL	OTHERS
Diarrhoeal Disease	69	14
Respiratory Infection	39	10
Malnutrition	21	0
Other Infections	3	0
Trauma	0	2
Congenital	2	2
Social	1	0
Ingestion	1	0
Miscellaneous	5	4
NO. OF DIAGNOSES	141	32
NO. OF PATIENTS	136	23

.....

TABLE III.9 Disease Incidence 18 - 23 Months

	ABORIGINAL	OTHERS
Diarrhoeal Disease	23	3
Respiratory Infection	10	9
Malnutrition	9	0
Other Infections	7	0
Trauma	4	0
Congenital	0	0
Social	1	0
Ingestion	0	0
Miscellaneous	5	5
NO. OF DIAGNOSES	59	17
NO. OF PATIENTS	56	14

.....

TABLE III.10 Disease Incidence 24 - 59 Months

	ABORIGINAL	OTHERS
Diarrhoeal Disease	44	24
Respiratory Infections	28	43
Malnutrition	12	0
Other Infections	15	2
Trauma	17	20
Congenital	5	0
Social	2	2
Ingestion	2	10
Miscellaneous	16	40
NO. OF DIAGNOSES	141	141
NO. OF PATIENTS	133	131

These tables suggest that up to eighteen months of age, and probably two years, in both racial groups the incidence of the two primary diseases, Diarrhoea and Respiratory infection remain fairly constant. Diarrhoea takes precedence in all age groups in the Aboriginals and up to eighteen months in the whites and part-coloured children when Respiratory infection takes the lead.

"Other Infections" and "Malnutrition" in the Aboriginals outnumber by far these diagnoses in the whites at

all ages, while the other diseases occur with similar frequency in each group up to two years. After two years of age poisoning and the miscellaneous illnesses predominate in the non-Aboriginals.

In the first two years of life malnutrition and "Other Infections" are ten times more commonly causes of admission in the Aboriginals. Similarly, diarrhoea is three and a half times and respiratory infection twice as often the diagnoses on admission in Aboriginal children as in the others. These figures are summarised in Table III.11.

TABLE III.11 Admissions in under Two Year Olds (1968)

	ABORIGINAL		OTHERS	
Diarrhoea	205	(32%)	45	(9.4%)
Respiratory Infection	120	(19%)	42	(8.8%)
Malnutrition	52	(8%)	4	(0.8%)
Other Infections	25	(4%)	2	(0.4%)
TOTAL ADMISSIONS UNDER TWO YEARS	402	(63%)	93	(19.4%)
POPULATION AT RISK	640		480	

It is clear that even the figures for Europeans and part-Aboriginals represent a high morbidity, but in the full blood group the number of admissions during 1968 corresponded to 63% of the population at risk.

Domiciliary Origin of Hospital Patients.

Some places have a higher morbidity rate than others. The areas where the respiratory infection rate is high, also have a high incidence of malnutrition and mortality. These phenomena are considered later under the appropriate headings.

Although there are exceptions, a generalisation can be made, that morbidity, as expressed by hospital admissions, is lower on Church Missions than elsewhere. Later it will be shown that this also holds true for mortality. There are two Settlements, of the total of ten, where the paediatric Health Parameters are relatively acceptable at present, and one of the three Missions where there is room for great improvement. The situation on the Cattle Stations and in the towns is so varied that little specific comment can be made except, once again, that the areas from which most patients come are those with the highest incidence of malnutrition, and where the living conditions are most conducive to the spread of infective disease.

As has been mentioned previously, the apparent morbidity at any particular place may depend very largely on the skill and courage of the nursing Sister on the spot. She may treat and cure cases which someone else would not hesitate to send to hospital. Thus any far reaching conclusions derived from the number of patients hospitalised

from an individual Mission or Settlement are scarcely warranted, unless further evidence can be brought forward. This corroborative information, particularly in regard to respiratory infection rate and malnutrition, is presented a little further on in this chapter.

At this stage only a general statement of the incidence of various conditions found in patients admitted from Settlements, Missions and Stations etc. is made. Table III.12 suggests that the Stations and towns have the worst record. This is true since in most instances there is no readily available nursing sister to initiate early treatment. The children living on stations generally have readier access to toxic materials like kerosene and weed killers, and stand a much greater chance of injury from machinery such as pumps, windmills, tractors etc. This group also includes those living in several establishments in and around Alice Springs itself. Some of these local patients suffer recurrent admissions with chronic lung disease, some are State Wards due to parental inadequacy and others may be surgical problems needing frequent hospital visits. Thus the number in the "Other" disease column for Stations etc. is misleadingly high since many of these children really belong on Settlements and Missions.

TABLE III.12 Disease Incidence in Domiciliary Groups

(Admission diagnoses, 1968, Under 5 Years)

	SETTLEMENTS	MISSIONS	STATIONS
Diarrhoea	126 (15%)	31 (11.1%)	92 (16.4%)
Resp. Infect.	86 (10.2%)	13 (4.6%)	49 (8.8%)
Malnutrition	36 (4.3%)	3 (1.1%)	25 (4.5%)
Other	31 (3.7%)	10 (3.6%)	82 (14.6%)
TOTAL	279 (33.2%)	57 (20.4%)	248 (44.3%)
POPULATION AT RISK (Approx.)	840	280	560

Infection Rates and Family Size.

In order to establish a relationship between family size and infection rate, one child from each of 154 separate families was studied from the point of view of history of infections. For these purposes an infection was taken to be an episode of respiratory infection, diarrhoeal disease or a combination of the two, severe enough to have warranted medical consultation or hospitalisation. The children were randomly selected from the history cards kept by the Aerial Medical Section of the N.T.M.S. and the number of infective episodes each suffered in the preceeding twelve months recorded. The number of children in each of the 154 families was also recorded in relation to the infections, and it was apparent

that as family size increased there was a commensurate rise in infection rate. The results are set out in Table III.13.

TABLE III.13 Family size and Infection Rates

NO. CHILDREN STUDIED	NO. OF SIBLINGS	INFECTIVE EPISODES	INFECT. PER CHILD IN 1 YR.
32	0	36	1.13
31	1	38	1.23
48	2 or 3	94	1.96
43	over 3	106	2.47

When the small size of the dwellings in which these 154 families lived, and in view of Smith's conclusions, it is reasonable to implicate overcrowding in the high morbidity rate.

DIARRHOEAL DISEASE

The title of this section is deliberately left fairly general, since both acute and chronic, infective and non-infective diarrhoeas are encountered. Unfortunately the tendency, certainly in the Alice Springs Hospital anyway, is to regard all diarrhoea as infectious, and to keep all patients with loose stools in one ward. This is dictated to a large extent by facilities and staff, however it often means that children with

loose stools for reasons other than infection of the gut, contract such an infection in hospital.

Up to half the patients admitted with diarrhoea sooner or later need intravenous alimentation, often for prolonged periods, to correct dehydration or electrolytic disturbances, both of which may be quite gross, or to provide gut rest in cases of severe and continuing fluid and electrolyte loss. Several well authenticated cases of acute weight loss of over 25% body weight have been seen, and some have, surprisingly, recovered following vast amounts of intravenous Dextrose/Saline. It is not uncommon to find serum Potassium levels as low as 1.2 milli equivalents per litre and Sodium over 200 milli equivalents per litre. Blood Urea is often over 100mgm. per 100 mls. and packed cell volume may be up to 50% or so in these cases. Fortunately such situations are becoming less frequent, as people in rural areas, realising the dangers, tend to initiate more effective treatment earlier and seek help if no improvement occurs within a short time.

Incidence.

In many parts of the world deaths from diarrhoeal disease outnumber those from any other single cause. (W.H.O., 1964) In Central Australia, especially amongst small children, the incidence of this disease is very high as already been indicated. Even considering only

those cases severe enough to need hospitalisation the attack rates are almost as high as in some developing countries such as Guatemala (Scrimshaw et al, 1968). If an estimate of the unseen, or unrecorded, cases of diarrhoea were to be included probably the incidence would be as high as anywhere in the world. Table III:14 and Figure III:2 set out the attack rates of acute diarrhoeal disease in various age groups.

The incidence of diarrhoea in neonates is relatively low but increases rapidly with age and by three months reaches a high level where it stays until eighteen months. It is the younger group which generally presents the problem of "weanling diarrhoea" spoken of in other countries (Scrimshaw, 1968).

A newborn baby is often kept, well wrapped up against flies, dogs and direct sunlight, in a large wooden coolamon or dish. Even in transit the mother will sling a strap under the coolamon and over her shoulder, and walk with the coolamon on her hip scarcely disturbing the baby at all. She will often breast feed almost continuously, even while carrying the baby. The child remains in the coolamon for most of its neonatal life. It is in this way, as well as because of the relatively hygienic breast feeding which is universally practised, that the neonate is protected against contact with

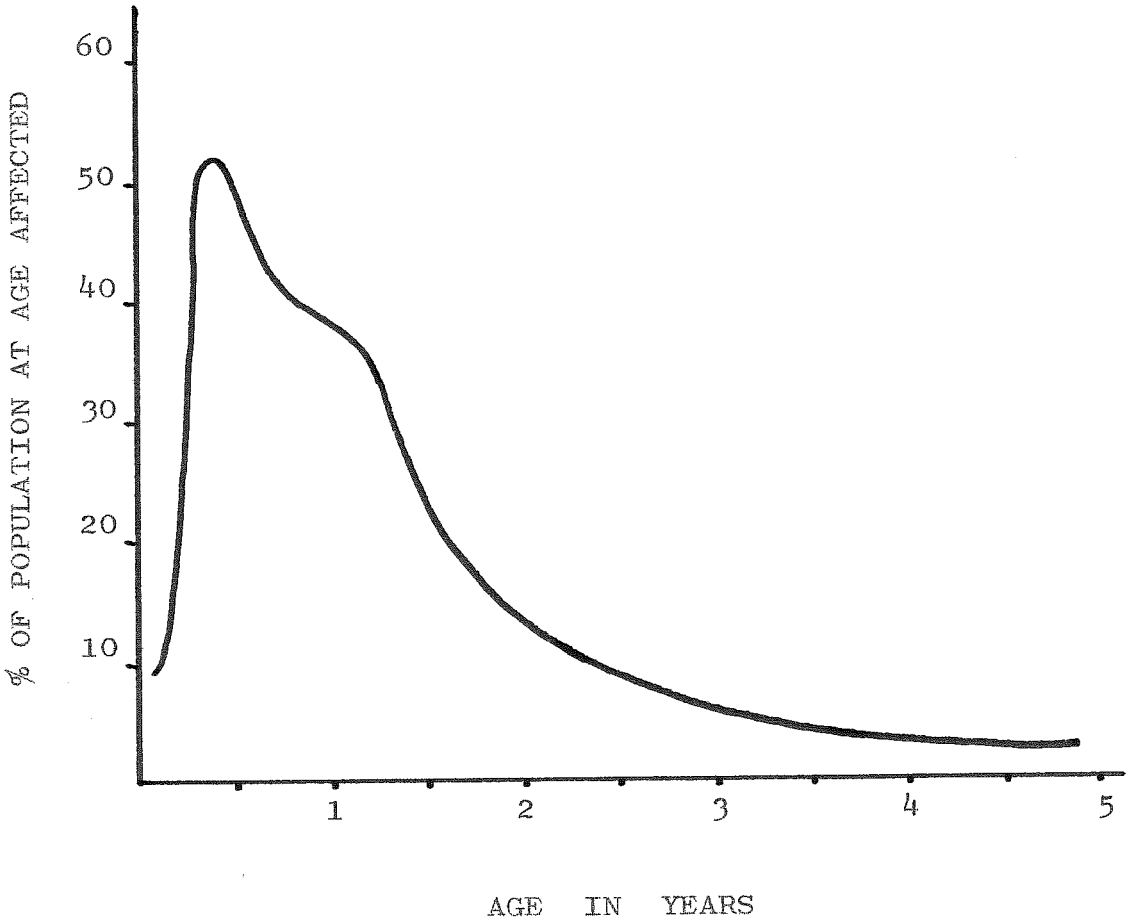

FIGURE III:2 Attack rates of Diarrhoeal Disease.

objects contaminated with enteropathogenic material. Unfortunately these mechanisms are less effective protection against droplet spread of infection so that respiratory infection tends to cause a higher morbidity than infective diarrhoea in neonates.

As the child's age increases, older children play with it more, and, as it becomes more active, it does not tolerate being tightly wrapped in a blanket, nor the close confines of the coolamon, and there is the consequent increased exposure to and incidence of bowel infection.

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TABLE III.14 Attack Rates of Acute Diarrhoeal Disease (1968)

AGE GROUP	NO. AT RISK (Approx.)	DIARRHOEAL CASES	ATTACK RATES (% per Yr.)
Less than 1 mth.	33	3	9.1
One month	30	4	13.2
Two months	29	7	24.2
Three months	28	13	46.4
Four months	27	15	55.6
Five months	25	8	32.0
0 - 5	172	50	29.0
6 - 11	170	63	37.0
12 - 17	165	69	41.8
18 - 23	160	23	14.4
Two years	320	29	9.1
Three years	318	11	3.5
Four years	316	4	1.3

Epidemiology.

Epidemiological division of the diarrhoeas is not simple, since all types occur with very much the same frequency in most areas. Probably in this situation a separation into acute and chronic, infective and non-infective disease would be possible, but these types overlap and interrelate so much as to make the divisions rather ludicrous. The individual aetiologies are

described later.

How far dogs and flies can be inculpated in the spread of enteric disease is not clear, although both are certainly involved. There is no need to go beyond a study of the living conditions of these people to understand why the incidence of diarrhoea is high.

In most big camps gastroenteritis is endemic and frequently epidemic. Some infants and small children contract acute diarrhoea with such frequency that it seems to be a chronic condition which interferes with nutritional development. Even under hospital conditions the bowels of these children sometimes take many weeks to normalise, and, in some instances, depletion of certain digestive enzymes has been postulated. It is clear that a large proportion of Aboriginal children between the ages of four or five months and eighteen months have many more relatively loose stools than do their white counterparts. Commonly these children have from two to six bulky loose bowel actions daily without causing their parents undue alarm. In these cases, usually no entero-pathogenic organism is found, although Giardia Lamblia, Trichomonas and Hymenolepis Nana infestations are sometimes seen, and may be at least partly responsible. Very frequently this chronic malabsorptive situation

responds to prolonged exposure to a disaccharide-free diet. Such problems are discussed more extensively in this chapter under appropriate headings.

The age distribution of patients with diarrhoeal disease has already been described (Table III.14), but it is of importance that the infective diarrhoeas (including viral gastroenteritis) are more common in the younger group, whereas the more chronic non-infective and debilitating situation mentioned above, albeit sometimes precipitated by an acute infection, is commoner over nine or ten months. It has been shown (Kirke, 1969) that malnutrition is at its most progressive stage at about nine months, and there is certainly an important relationship between this and the severe protracted diarrhoea seen at the same age. The "Diarrhoea of Malnutrition" has been described by others in relation to Kwashiorkor (Bowie, Brinkman and Hansen 1963 & 1965; Wharton, Howells and Phillips, 1968; Chandra, Pawa and Ghai, 1968), and it appears that a similar situation occurs as part of the marasmic malnutrition seen in Central Australia.

During the hot summer there are relatively more cases of diarrhoea hospitalised than at other times of the year. This is due partly to the ease with which infants become dehydrated when the ambient temperature

is well over 100 degrees Fahrenheit, and probably partly to the tremendous increase in the fly population in Summer. Other factors, such as evaporation of already poor water supplies, with concentration of both infective and chemical contaminants, and absence of nocturnal frosts enabling certain organisms, particularly the protozoan group, to survive in a free living form may well be important.

Shigellosis, and to a less degree Salmonellosis, are endemic in the community and there are clearly many "carriers" living in rural areas, and unfortunately many cases are contracted in hospital.

The living conditions of these people have been described, but when in addition to primitive and overcrowded housing, lack of hygiene and inadequate water supply, there is a high incidence of purulent rhinorrhoea and otorrhoea, widespread impetigo, mangy, flea-ridden dogs and inescapable clouds of flies, a low incidence of infantile diarrhoea would be a miracle.

Aetiology.

The cause of diarrhoea in Aboriginal children is not always easy to decipher, and, quite often, at least initial treatment, where it depends upon aetiological diagnosis, must be dictated by clinical experience. In

many cases several possible causes are superimposed and it is only therapeutic trial that decides which ones are significant. Usually the therapeutic problems, certainly in patients sufficiently ill to warrant hospitalisation, are primarily those of fluid and electrolyte balance in malnutrition. Appendix A outlines the general approach to diarrhoeal illness both in rural areas and in hospital. Of course in the ward situation intravenous management commonly enables electrolytic upsets to be rectified rapidly and good renal function to be established. Recently, administration of especially prepared intravenous solution of fat ("Intralipid") and amino acids ("Aminofusin"), have proved to be of value in malnourished children.

It is only following rehydration and so on that the aetiology of the diarrhoea becomes important. For the purposes of this discussion diarrhoeal disease is divided as Infective and non-infective, which headings to some extent correspond to acute and chronic respectively, but are not mutually exclusive.

- Infective Diarrhoea

It has been indicated that facilities for isolating enteropathogenic organisms are not entirely adequate, and there may well be some infective agents which are not discovered. On the other hand some evidence of parasitic

bowel infestation may be reported and yet have no bearing of the disease process. Since no virological facilities exist in the Northern Territory a diagnosis of "viral" enteritis is based on the absence of demonstrable enteropathogens and the clinical situation. Often several patients from the same area are notified simultaneously, all with a similar acute, and sometimes toxic, illness and if no organism can be isolated from their stools it seems fair to label them "viral".

a. Bacterial

Over the four years 1965 to 1968 the representation of Shigellosis and Salmonellosis varied. In this time the Shigelae (both Sonnei and Flexner strains) were much more common but near the end of the period the Salmonellae were increasing. Only rarely have there been even minor epidemics caused by either of these organisms, although sporadic cases appear quite frequently. Some places, no doubt those harbouring carriers, produce more Shigellosis than others. During the period under consideration, so common was Shigellosis that any child with mucus and blood in his stool, and even a mild fever, was treated as Shigellosis.

Generally 12 to 15% of diarrhoeal admissions in the under five year old age group have a Shigella organism grown from their first rectal swab. In some cases it

would seem that this may be a coincidental finding, their clinical picture being so unlike that expected with Shigellosis. However most of them present with blood stained, mucousy stools, high fever and prostration. Usually the Salmonella causes a less violent illness and its recovery from a routine rectal swab may come as a surprise.

Other bacteria probably cause a significant number of cases of diarrhoea, especially in debilitated children. The extent to which pathogenic E. Coli is culpable is unknown as it is only within the recent past that the laboratory has been able to differentiate them from the non-pathogenic strains. Pseudomonas Pyocanaea, however, is seen not infrequently, and has on several occasions, about five or six times per year, caused septicaemia. Probably not all of these septicaemias emanated from the bowel, and it is likely that some arose from infected intravenous cutdown sites, and so on. Proteus species may infrequently cause diarrhoea too.

b. Viral

It is this group that provided most of the epidemics of infantile diarrhoea and sometimes provokes what is called "white diarrhoea". In October of 1968 the epidemic of gastroenteritis at Yuendumu Settlement affected well over half the pre-school population of 200. These children had a minor upper respiratory infection,

apparently viral in origin, and frequent, almost colourless, watery stools. The fever was minimal and a mild lymphocytosis was common, but no enteropathogenic organism could be isolated from the stool. About twelve of these patients had to be evacuated to hospital and another twelve had intraperitoneal infusions of Normal Saline, some on several occasions, in order to maintain hydration and renal function. Only one death occurred as a result of this epidemic.

This condition where clear or slightly milky fluid stools, or "white diarrhoea", occur has acquired a very bad reputation since in the past it caused many infantile deaths, and it has been regarded with some superstition by both Aboriginals and Europeans. It usually occurs in epidemic form, no pathogens are isolated and it has a predilection for young children although it can affect all ages. Children with this infection deteriorate very rapidly and manifest gross electrolyte changes in their sera and electrocardiograph. Two factors seem to be important in this situation. One is that the severity of the situation may not be appreciated since the waterlike stools are mistaken for urine, and the other is that the diarrhoea fluid contains very high concentration of electrolytes. During 1966, a similar epidemic occurred in hospital and 15 samples of this "white diarrhoea" were

collected for electrolyte assay. The result was quite illuminating since the mean Sodium and Potassium concentrations were respectively 54 milli equivalents and 28 milli equivalents per litre. In several cases these levels, especially that of Potassium, were much higher than the mean. The range for Sodium was 41-85 and for Potassium 19-73 milli equivalents per litre. Thus a 10kgm. child who passes one stool of 5 ozs. containing, say 50 meq. of Potassium per litre, has lost one half of his normal daily potassium requirement. Small wonder then, that after ten such stools, his myocardium is affected unless huge amounts of Potassium are included in the replacement fluid.

These children suffering from the dreaded "white diarrhoea" presented a frightening picture of rapidly progressive hypernatraemia, hypokalaemia and dehydration. Generally speaking a "gut resting" Intravenous infusion of Half Normal Saline in 2.5% Dextrose with up to eight Grams of Potassium Chloride per litre rectified the situation fairly rapidly. Intraperitoneal infusions have proven effective in some cases, and particularly is it a useful technique in remote areas as a stop gap until evacuation can be arranged.

Another factor which not infrequently leads to late presentation in cases of acute infective diarrhoea is

pooling of fluid in the gut. Great importance is quite correctly placed on sudden weight changes, but in the situation where a large volume of electrolyte laden fluid is transuded into the bowel cavity and retained, the weight may not change. This fluid is not available and the circulation might be quite hypovolaemic. Unless signs of dehydration, other than weight loss, are looked for, the gravity of the condition is missed.

Not all the non-bacterial, acute, infective diarrhoea presents such a severe picture as the one described above and, of course, quite frequently cases of a much milder nature are encountered. In such cases the aetiology is usually not discovered and is probably of no real importance, at least from the therapeutic view point.

c. Parasitic

Various parasites are seen in rectal swabs and faeces specimens quite frequently. Whether or not they cause diarrhoea in an individual is difficult to ascertain other than by eradicating a particular organism and assessing the clinical response. Even this method is not accurate since the diarrhoea may improve spontaneously.

In children debilitated by malnutrition, diarrhoea or respiratory disease such organisms as Monilia and Trichomonas clearly can cause diarrhoea which may be

quite severe and protracted and which responds to appropriate treatment. Giardia Lamblia is another commonly occurring parasite which undoubtedly causes symptoms and frequently requires treatment.

Hymenolepis Nana, the dwarf tapeworm, has a fairly high incidence in children in this area and may well be responsible for some cases of diarrhoea. Enterobiasis (or Oxyuriasis) occurs relatively infrequently in the Aboriginals whereas the economically better endowed Europeans in the same area have a higher incidence. This phenomenon has been reported in Western Nigeria (Jung and Jeliffe, 1958) but its explanation remains unknown.

Two other parasites were seen in a group of 52 faecal specimens sent to the School of Public Health and Tropical Medicine, University of Sydney. They probably cause no significant disease. Table III:15 shows the results of this survey of alimentary parasites affecting Aboriginal children in this area (B. McMillan, personal communication).

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TABLE III.15 Alimentary Parasites

PARASITE	INCIDENCE
Giardia Lamblia	23.1%
E. Hymenolepis Nana	9.6%
Entamoeba Coli	1.9%
Enterobius	1.9%
Endolimax Nana	1.9%

Fortunately Hookworm (Ancylostoma duodenale and Necator Americanus) is quite rare in Central Australia, no doubt because of the dryness of the climate. The three cases seen between 1965 and 1968 all came from the North end of the Barkly Tableland.

The infective group comprises most of the acute diarrhoeas of infancy, although the parasitic ones can often run a prolonged chronic course.

- Non-Infective Diarrhoea.

Generally this group of conditions is more common in the older infants and one year olds i.e. over nine months, and, certainly as far as hospital admissions are concerned, almost exclusively in the malnourished members of that group. An exception to this rule is seen in younger children who develop diarrhoea following a sudden change in diet, such as occurs on admission to hospital.

It is not uncommon for a fully breast-fed baby with a respiratory infection to develop mild diarrhoea after a few days in hospital. There are several possible explanations for this occurrence amongst which are a change in intestinal flora due to antibiotics, ingestion of mucus, and possibly purulent material, coughed up from the lower respiratory tract and a change in diet from purely breast milk to one of much wider variety.

Another problem which has only relatively recently been appreciated in this country is the possibility of congenital or acquired deficiencies, or variations, in certain digestive enzymes, particularly Lactase (Elliott and Maxwell 1966; Elliott, Maxwell and Vawser 1967; Elliott, Maxwell, Kneebone and Kirke 1969). It has been shown that a high proportion of Aboriginal infants have a "flat" lactose tolerance curve and if much lactose is included in their diet, diarrhoea supervenes (Elliott and Kirke 1966, unpublished data). Most of the proprietary artificial milks in general use in this area contain a fair amount of Lactose, and sometimes diarrhoea can be attributed to a lactase deficiency. Much more rarely cases of Sucrose and/or Fructose intolerance occur. Generally speaking this phenomenon of sugar malabsorption only becomes a problem during attempts at rehabilitation of malnourished children, when recourse must be made to

special milks and diets with low disaccaride contents. The difficulty of supplying enough calories in an easily absorbable form then arises and is often responsible for the painfully slow recovery that some of these children exhibit. More will be said about sugar malabsorption in the following section, since it seems that the bulk of cases are acquired, rather than congenital, and usually closely related to malnutrition and environmental considerations.

DIARRHOEA AND MALNUTRITION

The relationship between malnutrition and diarrhoea is of utmost importance, and will be discussed at length.

A much higher proportion of infants and children hospitalised with diarrhoea are malnourished than occurs in this age group at large. Table III.16 shows a breakdown, by age and nutritional status, of 97 consecutive diarrhoeal admissions in 1966. No accurate figures for the nutritional status of the population at large in 1966 are available, but the relevant 1968 figures are quoted (Kirke, 1969).

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TABLE III:16 Diarrhoea and Malnutrition

AGE (Months)	DIARRHOEAL ADMISSIONS	MALNOURISHED PATIENTS	MALNOURISHED AT LARGE
0 - 5	23	60.9%	12.7%
6 - 11	26	81.5%	14.6%
12 - 17	29	72.4%	20.9%
18 - 59	19	83.3%	16.8%
0 - 59	97	74.2%	16.5%

There are three possible hypotheses to explain this relationship between malnutrition and diarrhoea. These are :-

a. That diarrhoea causes malnutrition,

b. that only malnourished children contract diarrhoea, and,

c. that diarrhoea in the malnourished is a more severe disease than in the well nourished children, (since it is the more severe cases which are admitted to hospital and appear in Table III:16).

During the widespread epidemic of gastroenteritis at Yuendumu Settlement in October 1968 there was very little difference between the nutritional status of the group seen with diarrhoea and that of the same age group in general. This instance, at least, suggests that well

nourished children if exposed to the infection are as prone to contract gastroenteritis as those less well nourished. Possibly the mother of a well fed baby is more likely to understand cross infectivity than a mother who rears malnourished children, and, even under epidemic conditions, the well nourished infants may be minimally less likely to be exposed and therefore infected as a group.

Diarrhoea, no matter what causes it, if sufficiently prolonged or recurrent aggravates pre-existing malnutrition. In occasional cases, seen in the Alice Springs Hospital, it has appeared that the onset of diarrhoea preceded that of malnutrition, and had been responsible in some degree for it, but much more commonly, when a well nourished child acquires diarrhoea (usually infective) it is non-debilitating, relatively short-lived and easy to treat. This is similar to the same disease, in all respects, in white children seen in Paediatric hospitals.

Whether or not an episode of diarrhoeal disease precipitates a steady and prolonged deterioration in nutritional status is not really important, since it is highly likely that the malnutrition syndrome has other causes, related to lack of calories and other nutrients in the second six months of life.

It has already been suggested that malnutrition can cause diarrhoea per se. In fact diarrhoea is described as being an integral part of Kwashiorkor (Wharton et al, 1968), and carbohydrate intolerance, particularly lactose, has been implicated as the major cause of the diarrhoea (Dean, 1952).

There is a fair amount of evidence to suggest that a similar mechanism exists in the marasmic situation which is commonly seen in Australian Aboriginal children. Florid Kwashiorkor is very uncommon. Since malnutrition interferes with development generally, it is not unreasonable to expect some atrophic changes not only in the bowel wall, pancreas and liver but also in some, or all, enzyme systems. It would not be surprising, also, when it is considered that the digestive disaccharidases reside in the superficial small bowel epithelium, if an episode of diarrhoea did not deplete these enzymes. As Chandra et al (1968) point out, the Lactase has a lower activity than Sucrase or Maltase and so becomes inactive through depletion and/or other changes first.

Elliott and Maxwell (1966) described the Lactase in the small bowel of Australian Aboriginal children and pointed out the predominance of a small molecular sized enzyme. This quite striking difference from the expected "normal" as seen in white children may be inherited,

or acquired and related to environmental factors. It is possible that this unusual Lactase, with an even lower activity than normal, may be highly susceptible to depletion and/or inactivation, thus explaining the very high incidence of Lactose maldigestion (to normal tolerance testing) amongst the Australian Aboriginals. Elliot and Maxwell quote figures for children on two settlements where only 5% at one place and 15.8% at the other showed a rise in blood sugar level of over 30mgm.% following a loading dose of lactose. In 1966, 26 Aboriginal children in the Alice Springs Hospital were submitted to Lactose tolerance tests and the result showed only 3 (11.5%) with a normal response. All 26 had a relatively normal d-xylose absorption. Table III.17 shows the results of these lactose tolerance tests and the weight percentiles. In the group shown here only 5 of 26 had weights over the 3rd. percentile and no definite conclusion can be drawn in this respect.

TABLE III.17 Lactose Tolerance Tests (1966)

RISE IN BLOOD SUGAR (mgms.%)	PERCENTILES	
	Less than 3	3 - 10
Less than 20	16	4
20 - 30	3	-
More than 30	2	1

The malabsorption of other carbohydrates may be, and sometimes quite definitely is, involved in the production of diarrhoea in ways similar to the Lactose maldigestion. Infection of the gut with bacteria or parasites also serves to injure the epithelium and therefore the dissaccharidase system, but malnutrition by itself is often sufficient to cause maldigestion in this way.

The presence of undigested Lactose (and/or other sugar) in the gut causes an osmotic force which sucks fluid into the lumen. The commensal flora in the colon ferment the sugars into organic acids which in turn irritate the large bowel and the consequent hyperperistalsis and intestinal hurry result in fluid loss, non-absorption of other nutrients, further epithelial damage and enzymatic depletion and so on. Fortunately this full blown vicious cycle is not common as evidenced by the relatively normal absorption of d-xylose by the 26 children studied, but, even when only partly developed, the situation is dangerous and difficult to manage.

The interaction between malnutrition and diarrhoea is complex but all three of the above hypotheses can be true. That is that diarrhoea can cause malnutrition, malnutrition can cause diarrhoea and, most importantly, that diarrhoea, from any cause, is a much more severe

disease in malnourished children than in those better nourished.

RESPIRATORY DISEASE

Although respiratory disease precipitates fewer hospital admissions in Alice Springs than does diarrhoea, lung infection is responsible for more deaths in the first year of life. Recent reports by Maxwell and his colleagues (1968) and Gandevia (1967) indicate a tremendously high incidence of both upper and lower respiratory tract disease in children at Papunya Settlement. By physical examination alone 27 - 28% of the children had abnormal lung signs, and in the same group 26% of 338 children were found to have abnormal chest X-rays.

More than twice the number of radio-medical consultations regarding Aboriginal children relate to respiratory disease than to all other medical conditions combined. Such is the efficacy of modern chemotherapy that even severe bronchopulmonary infections can be aborted by relatively little trained personnel in rural areas. It is also true that, although acute infection is often successfully supressed, without facilities for physiotherapy, radiology and proper control of hydration, many cases of pneumonia are left incompletely resolved. Bronchiolitis is excessively common at the beginning of Winter and, unless great

care is exercised, small patches of atelectasis and broncho-pneumonic consolidation tend to persist, only to cause considerable trouble later on. Many of the radiological abnormalities found, even in quite young children, must be classed as chronic destructive lung disease and clearly have their origin in an acute-on-chronic process stemming from the unresolved infection. Measles has been a very potent cause of lung disease and, in this malnourished community, is responsible for a great deal of morbidity. Morley et al (1967 and 1968) have pointed out that measles is a more severe disease in malnourished children.

The part played by other viruses, and related infecting agents, in the production of respiratory morbidity is unknown but presumably highly significant. Evidence for viral aetiology is sometimes manifest by a lack of response, both clinical and radiological, to a barrage of broad-spectrum antibiosis.

One of the most time consuming therapeutic problems in the current situation, both in rural areas and in hospital, is the management of chronic suppurative otitis media. Experience with this disease has led to the belief that none of the recognised forms of treatment is of lasting value unless accompanied by meticulous ear toilets three or more times daily. Even then it seems that tympanoplasty and non-return to the infective, fly-ridden home

environment must follow if the child's hearing, or what remains thereof, is to be saved. Significant deafness from suppurative otitis media is an increasing and rather overwhelming problem amongst school children. In one place half of a class of thirty should by virtue of their degree of hearing loss either sit at the front, wear a hearing aid or attend a special school for deaf children. A relationship appears to exist between chronic suppurative upper respiratory tract disease, destructive lung disease and malnutrition in that children suffering from one often have evidence of one, or more usually both, of the others.

Severe and extensive lung infections are becoming more common in Central Australia. Much of the disease is Staphylococcal and the incidence of Penicillin resistant Staphylococci has risen at a phenomenal rate over the last five years.

Table III.18 shows this increase through the medium of routine nasal swab cultures of new admissions to the Alic Springs Hospital.

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TABLE III.18 Rising Incidence of Penicillin-resistant Staphylococcus Aureus in Nasal Flora (1965-69)

	1965-66	1967-68	1969
Number of Staph. Isolated	23	32	18
Resistant to Penicillin	4	19	16
% Resistance	17%	59%	89%

During 1969, 93% of the Staphylococci isolated from nasal cultures of European paediatric inpatients were penicillin resistant.

Rowntree (1969) found that 40 out of 41 Staphylococci isolated from nasal cultures at Ernabella were Penicillinase producers, and it is tempting to relate the rising incidence of Staphylococcal pneumonia seen in hospital to the now relatively ineffective use of Penicillin for this disease in remote areas. It is of interest that Rowntree found 18.9% of children under 15 years to be Staphylococci carriers whereas only 6% of the adults were similarly afflicted. Possibly, if she had made the division at, say, 5 years the percentage may well have been higher in the younger group, since it is this group from which the hospital patients in the main are derived. No doubt many of these penicillinase producing Staphylococci originate in hospital, and are transported to rural areas in the noses of repatriated patients.

Incidence.

The incidence of major and minor respiratory infection in Aboriginal children is enormously high. During routine examination of any group, be it in hospital or in a rural situation, it is usual to find several small children with apparently symptomless acute otitis media. This infection would normally go undetected, and therefore untreated, and probably initiates many cases of chronic suppurative otitis media and eventual deafness. In most groups of school children at least half can produce a loose cough on demand, and amongst the younger, pre-school group, although assessment is not so easy, the proportion with a loose cough seems even higher. In a given place the number of children with acute respiratory infection fluctuates widely, as it does from place to place. Often, during one of the frequent times when some minor respiratory epidemic is present, it is extraordinarily difficult to find a child not affected.

CHRONIC SUPPURATIVE OTITIS MEDIA

The extent to which suppurative otitis media causes immediate and delayed complications is difficult to define. Several cases of meningitis have apparently originated in the middle ear and spread to the meninges via the mastoid bones, and, on occasion, infective foci in ears have been suspected of causing septicaemia. Certainly

cervical adenitis, often with abscess formation, quite frequently stems from otitis media. Apart from these relatively obvious problems there are other aspects to be considered. Each discharging ear constitutes a public health hazard, especially in view of the enormous numbers of flies available to transfer infective material to utensils, food, clothes and other people.

Whatever the cause of the initial acute upper respiratory infection, once chronicity has supervened, the bacteriological content of the pus is largely gram negative rods such as Proteus species, E. Coli and Pseudomonas, although some gram positive cocci may also appear. When the chronic condition is well established it is extraordinarily difficult to eradicate, and some degree of hearing loss develops eventually in a high proportion of sufferers.

When it is considered that many of the problems of Aboriginals relate to their lack of European education, then any factor rendering the children less easy to educate must be taken very seriously indeed. Deafness, and its causes, are therefore highly significant morbid factors in the community in question.

Generally, smaller population groups, usually those on Cattle Stations, have less ear problems than the much

larger, over-crowded groups on Settlements and Missions. The highest incidence of chronic suppurative otitis media is found in the infants and toddlers on the biggest Settlements.

As for some other conditions discussed in this thesis, data related to otic disease for the whole paediatric group are reported rather than restricting them to the infant age group since they are closely related.

Tables III.19 and III.20 show the extent to which ear disease affects under 5 year olds and school children in relation to the size of the population in which they live. The incidence of chronic suppurative ear disease is more than twice as great in some places where the paediatric population exceeds 50. This phenomenon may be due to over crowding. On the other hand the smaller groups are, as a rule, more isolated, have less infectious visitors and maintain a much smaller infective pool in their own communities. Even diseases with relatively short incubation periods cannot be maintained continuously in the smaller aggregations of people. This may really only underline overcrowding as the culprit. In two larger places, Papunya and Areyonga, for which recent figures are available, ¾ of the under 5 year olds have one or both ears continually discharging infective material. It is not

surprising that up to a dozen dead flies can be removed from such ears quite regularly, and thoroughly fly-blown ears, with the middle ear cavity and the external canal choked with maggots, are seen not uncommonly. The bacteriological content of the ear discharge is probably not related to the original aetiology, but by virtue of the predominance of the gram negative bacilli is likely to be responsible for the chronicity and difficulties in cure of the otitis media. Table III.21 shows the results of 36 consecutive ear swabs taken from April to June 1968. More often than not two organisms grew from the swabs. The degree to which these germs are responsible for disease in other systems is unknown, but Proteus and E. Coli are frequent isolates from rectal swabs. Certainly the infected ears maintain a large pool of such infective material, and the flies ensure its spread far and wide.

TABLE III.19 Ear Disease in Small Communities (Less than 50 Children) 1969.

AGE	NO. IN COHORT	CHRONIC EAR DISEASE
0 - 5 Years	45	14 (31%)
5 - 15 Years	109	11 (10%)

.....

TABLE III.20 Ear Disease in Large Communities (50 Children or More) 1969.

AGE	NO. IN COHORT	CHRONIC EAR DISEASE
0 - 5 Years	303	200 (66%)
5 - 15 Years	451	131 (29.1%)

TABLE III.21 Bacteriology in Ear Infections (1968)

Proteus species	20
E. Coli and Coliforms	16
Pseudomonas Aeruginosa	7
B Haemolytic Streptococci	5
Staphyloccus albus	2
Staphylococcus aureus	3
Klebsiella species	2
Pneumonococcus	1

Deafness.

Significant acquired hearing loss, presumably resulting largely from chronic infection, affects up to one quarter of school age children in the areas of larger population. Unfortunately it is technically almost impossible to perform adequate audiometry in the under 5 year old group, especially in rural areas, so the

results of audiometric surveys, reported in this section, are necessarily related to older children. Since the disease, which undoubtedly causes the vast majority of deafness, usually becomes apparent in the infants it is reasonable to report the findings in this thesis.

Table III.22 shows briefly the audiometric survey results in 945 school children during 1967-69, The conditions, under which these children were tested, were not uncommonly somewhat inadequate. The audiometers used of necessity had to be light-weight and eminently portable, and the extraneous noise level in the testing rooms was sometimes high. However some allowance was made for noise and other distractions and it is felt that the results quoted are sufficiently accurate to be meaningful.

It is obvious that even in small communities deafness is a problem. The incidence of chronic ear discharge, and deafness, at the small and large population centres are very similar and there can be no doubt that a causal relationship exists between the two.. Table III.23 takes the figures from Tables III.19, III.20 and III.22 and serves to underline this parallel incidence.

TABLE III.22 Incidence of Hearing Loss.

Audiometric Survey of School Children in Small Communities (less than 50 Children) and Large Communities. (more than 50 Children)

	SIZE OF COMMUNITY	
	SMALL	LARGE
Number in cohort	216	729
Hearing Loss:- Unilat. or Moderate	9	148
Bilateral & Severe	4	36
Total significant Hearing Loss	6%	25.2%

TABLE III.23 Hearing Loss and Chronic Ear Discharge

Relationship between Chronic Ear Disease and Hearing Loss

(5 - 15 year olds)

	INCIDENCE OF CHRONIC EAR DISEASE	INCIDENCE OF SIGNIFICANT HEARING LOSS
Small Communities	10%	6%
Large Communities	29.1%	25.2%

.....

RADIOLOGICAL LUNG DISEASE

During the latter part of 1969 a mobile X-ray unit visited several parts of Central Australia. Owing to problems of transportation the unit used had some limitations, especially in its application to small children. Some of the films of this group are unreadable, or at least difficult to interpret, due to the subject's respirations during the required relatively prolonged exposure time.

The survey is not yet complete but, for the purpose of this thesis, films of an adequate and representative sample of the paediatric population are available. Approximately one third of the population under 13 years old are involved, and this sample includes groups from the places which, from clinical experience, would be expected to demonstrate the highest and lowest rates of abnormality.

Postero-anterior films of children 12 years and under have been examined and those from many places indicate an abnormality rate exceeding even that found by Maxwell et al (1968) at Papunya. Only 60% of the whole group were considered to be normal.

To simplify record keeping the films were segregated into three groups under the broad titles of Normal, Minor disease and Severe disease, and where a radiological

lesion was seen, the lung lobe in which it appeared was noted. Some difficulty in defining the exact location of several lesions was experienced due to the absence of lateral films. In many cases more than one lobe was affected.

The films labelled "Severe disease" included those indicating a need for relatively urgent clinical follow-up. Such abnormalities as lobar collapse, presumed to be of long-standing, changes suggestive of Tuberculosis, both peripheral and hilar, and a rather ill defined group, which experience dictates must be called "bronchiectasis until proven otherwise", are included under the heading "Severe". This latter diagnosis, which forms by far the most common severe situation, may variously be suggested by crowded bronchial markings, usually basal, and mottled or tubular opacities in the middle or lower lobes.

There are several films of children who had previously been diagnosed as bronchiectatic, and sent to the Adelaide Children's Hospital, where bronchography confirmed the condition. A few may be interpreted as indicating pulmonary tuberculous disease. One of these also showed a mid-thoracic spinal lesion almost certainly of tuberculous origin.

The cardiac shadow, and in some cases the pulmonary

vasculature, in 11 films suggested some sort of valvular disease. Most of these films were of older children and probably represented rheumatic damage.

Under the heading of minor disease is a protean group, not the least numerous among which are probably artefact, due to technically poor films, and misinterpretation, due to inexperience and an overly high index of suspicion, of real or imagined lesions. However, although this may be so, there is good correlation between independent reports by Professor Maxwell, the Department of Radiology at the Adelaide Children's Hospital, and the figures reported in this chapter for at least two groups of films from major centres of population.

Other minor changes, mostly of unresolved inflamation and acute infection, are included under the second heading. The line of demarcation, for example between severe and minor basal changes, was on occasion rather indefinite and some difficulty in maintaining a consistant approach was experienced during the reading of the 1271 films. Thus strict accuracy is not claimed for the following figures, but it is believed that the problem of respiratory disease in this community is indeed as extensive as these figures suggest.

Table III.24 summarises the radiological findings

in one third of the Central Australian Aboriginal paediatric population.

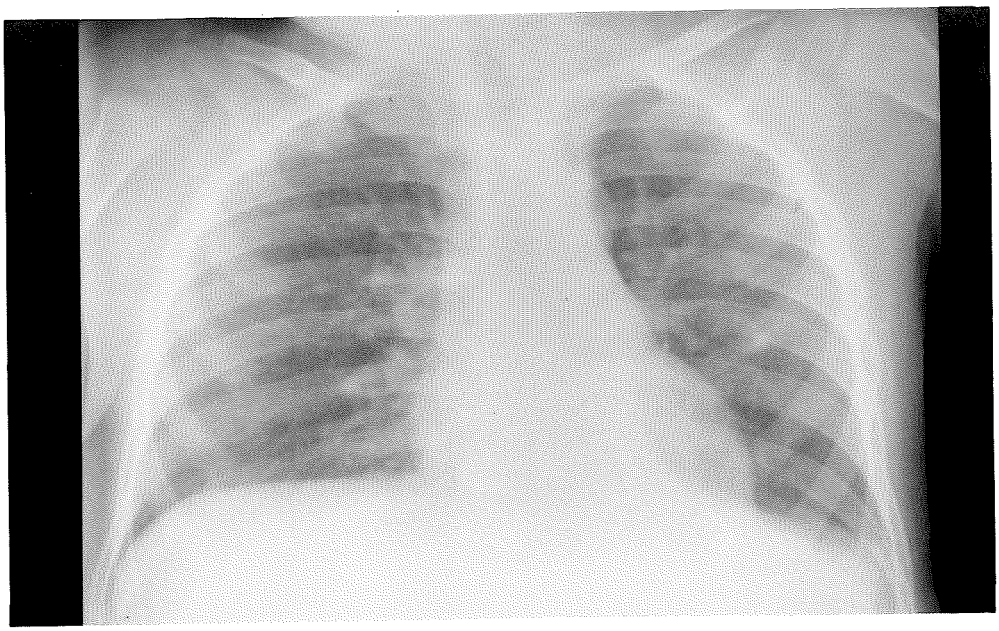
Using the same criteria, for films taken during the survey of 45 European children living in rural areas, there were no cases of severe lung disease, six with some minor lesion, and one with cardiomegaly.

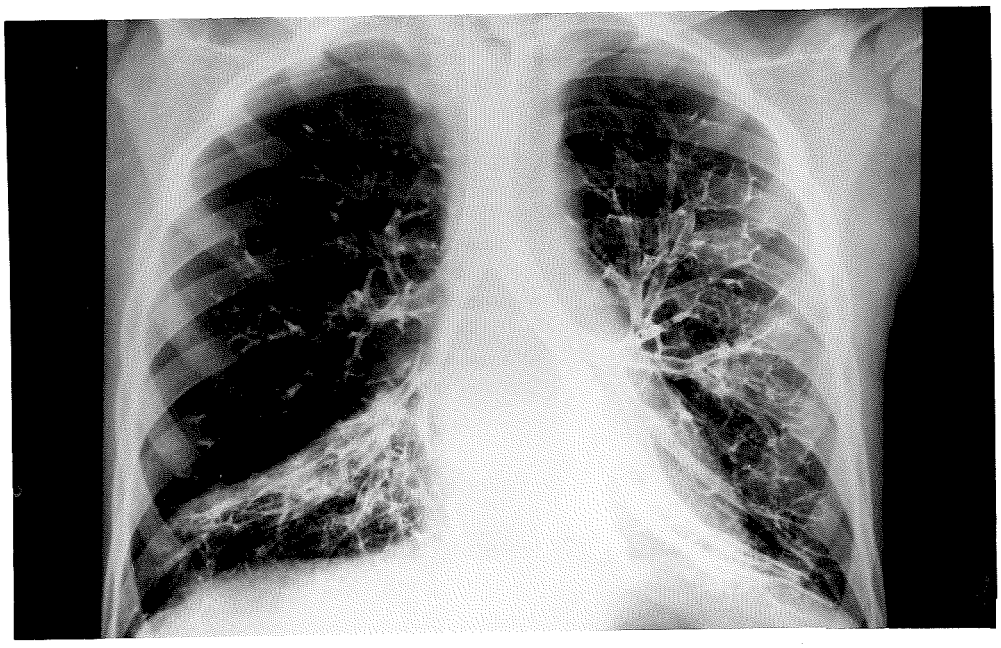
It is difficult to draw many far reaching conclusions from the table except the patently obviously one that radiological respiratory disease is excessively common. Both minor and severe lesions show a generally increasing incidence, steep at first, over the first four or five years.

Although the high incidence of what amounts to bronchiectasis in young children suggests a congenital basis, the rapidly rising incidence with age makes an acquired pathology much more likely. Indeed it has been impossible to demonstrate any pathological evidence of a congenital cystic condition at all. It has already been mentioned that measles is common and severe in this group of children and certainly plays a part in the production of chronic lung disease. Whooping cough and cystic fibrosis of the pancreas have traditionally been associated with the production of bronchiectasis, but these conditions are uncommon in Aboriginal children in Central Australia and are probably insignificant in this situation.

1. Chest radiograph of a seven year old boy, showing left lower lobe consolidation and atelectasis, and some abnormality on the right side.

2. Bronchogram of the same child showing extensive bronchiectasis.





After the age of six years there is a moderate fall in the amount of minor disease and a slight fall in the severe manifestations. Figure III.3 demonstrates these trends.

TABLE III.24 Radiological Lung Disease. Incidence of Minor and Severe Radiological Lung Disease In Age Groups

- 1969 -

AGE IN YEARS	SEVERE DISEASE	MINOR DISEASE	NORMAL	TOTAL
Less than 1	3 (8.3%)	4 (11.1%)	29 (80.6%)	36
1 to < 2	13 (11.2%)	39 (33.6%)	64 (55.2%)	116
2 " < 3	19 (15.7%)	33 (27.1%)	70 (57.2%)	122
3 " < 4	19 (16.2%)	34 (29.1%)	64 (54.7%)	117
4 " < 5	13 (11.5%)	42 (37.2%)	58 (51.3%)	113
5 " < 6	16 (15.4%)	29 (27.9%)	59 (36.7%)	104
6 " < 7	22 (19.8%)	32 (28.8%)	57 (51.4%)	111
7 " < 8	20 (18.0%)	24 (21.6%)	67 (60.4%)	111
8 " < 9	16 (15.3%)	20 (19.0%)	69 (65.7%)	105
9 " < 10	14 (16.1%)	20 (23.5%)	53 (60.4%)	87
10 " < 11	13 (14.1%)	15 (16.3%)	64 (69.6%)	92
11 " < 12	6 (9.0%)	16 (23.9%)	45 (67.1%)	67
12 " < 13	10 (11.1%)	19 (21.1%)	61 (67.8%)	90

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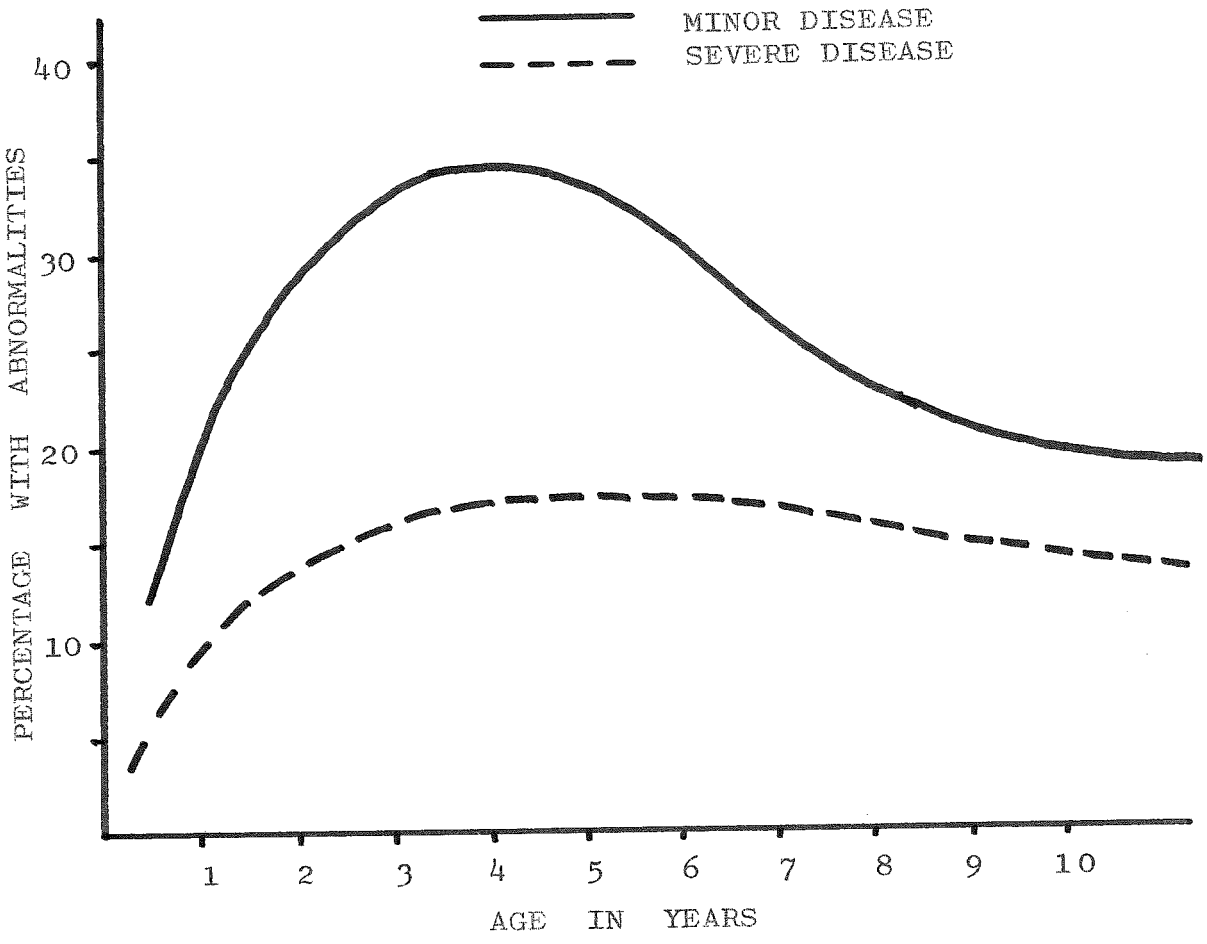

FIGURE III:3 Incidence of minor and severe radiological lung disease (1969).

The fall in incidence of severe disease with increasing age over about six years may only be apparent, in that some children, who develop severe destructive lung disease early in life, in fact die during this period. At any point in time several children with severe bronchopulmonary disease are likely to be in Adelaide for bronchography and possible surgery. The thoracic surgeon involved prefers these patients to be at least five or six years old, and so at the time of the survey a few children with severe disease and over six years old were not included.

It may also be that less children contracted severe and chronic disease five to ten years ago and the incidence thereof is therefore lower now in the older children.

The steepness of both curves during the first three years indicates that most of the respiratory disease is contracted very early in childhood. This is supported by the number of young patients suffering from bronchopulmonary infection admitted to hospital.

Geographical Incidence of Radiological Lung Disease.

The differences in incidence of radiological disease between various places is quite striking. There is less difference between Missions, Stations and Settlements as groups than there is between individual settlements and so on. Table III:25 shows these variations.

TABLE III:25 Geographical Incidence of Radiological Lung Lesions. Aboriginal Paediatric Lung Disease - 1969. Chest X-ray Survey.

PLACE	NO.	SEVERE DISEASE	MILD DISEASE	N.A.D.
Papunya	307	50 (16.3%)	86 (28.0%)	171 (55.7%)
Yuendumu	264	35 (13.3%)	59 (22.4%)	170 (64.3%)
Warrabri	194	12 (6.2%)	41 (21.1%)	141 (72.7%)
Docker River	63	18 (28.6%)	21 (33.3%)	24 (38.1%)
Amata	87	17 (19.5%)	24 (27.6%)	46 (52.9%)
Indulkana	76	4 (5.3%)	26 (34.2%)	46 (60.5%)
Ernabella	96	14 (14.5%)	35 (36.5%)	47 (49.0%)
Santa Teresa	184	34 (18.5%)	35 (19.0%)	115 (62.5%)
	1271	184 (14.5%)	327 (25.7%)	760 (59.8%)

The figures quoted for several places are falsely low since at the time of the X-ray survey several children with severe lung disease were in hospital. In fact at that time there were five patients from Indulkana and four from Ernabella thus situated and so the percentage with severe disease from these two places could read 11.3 and 18.0 respectively. Similarly four patients from Papunya and two from Amata were hospitalised and missed the Survey, and relevant percentages should be 17.4 and 21.3,

respectively.

One very intriguing feature of the geographical incidence is that the worst affected areas are those populated principally by Pitjantjatjara and Pintupi people, that is to say the Western Desert group of tribes. Once again there is no apparent reason why this should be so with respect to gamma-globulin levels and so on, although these are the tribes which tend to be rather more nomadic, to speak less English and to follow old cultural patterns more than others. They are consequently more resistant to investigation, prophylaxis and treatment of ailments of their children, of which malnutrition must take pride of place, but respiratory infection is a close second.

Severe Lung Disease and Malnutrition.

It is of significance that places with a high incidence of severe lung disease are also those with widespread childhood malnutrition. Table III.26 shows a striking similarity between numbers at various places with low weight percentiles, recorded less than six months before the X-ray Survey took place.

TABLE III.26 Geographical Distribution of Severe Lung Disease and Malnutrition.

PLACE	SEVERE LUNG DISEASE PERCENTAGE	WEIGHT LESS THAN 3rd CENTILE
Papunya	17.4	23.2
Yuendumu	13.3	11.6
Warrabri	6.2	7.5
Santa Teresa	18.5	17.3
Ernabella	18.0	20.8

Maxwell (1968) has shown that there is some relationship between Respiratory Tract Infection and malnutrition and Table III.27 gives further support to this hypothesis. It is appreciated that there are environmental factors which cause both malnutrition and lung disease, (and incidentally Chronic U.R.T.I.) so the relationship between the two may be causal only within limits.

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TABLE III:27 Radiological Lung Disease and Nutrition

	WEIGHT PERCENTILE	
	LESS THAN 3	3 AND OVER
Severe disease	11 (30.6%)	17 (12.0%)
Minor disease	9 (25.0%)	50 (35.2%)
Normal X-ray	16 (44.4%)	75 (52.8%)
	36	142

Since malnutrition reaches peak incidence in the second year of life and the respiratory infection rate continues to rise until the fifth or sixth year, it can be said, considering Table III:27, that the malnourished children have a higher incidence of severe lung disease than the better nourished. It seems that malnutrition, as expected, potentiates respiratory disease, and not so much that respiratory disease causes malnutrition.

No significant relationship between nutritional status and minor bronchopulmonary disease is apparent.

Aetiology.

Staphylococci and the Measles virus have previously been alluded to in this section. There is no evidence to suggest that the spectrum of infecting organisms is different in this population to that of any other.

The incidence of bronchial foreign body is very low since small plastic toys and peanuts have little place in this society. It might be anticipated that grass seeds, small stones and pieces of bone would occasionally become lodged in bronchi but if so they come to notice very rarely.

Four years ago, at thoracotomy, a sewing needle was removed from a two year old's pericardium. The needle, which fell in two on being handled, had apparently lodged in the Middle lobe via the bronchial passages and found its way, causing enroute an extensive middle lobe pneumonia, into the pericardial sac.

Ingestion of volatile hydrocarbons is a fairly infrequent cause of respiratory problems, which may allow opportunistic bacterial infection and chronicity.

Specific bacteriological categorisation of pulmonary infection is difficult, time consuming and usually unnecessary, since a broad classification can often be made on clinical, historical and radiological grounds. Table III.28 summarises the significant cultural findings in 100 consecutive positive routine nasal swabs taken on admission to hospital. The prependerance of gram positive cocci, in particular Streptococcus Pneumoniae, is not unexpected but there is a relatively high number of

such organisms as E. Coli, Proteus and Pseudomonas species. This is not really surprising in view of the content of ear discharge.

TABLE III.28 Nasal Flora.
Aboriginal children in the Alice Springs Hospital (100 Consecutive, positive cultures - 1969).

PREDOMINANT ORGANISM	NUMBER OF CASES
Pneumococcus	62
E. Coli, Proteus, Pseudomonas etc.	15
Staphylococcus Aureus	12
Alpha Haemolytic Streptococcus	4
Klebsiella species	4
N. Catarrhalis	2
Monilia	1
	100

The antibiotic sensitivities of these micro-organisms in vitro are generally as expected with two exceptions. The first, which has already been discussed, is related to the rising incidence of penicillin resistant Staphylococci and the second an apparent resistance, also to Penicillin, developing in the Alpha haemolytic Streptococci. Of the four Alpha Streptococci mentioned in Table III.28, two showed in vitro resistance to

penicillin. In order to delineate this unexpected finding the reports on 218 throat swabs from the same group of patients were examined. The results are shown in Table III.29. No definite clinical evidence of in vivo resistance to Penicillin by these streptococci is available, but it appears significant, that, during the same period, only two of the forty-three Alpha haemolytic Streptococci isolated from European patients of the same age group showed this in vitro resistance. Further study on this subject is currently in progress.

Traditionally the Alpha Streptococci are considered to be "normal" pharyngeal residents and only capable of causing disease by direct extension, as in paranasal sinusitis. Cases of Alpha Streptococcal endocarditis have been reported not infrequently, and since the operative factor here is apparently prior damage to the cardiac valves it may be possible that other organs, previously damaged by another infection, could become colonised by these organisms and chronic suppuration ensue. The middle ear and ectatic bronchi are particularly prone to chronic suppurative disease in these Aboriginal children and neither is far distant from the throat.

There is reputedly a causal relationship between

suppurative bronchiectasis and chronic paranasal sinusitis. Several children have demonstrated opaque sinuses on X-ray and a chronic purulent rhinorrhoea, and certainly destructive lung disease is common, but at present the group with radiological evidence of sinusitis is too small to warrant any definite conclusions as to causal relationships in this situation.

TABLE III.29 Pharyngeal Flora. Throat Swabs of 218 Aboriginal Children under 5 years old. (1968 - 1969)

PREDOMINANT ORGANISM	NUMBER
Alpha Haemolytic Streptococci (sensitive to Penicillin in vitro)	78
Alpha Haemolytic Streptococci (resistant to Penicillin in vitro)	51
Monilia	25
E. Coli	16
Beta Haemolytic Streptococci (sensitive to Penicillin in vitro)	14
Pneumococcus	11
Haemolytic Coliform	8
Klebsiella species	8
Proteus	3
N. Catarrhalis	3
Staphylococcus Aureus	1
	218

SUMMARY

Whatever the inter-relationships between chronic ear infection and destructive lung disease prove to be, it is clear that these two conditions are highly significant causes of morbidity in Aboriginal infants and children. Both are very much more common than in the white population and the highest incidence occurs in places with large population aggregations.

There appears to be a potentiating interaction between malnutrition and severe lung disease, but as yet no such relationship between nutritional status and chronic ear disease is apparent.

Chronic suppurative otitis media is a potent cause of significant hearing loss, which is of vital importance in view of the great need for education.

The aetiology of both severe lung disease and the suppurative ear condition is closely related to environmental factors, as with other infective conditions affecting infants and children. Overcrowding seems to be important, both in the sense of family sleeping units and of large communities, and its deleterious affect is traceable once again to poor hygienic practices, and lack of understanding.

Another important factor, emerging during the past five years, is the rising incidence of penicillin resistant Staphylococci. Medical workers in rural areas often have no effective antibiotics against such organisms and this may account for the increase in severe lung disease seen in hospital.

Measles has been an important cause of severe lung disease. Use of "Koplivac" vaccine may, in the immediate future, render measles insignificant.

MALNUTRITION

Malnutrition in young children is one of the principal health problems in Central Australia. It is difficult to make the public, especially the fund controlling administrators, realize the extent of the problem. So often the physical signs in a malnourished child are nonspecific and may pass quite unnoticed by untrained personnel. For example, unless the weight of a small, apparently healthy, 18 month old child is compared to a standard weight chart, he could pass for a well-nourished 10 month old.

The type of malnutrition common in this area causes delayed tooth eruption, slowed bone development, and so on, which makes the individual appear younger than he is and easier to label as well nourished at first sight.

Workers in other countries (Jeliffe, 1966) have pointed out that high mortality rates at particular age periods can be used as indicators of certain types of malnutrition. Although there is clearly a close relationship between malnutrition and mortality, the variable incidences of the other big contributors to mortality, pneumonia and diarrhoea, at different ages, make the relationship a little indistinct.

The level of childhood malnutrition in a community is an excellent public health index (Kirke, 1969). The syndrome of malnutrition may be contributed to by many factors other than intake of nutrients. Such biological variables as birth order, twinning, parental size, environmental considerations like climate, socio-economic level, infective and parasitic illnesses and even emotional deprivation (Whitten, Pettit and Fischhoffe, 1969) may all produce their effect on growth. In the past when the infant mortality was 250 per thousand live births it was probably an indication of malnutrition in this age group but now morbidity is of increasing importance and is intricately tied to malnutrition.

Classification of Malnutrition.

Broadly speaking Malnutrition may be defined as a pathological state arising from relative or absolute lack,

or excess, of one or more essential nutrients.

Malnutrition can generally be divided into four types, each of which may be clinically obvious or may only be detected by biochemical or physiological tests. The four types except the first and the last are by no means mutually exclusive.

(i) Subnutrition results from inadequate intake of food, usually over a long period. Marasmus and Inanition are terms used for the severe forms of subnutrition, and Starvation implies an almost complete lack of food, producing acute subnutrition.

(ii) Specific nutritional deficiency results from relative or absolute lack of a single nutrient.

(iii) Imbalance is the state resulting from disproportion among essential nutrients with or without deficiency.

(iv) Overnutrition results from excessive caloric intake, usually over an extended period.

It can be said that malnutrition of any sort interferes with normal growth. Therefore an assessment of infant and toddler growth patterns will suggest the nutritional status of the group and hence provide an index

of its morbidity load (Kirke, 1969).

There are many more specific ways of assessing nutrition than merely measuring a few simple physical parameters and comparing them to standard charts, however for the health worker in remote areas many sophisticated methods are precluded by time, expense, distance, lack of trained personnel and non-acceptance by the community. Some of these more complex tests have had limited use in this area, but have not been found superior to simple anthropometry. Needless to say, specific tests are often necessary to delineate specific deficiencies, but basic growth screening by relatively untrained personnel, at least brings to notice patients who require further investigation.

The most common type of malnutrition in the Central Australian Aboriginal community is Marasmus, occurring mainly in the first eighteen months of life. A similar picture exists in Chile, but in general it is in contrast to the situation in many other developing regions where Kwashiorkor is common and occurs in the 1-4 year old group.

In the local situation malnutrition is more closely related to morbidity and mortality in the first and second years of life, rather than later.

Simple subnutrition produces no pathognomonic clinical or biochemical lesions except for the loss, or lack of gain, of body mass. In practice of course the situation is rarely simple, as several specific deficiencies and possibly some infections are superimposed. The lack of characteristic signs in Marasmus serves to stress the importance of nutritional anthropometry in early diagnosis in this condition.

Assessment of Nutritional Status.

The lack of characteristic clinical signs in undernutrition has been stressed, although, obviously, advanced Marasmus produces a recognisable clinical picture. It is the less severe, albeit serious, degrees of the condition which need recognising early if lives are to be saved and therapeutic problems simplified. Physical examination remains an important part of general nutritional assessment. Other parameters such as Haemoglobin, Protein and Ascorbic Acid are dealt with as specific deficiencies.

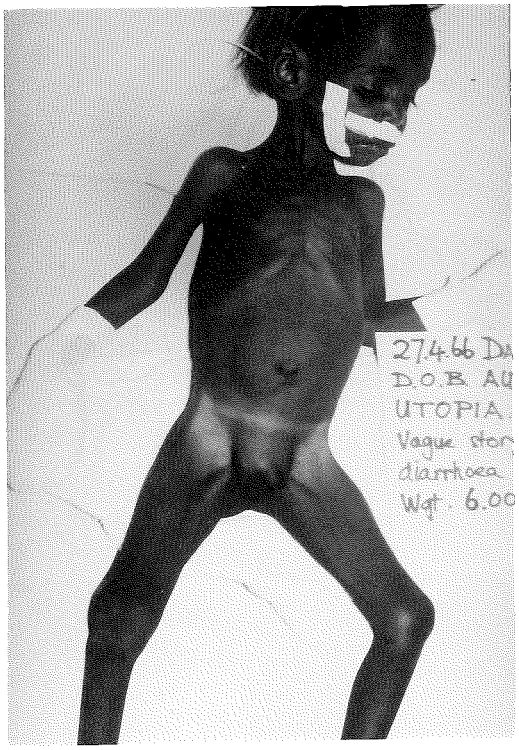
Physical Examination.

The full blown clinical picture of Marasmus could almost equally well be applied to some children with severe congenital heart disease, fibrocystic disease and so on. However the syndrome as seen in Central Australia is sufficiently distinctive to warrant description. Normally anthropometry would be included as part of the

1. Twenty month old boy with severe marasmus.

2. Same child after several weeks of adequate nutrition.

3. Dry, brittle and patchily depigmented hair seen in malnutrition.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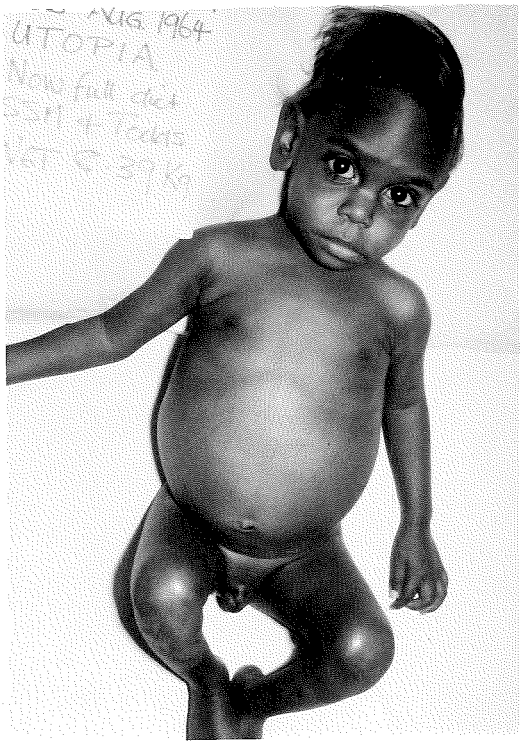
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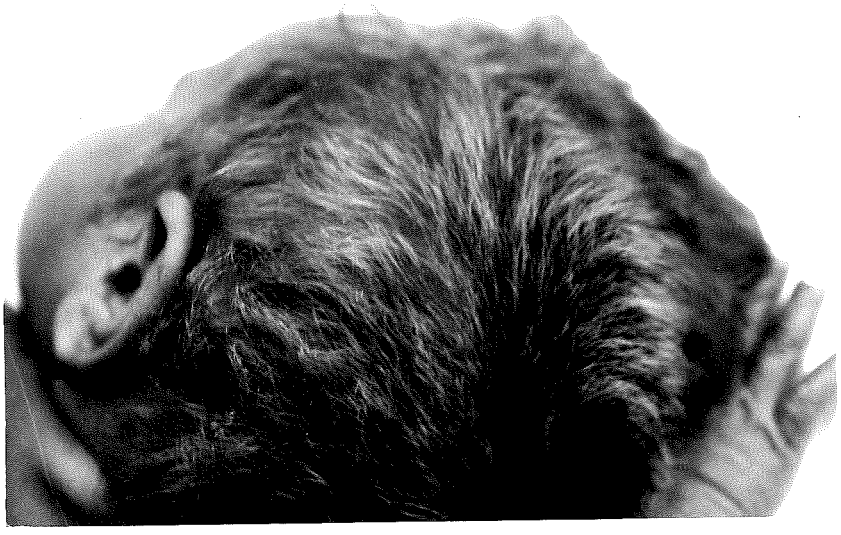

part of the general physical examination, but, owing to its vital importance in this situation, it is discussed fully in the next section.

First impressions, on examination, are of wasted muscles, sunken features and loss of adipose tissue. Oedema is not present although it may develop very easily and rapidly following injudicious intravenous fluid therapy. Often the head appears overlarge in relation to the trunk and the limbs inordinately skinny.

The more obvious signs may be classified as ectodermal, psychomotor and psychological. Gomez et al (1954) described "advanced malnutrition syndromes" which in many ways resemble the situation being described. There is considerable variation regarding which of these aspects appears most prominent, but usually all are present.

a. The Ectodermal signs include dry, wrinkled skin, lack-lustre hair and brittle, cracked nails. The "flaky paint" dermatosis associated with protein deficiency is not seen in the Central Australian children, although rarely a rather immature form of the "flag sign" is seen in their hair. This sign comprises bands of depigmented hair, presumably corresponding to periods of severe dietary lack of protein, and possibly other

nutrients.

One other physical finding, very commonly seen in Aboriginal children and sometimes thought to be a manifestation of malnutrition, is the "Mongolian Spots". These slate-blue, sharply demarcated areas are present over the buttocks and back in over 75% of Aboriginal babies at birth and remain visible for up to twelve months or more. Despite their name these spots have no anthropological significance and are not related to scorbutic or other nutritional bleeding tendencies, or to trauma of any kind.

b. The Psychomotor signs are primarily related to delay in achieving behavioural milestones. There is consistent failure to sit, crawl, stand, walk and talk often until many months after the expected ages. Lack of stimulation is probably partly responsible for this backwardness, especially in long term hospital inpatients. The Aboriginal mothers have no understanding of measuring time, and can never supply ages for their children. The developmental milestones are not eagerly anticipated at certain ages, as they are in the white community. Thus the motivation to stimulate the onset of these skills is partly absent, particularly at what white people feel are appropriate times.

Despite the foregoing it is clear that children with poor growth achievements also lag behind their better fed peers in these other ways.

c. The psychological changes are less specific but non-the-less very real and can contribute additional problems to an already difficult therapeutic exercise. The primary psychological difference between the marasmic and the well-nourished child appears in the attitude to their immediate environment. Unlike the normal infant the undernourished one shows a marked indifference to his surroundings. This is so even in the older children and an occupational therapist's task is made more difficult thereby.

Irritability and prolonged anorexia are other prominent features of the condition which can be considered as psychological changes. The latter is sometimes very severe and may last for many weeks.

The pot-belly is often quoted as a sign of malnutrition. In the children with Inanition in Central Australia the abdomen tends rather to being scaphoid and it is only after some days or weeks of treatment that it fills out and could warrant the description pot-bellied.

Although an autopsy can scarcely be considered as part of physical examination, it is of interest that the liver has invariably demonstrated some degree of fatty change in children who had growth retardation prior to death. It is also true however that these same children had almost always suffered from some form of relatively severe infection, often chronic, and this may have caused most of the liver changes. Some descriptions of Marasmus state that the liver is free of fatty change (Gopolan, 1967).

Anthropomometry and Percentiles.

The need to compare weights and heights with standard charts has been mentioned. In this Aboriginal community it is difficult to define a standard of reference since a statistically adequate, healthy, well-fed group does not exist, and exact standards are impossible to prepare (Kirke, 1969). There seems little point in manufacturing mean growth curves for unhealthy communities unless they be designed only as records of the situation at a point in time.

In the general European community there is a trend towards heavier and taller people which may render old standards obsolete. Overfeeding is becoming a more general feature in the white population, as is improved

infective disease control, so that in view of the rising incidence of obesity, arterial disease and so on, it may eventually become necessary to have "desirable" growth charts (Jelliffe, 1966). At the present time this discussion is rather academic when related to Aboriginal children, since their pressing need is merely for some simple and acceptable growth standards for use in the diagnosis and prevention of malnutrition, in particular, subnutrition.

Although there are no statistically significant groups of elite Aboriginal children, there are scattered individuals and small groups whose environment is superior in some respects to that of their peers. These children achieve "normal" growth patterns according to the Caucasian Standards prepared in London (Tanner, 1958), Boston (Stuart, 1959) and Melbourne (Dale, 1957). Figure III.4 compares the distribution of weights for age, as percentiles, of 48 children, 0-2 years old, who live on cattle stations where the infant feeding is not supervised, with 31 children of the same age whose feeding is overseen by someone with nursing training. In the first group only 8 (17%) of the weights are above the Caucasian Median, whereas in the second, 17 (55%) are above it. The two histograms show a great difference between these groups.

Whether or not, in the unlikely Utopian situation of all Aboriginal children becoming healthy and well fed, their "normal" growth patterns will be similar to the white standards, is immaterial at this time. The fact that some of them can, and do, achieve these standards implies that there is no underlying genetic or biological reason why most of them should not do so. Additional weight to this argument is presented by the fact that the under six month old, fully breast fed group adhere closely to these internationally accepted standards. This suggests, since after six months of age there is commonly a divergence from the growth pattern established earlier in life, that there is an acquired lesion.

In Central Australia the N.T.M.S. Rural Health Section has for some years used infant and toddler longitudinal weight cards with the Caucasian medians marked on them. As many children as possible in remote areas are examined every six weeks and their weights recorded. As an adjunct to these cards, the percentile system has been introduced as a more accurate means of detecting deviations from normal growth patterns. Any child whose weight falls outside the range expressed by the 3rd. to the 97th. percentile is regarded with suspicion, and, certainly if on previous occasions, the weight was within this range, the child is admitted to hospital for

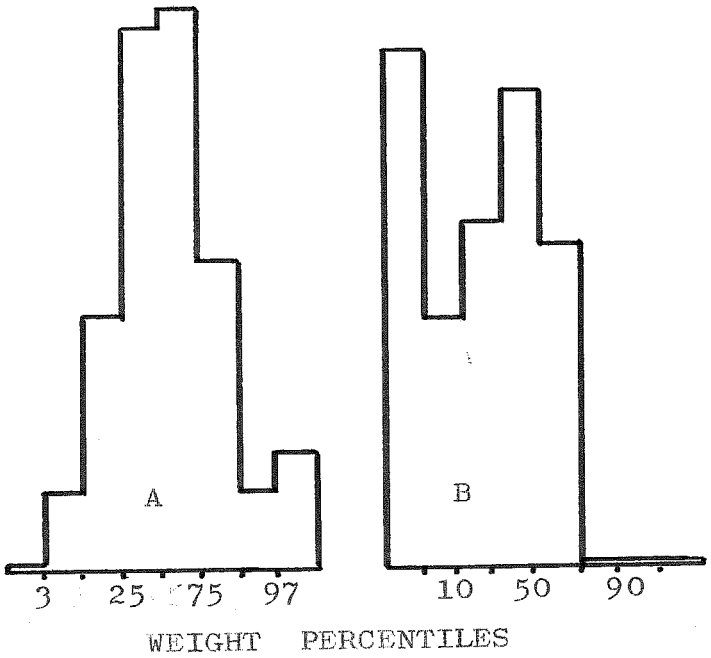

FIGURE III:4 Weight distribution by percentiles of 0 - 2 year old children whose feeding is (A) supervised and (B) not supervised by a trained nurse.

investigation. The 3rd. percentile, which corresponds roughly to 80% of the median value (or to two standard deviations from the mean of the "normal" population), has come to be regarded as the borderline between malnutrition or high risk and better nutrition or lower risk. The exception to this rule of course is the child whose weight is established, say, on the 75th. percentile curve and then deviates to say, the 10th. percentile. He is in as much danger, and is probably as badly nourished, as the one who drops from the 10th. percentile to below the 3rd.

Since this approach has been taken, and the growth range expressed by these standards accepted as desirable for Aboriginal as well as white children there has been a dramatic fall in mortality. It means however that the paediatric wards in the Alice Springs Hospital are chronically over crowded, which in itself provides hazards of cross-infection and so on.

The anthropometric method of screening communities for malnutrition is very satisfactory provided there is strict adherence to the adages that "any child whose weight (or height) deviates significantly below the percentile line on which it fell when last measured, must be further investigated". In this way very few

children with any form of malnutrition are missed.

During the period under consideration a great number of anthropometric nutritional data have been collected. Many of those presented here are from work previously published (Kirke, 1969).

Growth Rates.

The heights and weights of 870 full blood Aboriginal children were classified according to age groups and percentiles using the tables from London (Tanner, 1958). An example of such a chart is shown in Figure III.5. Histograms were drawn to express relative representation in percentile groups. These are reproduced as Figures III.6 and III.7. Figure III.9 is a schematic normal histogram for comparative purposes. It can be readily appreciated that in the first six months of life both height and weight are distributed "normally". However some factor or factors, interferes with the growth pattern established early in infancy and between 6 and 24 months an increasing over-presentation in low percentile groupings becomes apparent. In the 18 - 23 month olds 41.5% are below the third percentile for height and 25.5% for weight. During the ensuing three years there is a gradual improvement, which is due partly to the older child's ability to feed himself and partly to a high mortality in the low weight for-age infants and toddlers.

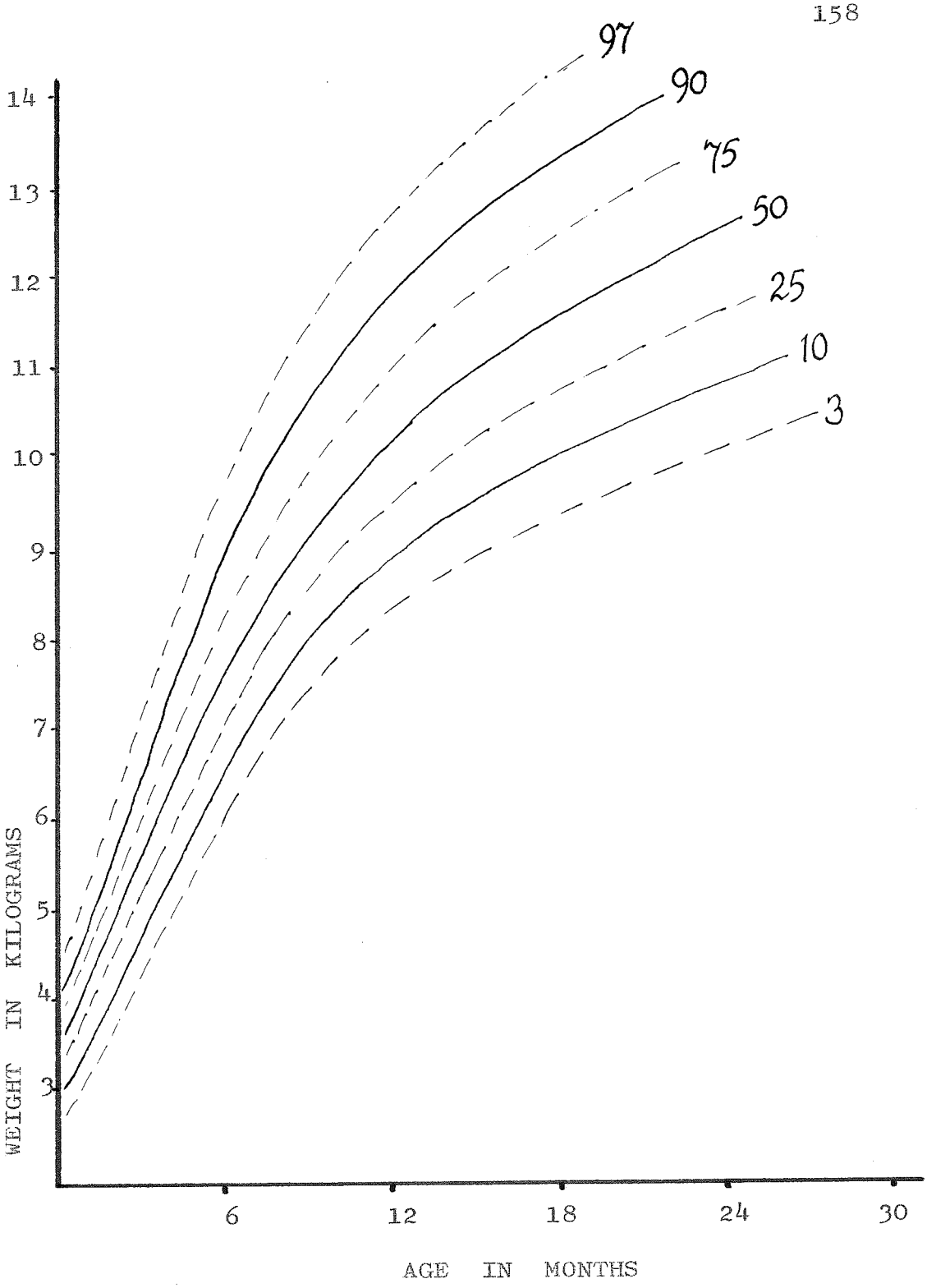

FIGURE III:5 Example of a weight chart with Percentiles.

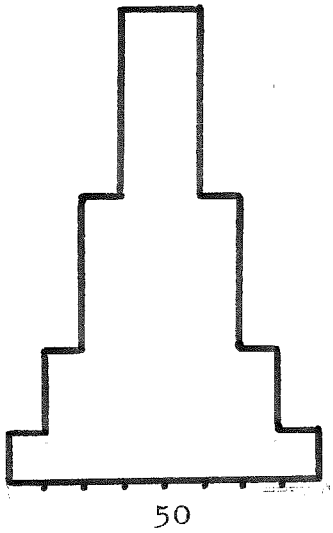

FIGURE III:9 Schematic histogram distributed evenly about a median for comparative purposes.

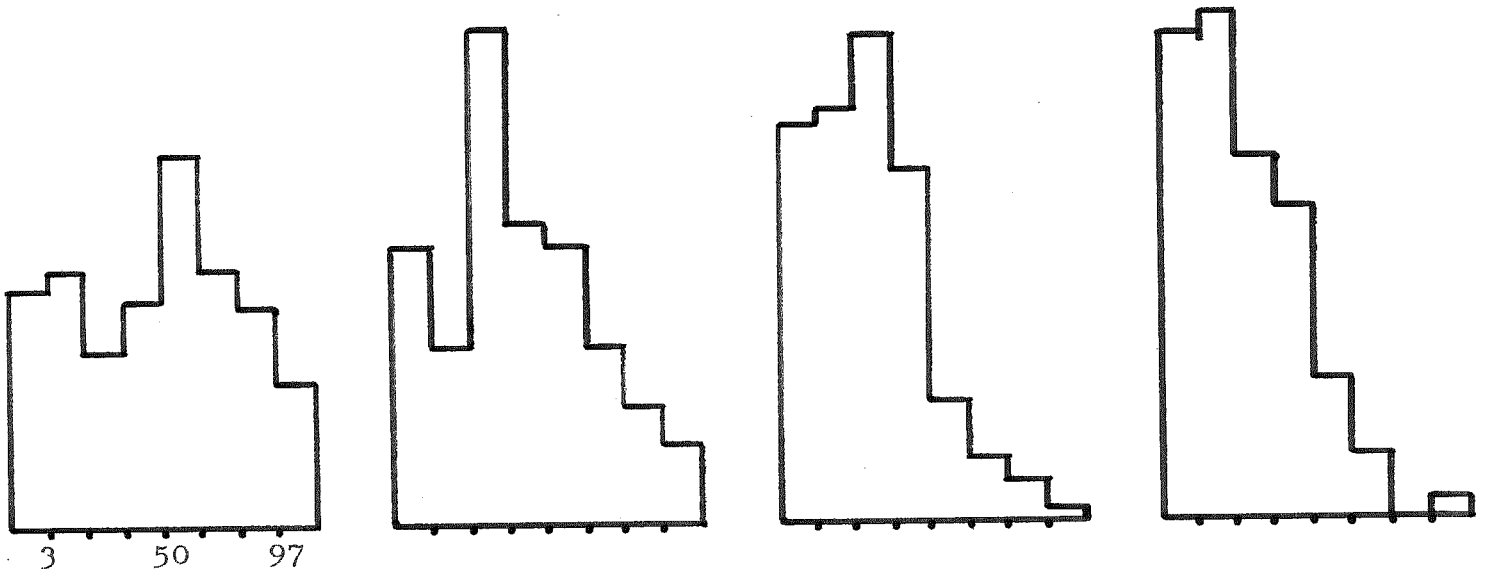

FIGURE III:6 Weight for age distribution by percentiles (0 - 23 Months).

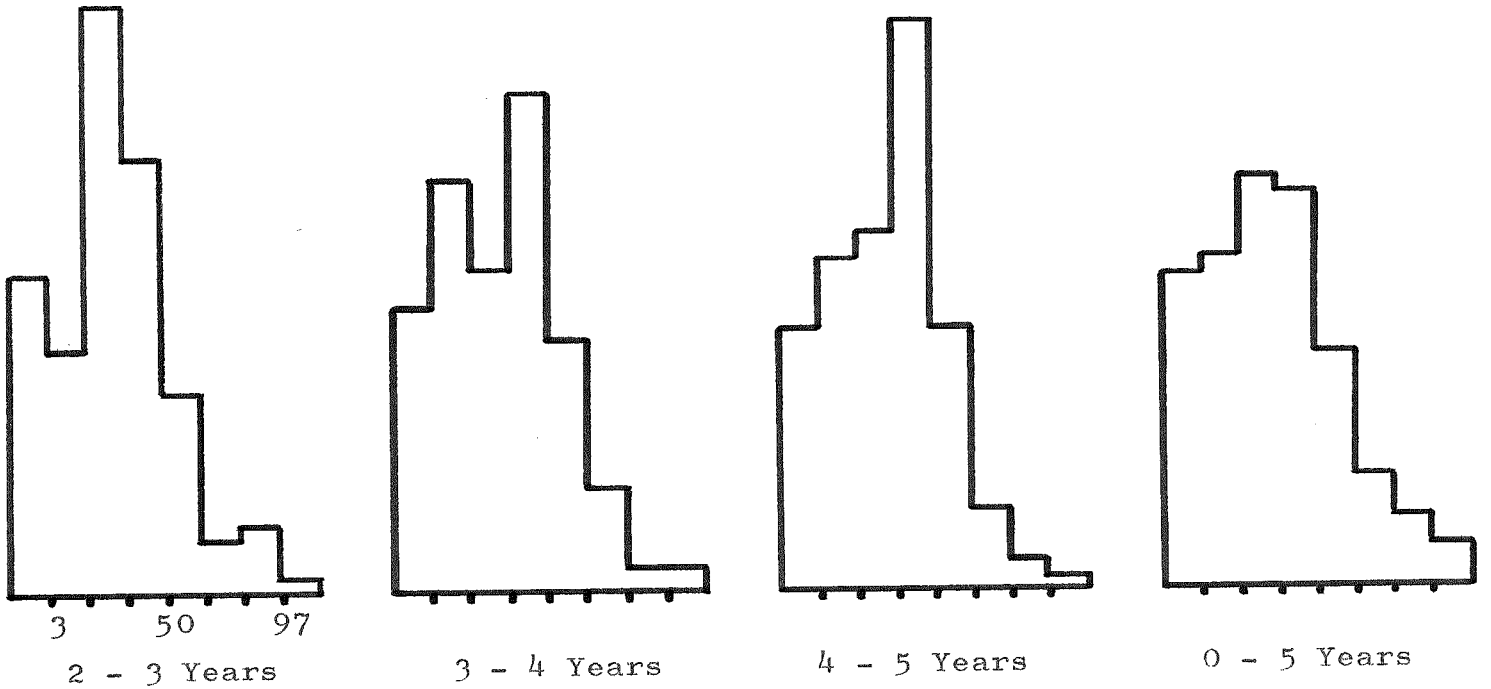

FIGURE III:6 Weight for age distribution by percentiles (2 - 5 Years).

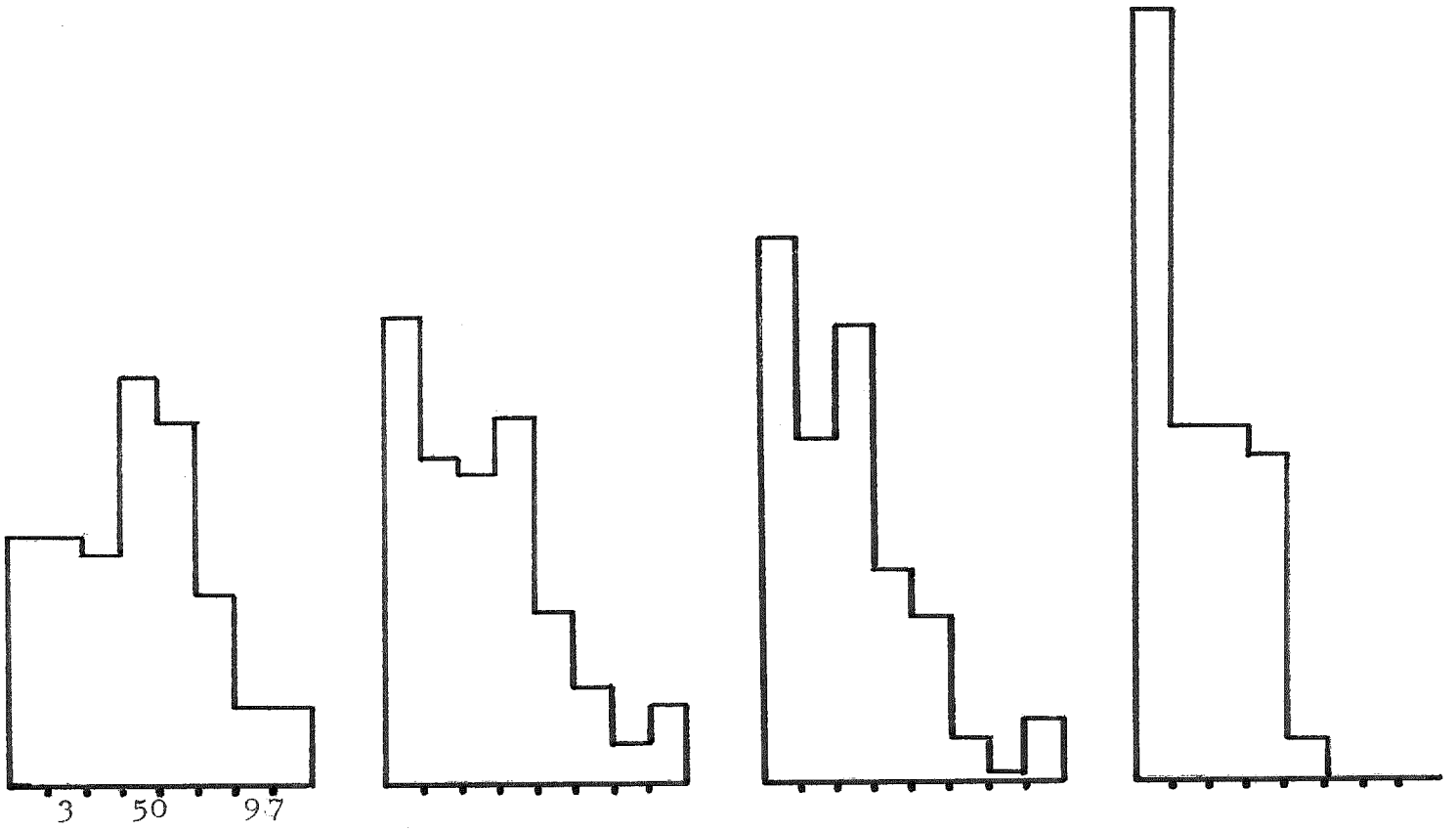

FIGURE III:7 Height for age distribution by percentiles (0 - 23 Months).

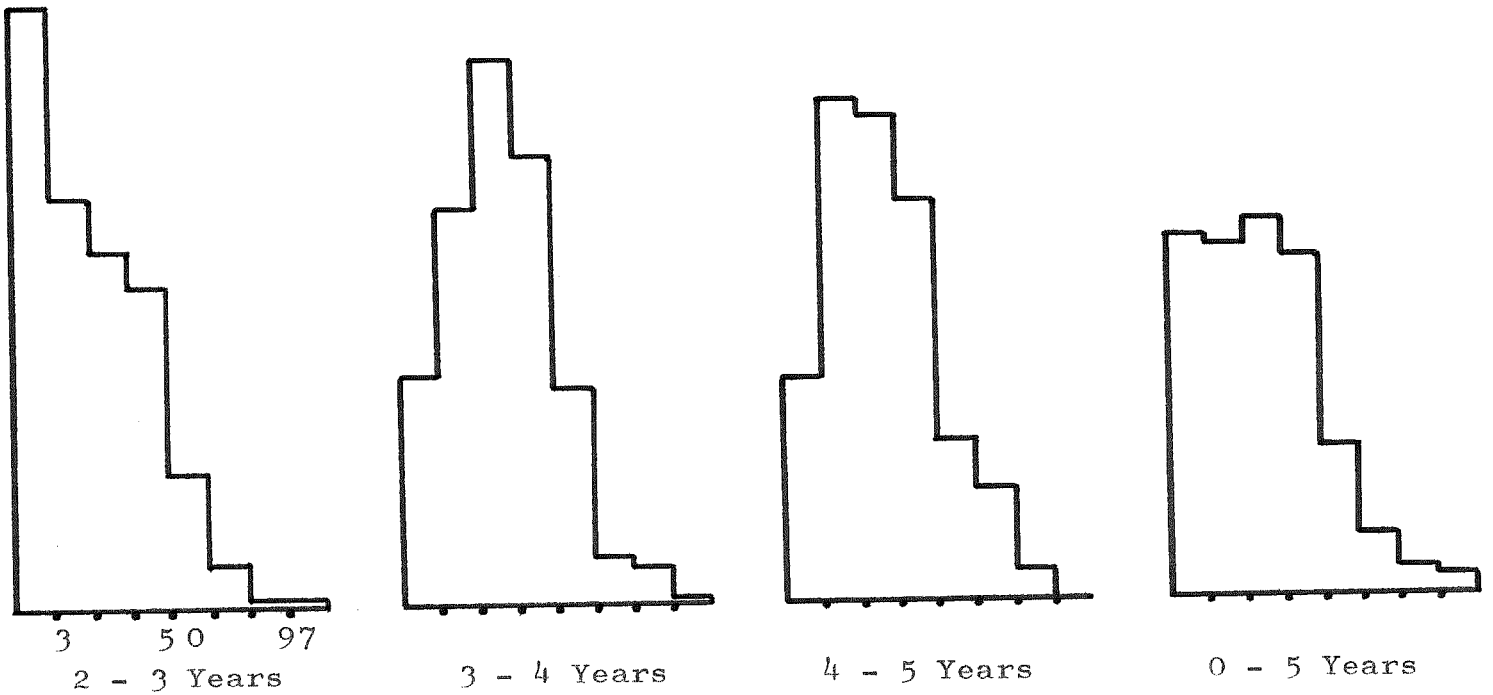

FIGURE III:7 Height for age distribution by percentiles (2 - 5 Years).

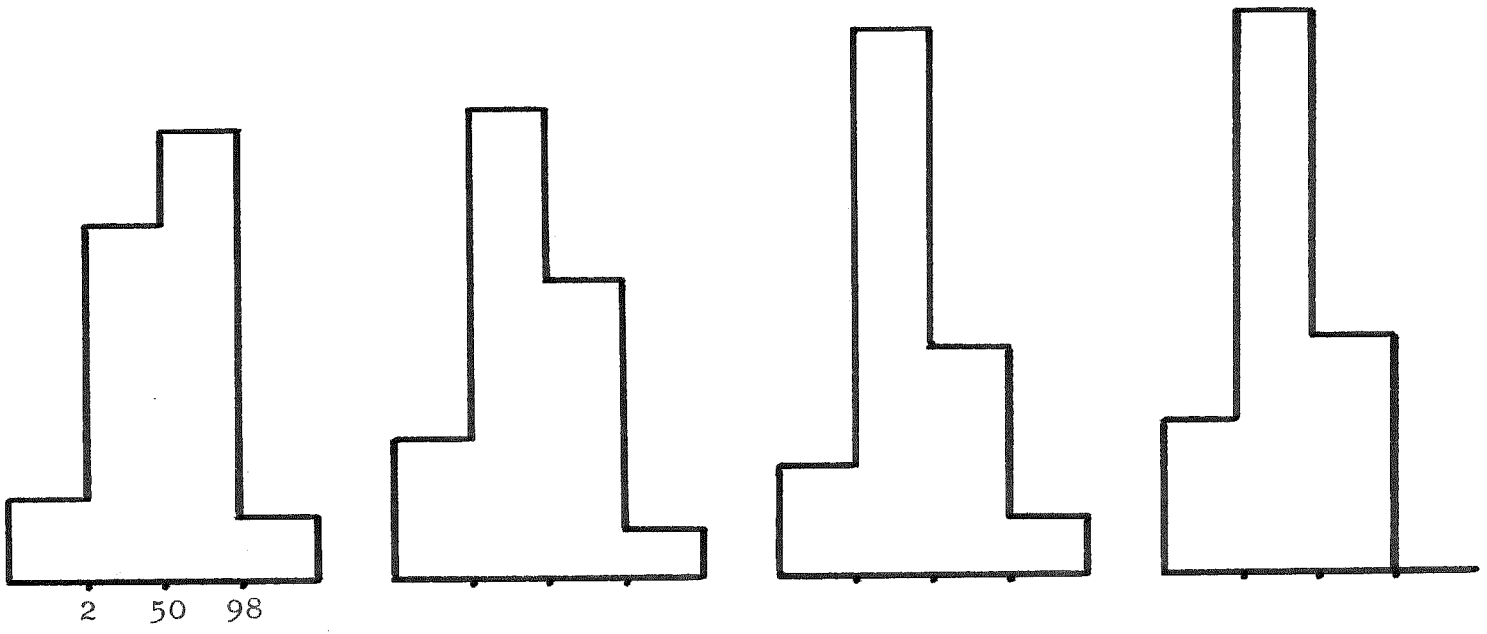

FIGURE III:8 Head-size for age distribution by percentiles (0 - 23 Months).

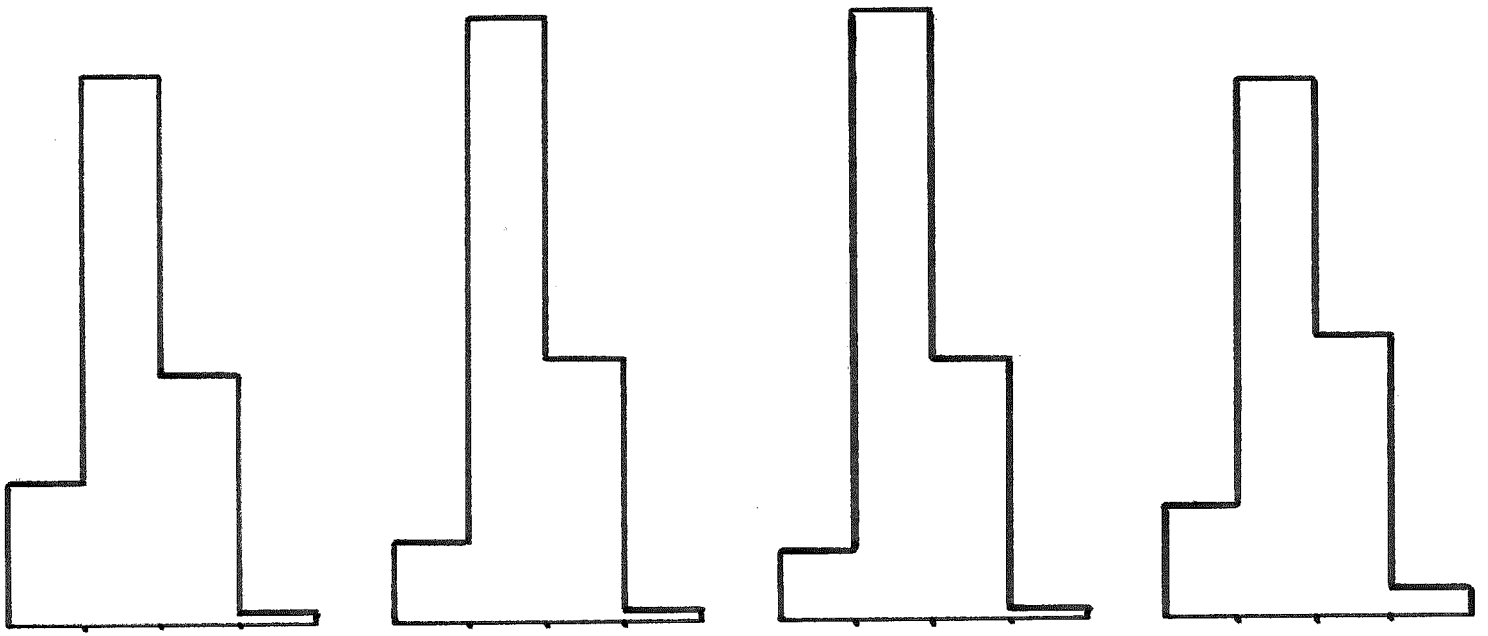

FIGURE III:8 Head-size for age distribution by percentiles (2 - 5 Years).

Throughout these changes the respective histograms for height and weight look very similar in the various age groups.

Head circumferences were measured at the same time as the other parameters and the relevant data are presented, in the same way (Figure III.8) and similar trends are apparent.

The cause of the abrupt and marked deviation from expected growth patterns around six months onward is clearly nutritional. It has already been established that the incidence of the common infective illnesses is high well before the growth aberrations occur. Thus although these diseases may play a part in the production of altered growth patterns it appears that some other factor is primarily to blame.

It is well known that breast milk ceases to be adequate for normal growth after about six months and some sort of supplementary educational diet is necessary. Very few Aboriginal infants receive sufficient, if any, extra food, often until such time as they produce teeth and can manage some adult diet. It is not at all uncommon to see two year old children weighing less than they did at six months (photograph). Further support to the idea that undernutrition is behind the abnormal

growth situation comes from the rapid rates of weight gain of many children in hospital. Figure III:10 shows several of these gains relative to the 50th. percentile line, and it is quite obvious since the children were fed basically only a "normal" white child's diet in hospital that they must have been eating a good deal less at home.

Sometimes Aboriginal women breast feed their children well into the second and even third year of life, and it is probable that the minimal nutrition derived thereby keeps these children alive but not growing. During the study period it was very rare indeed to see an infant, on his home ground, being fed anything but breast milk and an occasional tin of prepared baby food. In some places adequate foods for infant feeding are available but the wherewithal for their preparation and understanding of the need are lacking. Many of the naturally occurring foods, or parts thereof, would no doubt be suitable for educational diets, but there are often cultural blocks which preclude their use. An example of this aspect of the problem is kangaroo brain. Despite its proven value as baby food, it is usually reserved for the old men and even they may eat it only having first consumed the ears and the eyes. It is said also that certain parts of the goanna are sufficiently

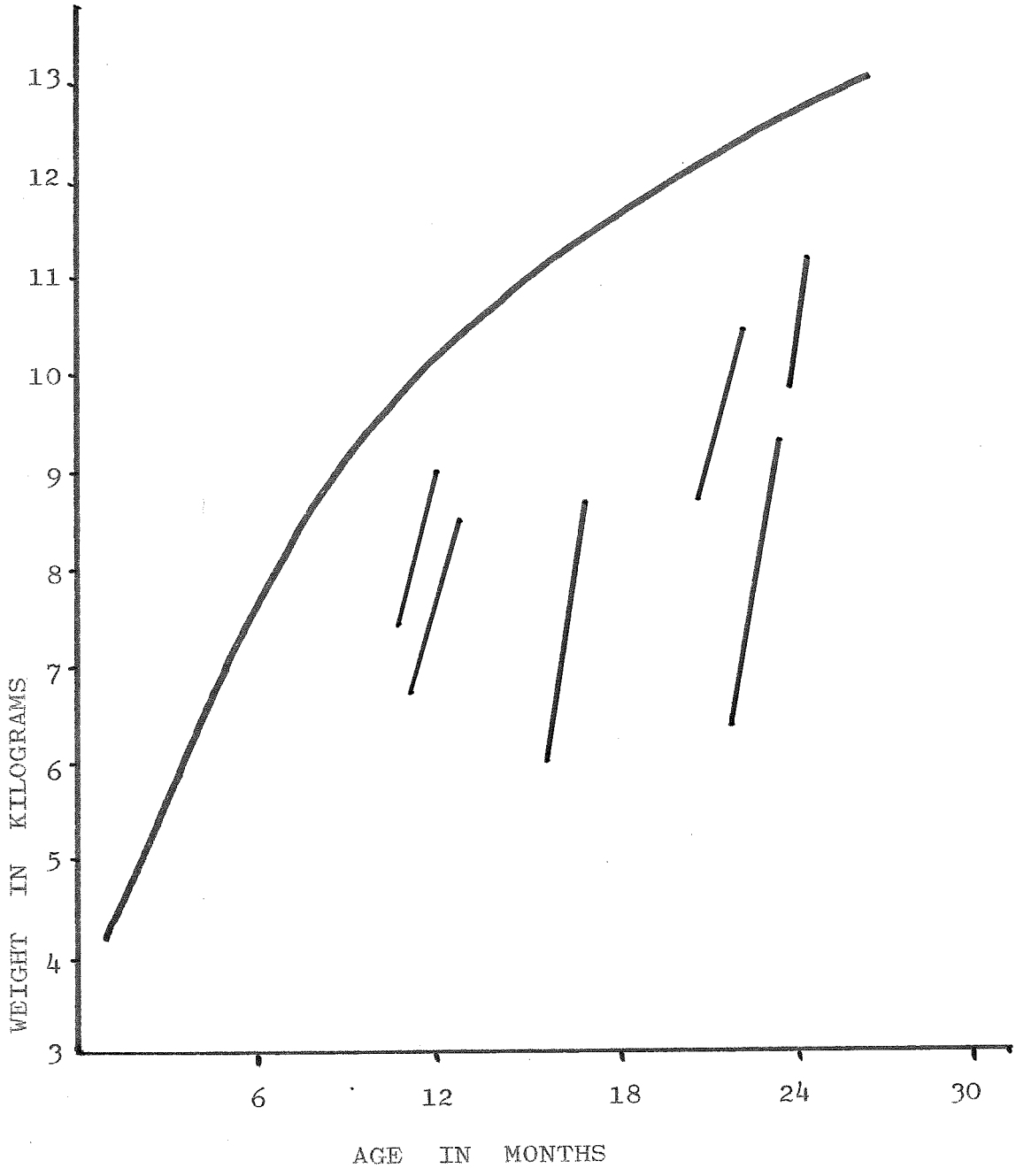

FIGURE III:10 Representative weight gains achieved over short periods by hospital inpatients.

soft and nourishing to have been used for infants, although no evidence of this custom still applying has been found in Central Australia during the last five years.

The most important basic consideration in the field of infantile malnutrition is the lack of appreciation by the mother of relative food values and the need for supplementary diet in infants. Even the adults themselves regard a full stomach as indicating an adequate feed irrespective of the type of food consumed. This means that the sorts of diet available for small children in the camp situation consist largely of cheap, easily prepared and filling things like bread, damper, tinned beef, jam and tea. Vast amounts of white sugar are consumed in black tea by both adults and children. This sort of diet may be supplemented to some extent, depending on geographical and other factors, by proteinaceous "bush tucker" some of which could be of value to small children but its availability is inconsistent and its preparation inappropriate in the extreme. In places where the mothers can be encouraged to use high protein, prepared baby food the problems are less severe.

Apart from any other considerations, the earning power of an average Aboriginal family is insufficient to

provide for proper food and clothing, even if the parents had the knowledge and motivation to make such provisions.

There seems little possible doubt that the high incidence, particularly among some geographical groups, of physically stunted children is a direct measure of the level of undernutrition.

Incidence of Poor Growth.

There is now a great deal of evidence to suggest that malnutrition, as assessed by growth parameters, occurs amongst Aboriginal children throughout the Commonwealth (Jacobs, personal communication; Jose, 1969; Kettle, 1966; Kirke, 1969; Maxwell, personal communication; Propert, Edmonds and Parsons, 1968).

These studies have produced figures from the points marked on the map (Figure III.11). The authors have presented their data in different ways but despite the wide variations in location and reported infective disease spectra, there is a notable consistency in the incidences and degrees of growth failure. The greatly diminished growth rate after six months of age is common to all these studies and there is general agreement that its cause is undernutrition.

Table III.30 shows the percentage of Central Australian children with weights, heights, and head circum-

ferences below the 3rd. percentile. The children up to five years were examined in 1968 and the other ones in 1969.

TABLE III.30 Incidence of Low Percentiles

AGE	PERCENTAGE LESS THAN 3RD. PERCENTILE			NO. EXAMINED
	WEIGHT	HEIGHT	HEAD SIZE	
0-5 months	13	14	9	118
6-11 "	15	25	13	96
12-17 "	21	30	13	110
18-23 "	26	42	16	79
2-3 years	17	31	15	185
3-4 "	15	12	9	159
4-5 "	14	12	8	119
5-15 "	10	5	5	560
0-15 years	14	13	9	1430

Similar overrepresentation in the low percentiles occurs in the three parameters, and in each it reaches a peak in the 18 - 23 month old group. These high percentages would have been even higher had not the enormous infant mortality affected mainly children in the lowest percentiles.

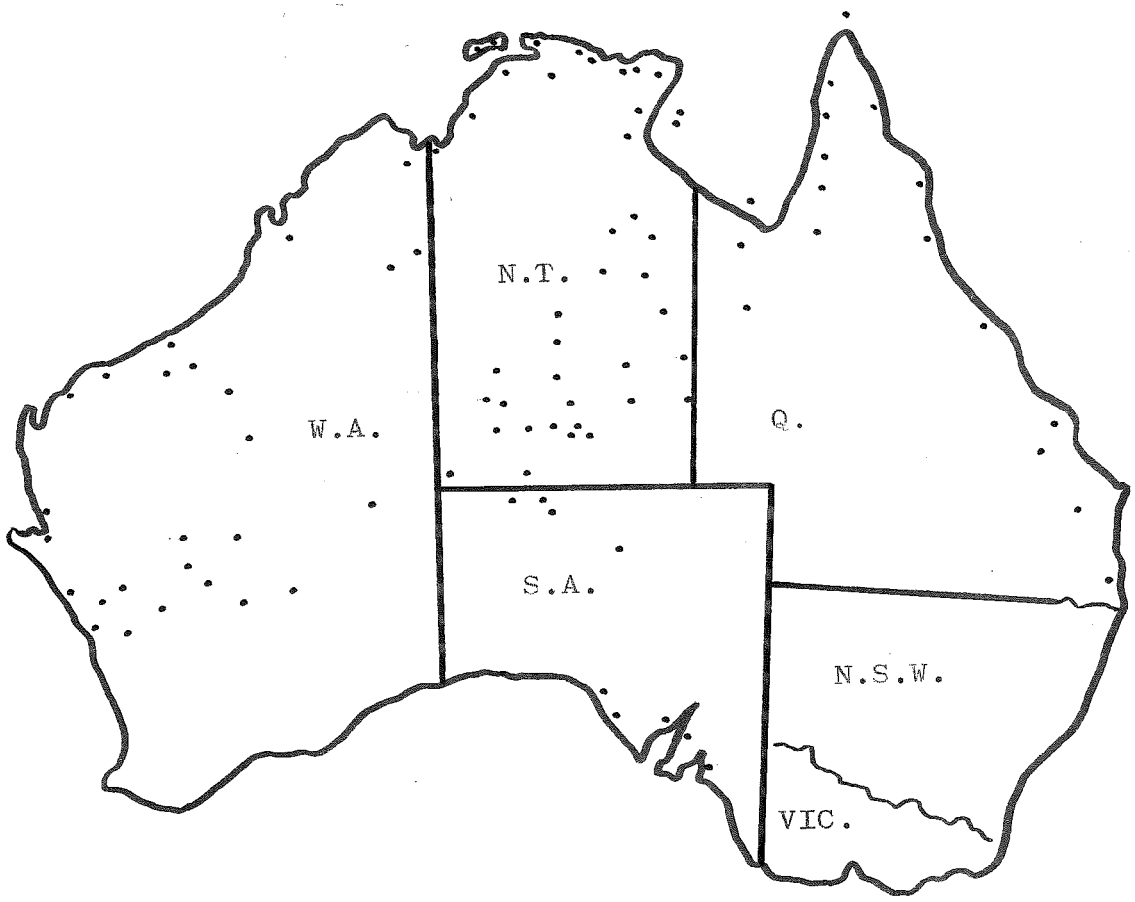

FIGURE III:11 Areas in which Aboriginal childhood growth patterns have been investigated by various workers during the period 1964 - 1969.

The marked improvement in each parameter from the age of two years onward is paralleled by steep decreases in the incidence of infective diseases (previously discussed) and death rate. Thus selective mortality has little to do with the trend of growth parameters towards more acceptable levels in these older children.

At two years these children generally have enough teeth to cope with a diet of meat, vegetable, fruit and so on prepared for adult consumption. Roughly at the same age independant mobility is established and the individual becomes more articulate in his demands for food. These factors all make for improved nutrition.

Height is a more sensitive index than either of the other parameters, since the percentage of heights less than the 3rd. percentile rises and falls much further and more rapidly. However when considering an individual child, weight serves as a better criterion of health as it is much easier to measure, and it reflects relatively acute illness immediately. For example it is far more valuable, clinically, to know, at the time of examination, that a child has lost 1.5 Kgm. than to wait three months to see if he has stopped growing in length or head size. This example again stresses the importance of maintaining longitudinal weight records.

The huge change in representation of low heights for age is related to retarded bone development in infants and toddlers. These bony changes are discussed in the next section, since they represent another avenue of nutritional assessment.

Radiography.

Greulich and Pyle (1959) state that radiographic study of selected skeletal areas provides a more reliable assessment of a child's development than can be derived from height, weight and age.

It might be argued that calcium lack and/or protein deficiency generally produce the bony change, which therefore should be more properly constitute a specific syndrome and be considered as such. Many other factors can produce retarded bone growth and bony immaturity, including simple undernutrition and severe infection, so this subject is included under the broader heading.

Unfortunately the maturation of the skeleton is a more valid guide to an older child's development than to that of an infant. However, despite the relatively wider physiological range in the bones of normal infants, there is some correlation between retarded bone age, height age and weight age, in malnourished babies. These ages are calculated from a standard growth chart. For example a

child weighing 3 Kg. would have a weight age of 7.5 months since at this age, 8 Kg. falls on the 50th. percentile line.

Radiography of various skeletal areas as a screening procedure for infantile malnutrition in remote areas has serious limitations. The machinery and other equipment such as generators, dark rooms and spare casettes are difficult to render sufficiently mobile. Thus such a profect, especially since it would have to be frequently repeated, becomes very expensive. The technique is, of course, used during inpatient assessment and the results of a study of one group is reported in this section.

In addition to radiological developmental criteria, which depend largely on the appearance of bone growth centres, other information is often available. The changes of Rickets and/or Scurvy may be apparent, but of more general significance are poor mineralisation and lines of increased density, often seen in films of young children with nutritional deficiencies.

During 1968, 34 children had radiographs taken of appropriate areas, usually knees and wrists, in order to assess bony development and the incidence of deficiency disease. The ages of this group ranged from six months to eleven years.

In general the films were within normal limits, but a few of the older children had slight widening of the epiphyseal plates of the distal femur and proximal tibia and of the epiphyseal zones of the distal radius and ulna. These findings suggested mild Rickets but were only minimally outside the normal ranges (McCoy, 1970; personal communication).

Many of the films of the younger children showed transverse lines of increased density, or growth arrest lines, in the metaphyses of the same bones. These presumably represented episodes of intercurrent illness be it nutritional or infective. The lines are said to appear particularly commonly during re-alimentation of malnourished children. Several radiographs had the appearance of generalised loss of bony density, and in some there is mild white line formation at the metaphyses. This brought to mind the possibility of early Vitamin C deficiency, although no example of florid Scurvy occurred in the whole series.

The bone ages of these children (Greulich and Pyle) were calculated and compared to chronological age and the anthropometric ages already described. Figures III.12, III.13 and III.14 show quite clearly that there is a closer relationship between bone age and chronological

age or height age than a composite anthropometric age comprising a third of the sum of the height, weight and head ages.

Although the growth arrest lines were very common in the films of the young children, they were notably absent in the older group. This suggests that either the diseases causing the appearance of such lines were not so common seven to ten years ago or that the lines tend to disappear with increasing age. Garn, Silverman, Hertzog and Rohmann (1968) indicate that once present these lines tend to persist into adult life. It may be that this situation is explained by the previous high mortality and the small size of this series.

No relationship could be discovered between retarded bone age and the incidence of lines of increased density.

Radiography of developing bony areas then in general provides a good deal of information and may virtually exclude some specific deficiency syndromes. As a means of assessing physical development it seems that simple height measurements provide much the same index as bone age, and so once again there is no place for using the rather complicated radiographic techniques in remote areas.

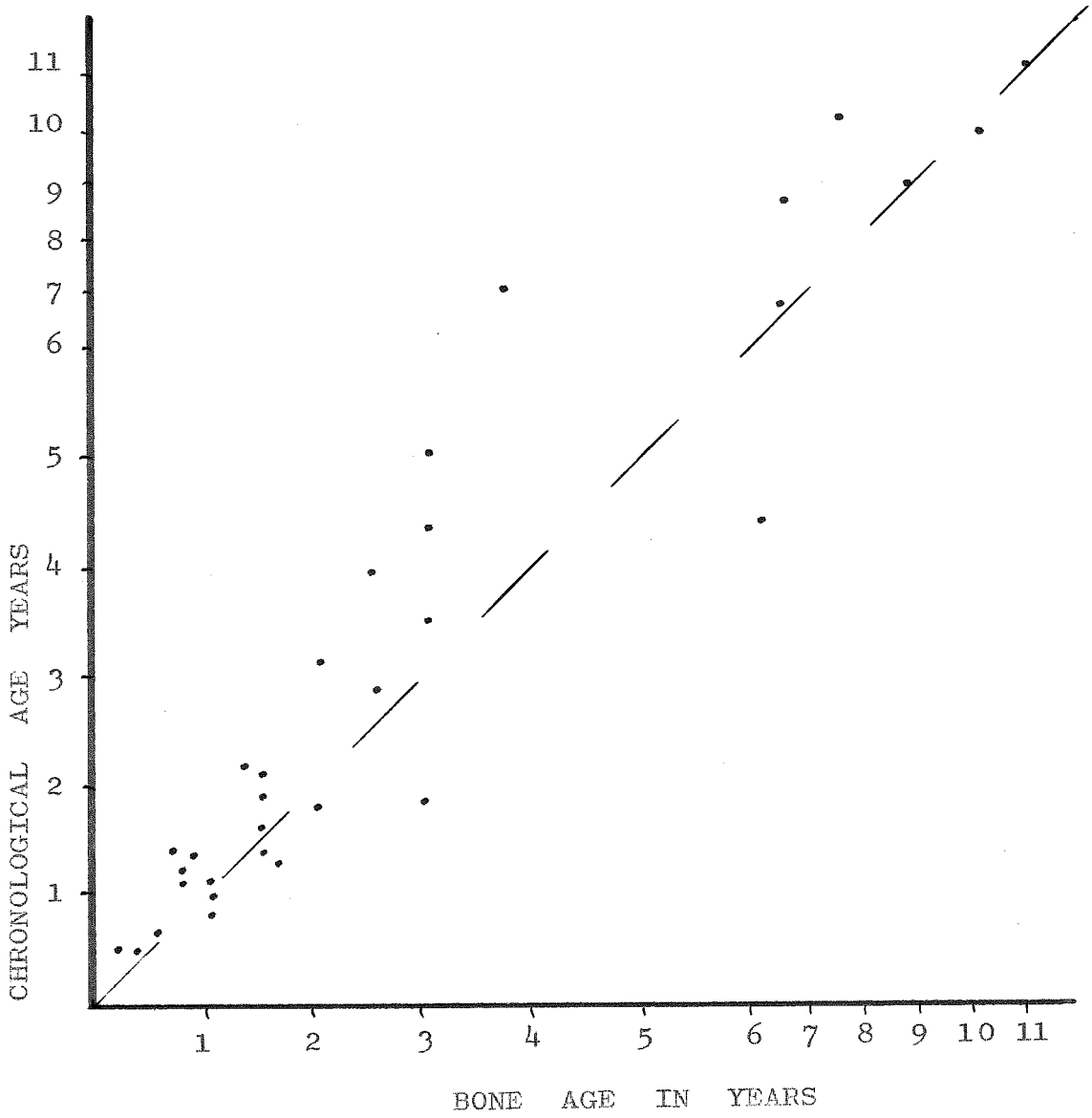

FIGURE III:12 Relationship between Bone Age and Chronological Age.

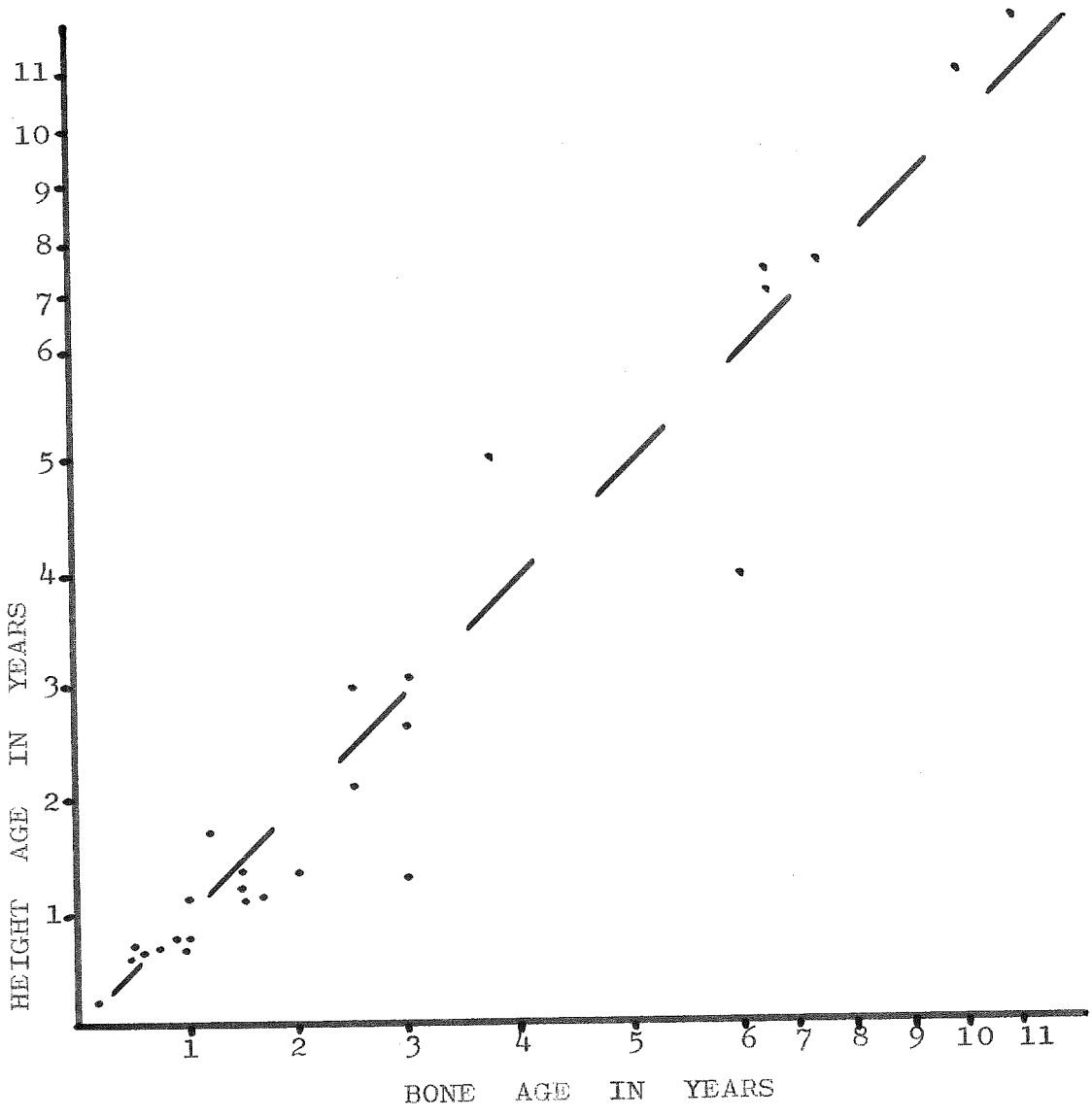

FIGURE III:13 Relationship between Bone Age and Height Age.

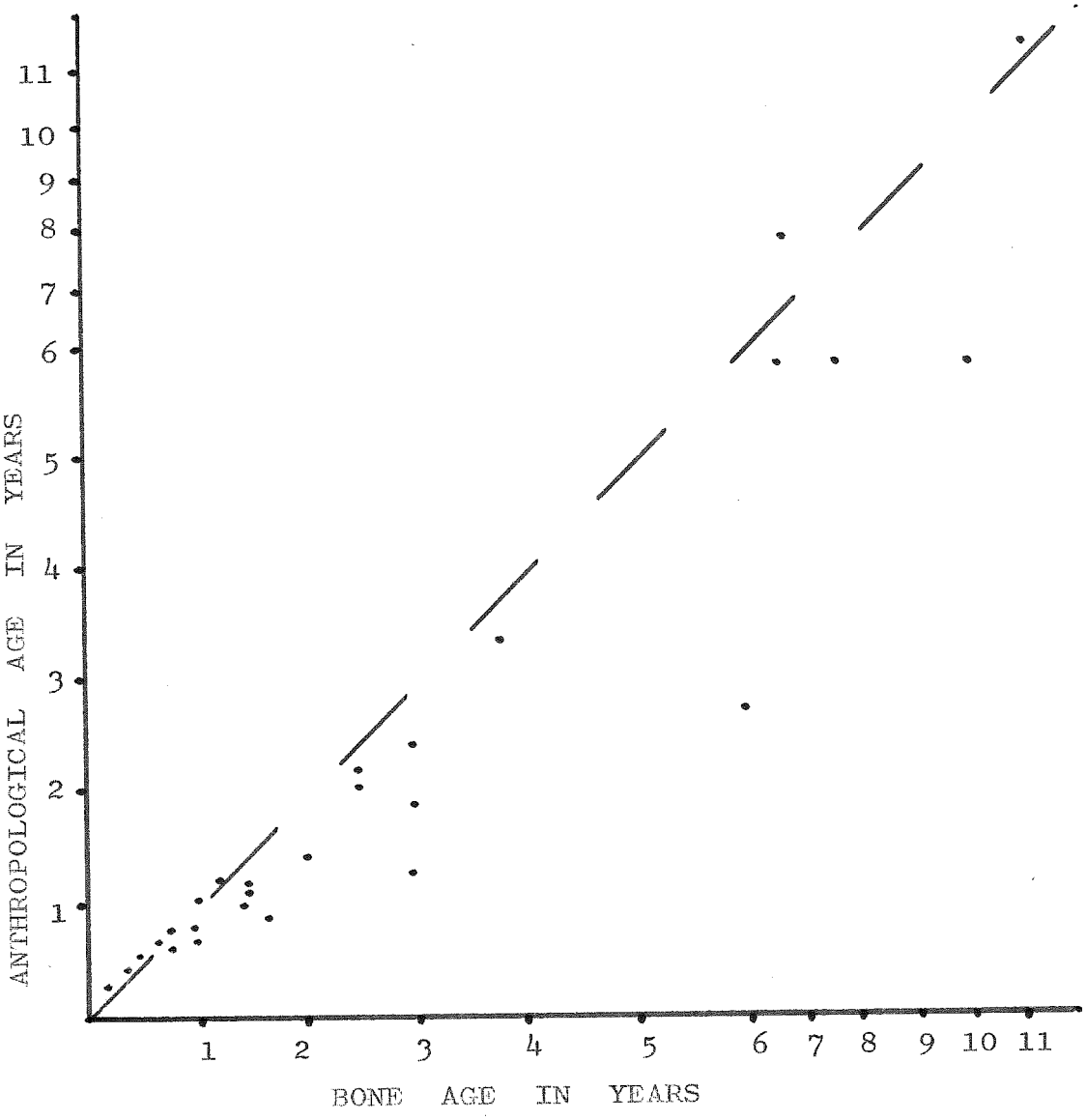

FIGURE III:14 Relationship between Bone Age and an Anthropometric Age based on height, weight, head circumference and chronological age.

Cytology.

The differental uptake of stain by buccal epithelial squames has been used as an indicator of poor nutritional status due to protein-calorie deficiency (Squires, 1965). The technique depends on the distinctive colours exhibited by cornified and non-cornified cells when exposed to certain stains, and that the proportion of non-cornified cells has a relationship to the level of protein-calorie intake.

As a screening method in nutritional surveys, this technique, since it involves arduous, although relatively unskilled, staining procedures and microscopy, is too time consuming to be of practical value in remote areas. However to test its relevance to the local situation, 24 smears were taken from a random group of children at Papunya in 1968. The age range of these children was 4 - 90 months and relevant anthropometric data was collected at the same time. This study was preceded by several weeks of perfecting smearing and staining techniques and tediously repetitive cell counts to ensure sufficiently reproduceable results. The staining method used was a single stage process described by Gurr (1962), omitting the preliminary haematoxylin which the original "Shorrs' Stain" (1941) for vaginal smears included.

The young non-cornified cells stained green throughout, and as the cell aged the nucleus became darker. The cytoplasm, initially pale, clear green, was quite often stippled, and as the cell aged it became orange-brown. Eventually the nucleus faded and the cell disintegrated. Squires (1965) claims that cytoplasmic stippling is seen in smears from marasmic patients, and that the squames mature more rapidly than normal in cases of protein-calorie deficiency. Thus the proportion of young, green stained cells is lower in poorly nourished children.

During counting, the nuclear colour of between 200 and 500 cells was noted, and an assessment of the incidence of cytoplasmic stippling made. Only single cells, and clumps of up to three cells, were included. Each slide was counted three times and the figures reported are the mean of the three.

Table III.31 lists the percentage of non-cornified cells in relation to the presence or absence of significant stippling, and the weight percentiles of the 24 Papunya children.

In this series no relationship could be found between low weight percentiles and a decreased number of non-cornified buccal squames. There was however an obvious, but inconsistent, association between low weight

percentiles and the presence of stippling in the cytoplasm. Even allowing for discrepancies in staining technique, variable time intervals between fixing and staining and probably inconsistent smearing it appears that Buccal Smears do not have a potential application to nutritional screening in Central Australia.

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TABLE III.31 Buccal Smears and Percentiles

PATIENT NUMBER	% NON-CORNIFIED CELLS	STIPPLING OF CYTOPLASM	PERCENTILES
1	3.8	x	Less than 3
2	5.4	x	" 3
3	11.0		3 - 9
4	69.4		10 - 24
5	32.2		25 - 49
6	10.8	x	3 - 9
7	4.8		90 - 96
8	13.8		25 - 49
9	11.6		50 - 74
10	2.0	x	3 - 9
11	1.8		3 - 9
12	0.8	x	25 - 49
13	1.0		3 - 9
14	0.2	x	10 - 24
15	1.0		25 - 49
16	2.5		50 - 74
17	0.6		50 - 74
18	1.0		10 - 24
19	55.6		Less than 3
20	30.6	x	3 - 9
21	31.8		Less than 3
22	21.5		3 - 9
23	4.2	x	Less than 3
24	12.6	x	Less than 3

SPECIFIC DEFICIENCIES

The point that most of these specific deficiencies can be used as indices of general nutrition has already been made. However, since they produce specific syndromes which do not necessarily directly affect body mass, they are considered as distinct lesions.

Haemoglobin.

Dietary iron deficiency is the commonest cause of significant anaemia in Central Australia whereas, further North, Hookworm infestation takes precedence.

The common haematological picture in the infants and young children is one of a relatively mild, hypochromic, normocytic or microcytic anaemia associated with a normal or slightly low haematocrit level, Usually the erythrocyte count is within expected limits. The sedimentation rate is often high, which phenomenon has been found elsewhere in asymptomatic Aboriginal Children (Jose, 1969). In the group under consideration various infections, anaemia and radiological lung disease were so common that raised sedimentation rates would not be unexpected.

Laboratory facilities in Alice Springs did not allow for such estimations as Serum Iron and Iron Binding capacity, however during 1966, 16 sera from children with haemoglobins below 10gm. per 100 ml. were sent to

Adelaide for Iron assay. Each was reported as containing significantly less than 50mcg. of Iron per 100 mls. of plasma. Since hypoproteinaemia is uncommon, and serum Transferrin levels are generally normal or even a little high (Penfold, 1969, personal communication), the possibility of some sort of protein losing enteropathy causing iron deficiency is remote. Even the children with quite severe anaemia have low reticulocyte counts, which suggests a depleted iron store

Generally the patients with low Haemoglobins respond rapidly to Iron administration and produce a marked reticulocytosis, although very often much larger doses of Iron than those recommended for well nourished Caucasian children are necessary. Presumably a large proportion of the Iron administered goes to replenishing body stores. Certainly children recovering from malnutrition, particularly those on specialised diets, have a greater demand for Iron than normal. It is common to see a child, admitted to hospital with marasmus and a relatively high haemoglobin level, become progressively more anaemic as his nutrition improves. This phenomenon has been reported from Uganda in children with Kwashiorkor (Trowell and Simpkiss, 1957), although in their case blood transfusion is often necessary. The children in this situation require enormous amounts of parenteral iron, frequently more than five times

the dose recommended by the manufacturer from the beginning of their treatment.

In remote areas there is great difficulty in ensuring adequate dosage with oral iron preparations and the tendency is to use iron dextran (Imferon) by intramuscular injection instead.

Occasionally Folic Acid deficiency occurs and complicates the situation. This again is a dietary factor and oral Folic Acid given concurrently with the Iron is effective in correcting the anaemia. The main problem being of course, to convince the parents of the child that he needs the "little yellow pills" for many months, and thus prevent a recurrence.

The folate deficiency is usually suspected when two adequate courses of intramuscular iron have failed to achieve satisfactory results. Normally folic acid is given arbitrarily in this situation, unless the patient is in hospital, when a blood film may show some macrocytosis to support the diagnosis. Again in 1966 some blood specimens were sent to Adelaide at various times for estimation of folate activity and vitamin B12 levels, as part of the work up in five cases of macrocytic anaemia. Bone marrow examinations were also performed. The results in all five cases were conclusively in favour of folic acid

deficiency, and there was no evidence of B12 lack. This is a very small series, but similar pictures have emerged in several patients sent to the Adelaide Children's Hospital (Inc.) for primary conditions other than anaemia.

Other causes for anaemia have been seen from time to time. Probably the most significant cause, after Iron and Folic Acid lack, in severe and/or chronic infection. Generally this is an Iron deficiency anaemia too although haemolysis can occur in some streptococcal diseases and septicaemias. The high incidence of chronic lung disease and other infections in remote areas has been discussed. Even in the areas with the highest infection rates the anaemia can be controlled by administration of iron, so possibly dietary restrictions are as important as infection itself in the production of low Haemoglobin levels.

Incidence of Anaemia.

A Haemoglobin level of less than 10gm. per 100mls. of blood is taken to represent a significant anaemia in children three to fifty-nine months old. This degree of anaemia in a baby under six months suggests inadequate Iron stores at birth and probable maternal anaemia. In fact this problem is quite common, especially in certain areas where antenatal care is non-existant.

In 1968 routine haemoglobin surveys in rural areas

and treatment of anaemia with intramuscular iron-dextran were introduced. During that year some places were examined for low Haemoglobins regularly and sufferers adequately treated. In other areas the treatment followed only a single Haemoglobin survey and in others again there was no treatment during that year and the incidence of anaemia actually rose somewhat. The Haemoglobin estimations were carried out in the field using "Spencer" haemoglobin-meters (American Optical Co. Catalog 1000D), by members of the N.T.M.S., Rural Health Section.

The results of Haemoglobin surveys, twelve months apart, in the three areas mentioned above, are reported in Table III.32. In 1967, 624 children were examined and 176 (28.2%) found to be significantly anaemic. In 1968, following routine Iron therapy in some areas, of 668 children 113 (16.9%) were anaemic. At one particular place, Areyonga Settlement, over the twelve months the incidence of low Haemoglobins fell from 21.4% to zero, merely due to routine Iron therapy with "Imferon" and "Plesmet" (Ferrous Succinate).

.....

TABLE III.32 Incidence of Anaemia and Response to Routine Iron Therapy.

AREA	1967		1968	
	NO.	% ANAEMIC	NO.	% ANAEMIC
Fully treated	238	31.9	273	5.7
Treated once	189	30.7	188	15.4
Not treated	198	21.2	207	33.3
TOTALS	625	28.2	668	16.9

The improvement following Iron is obvious from the table. It seems that in a totally untreated community the incidence of significant anaemia in this age group would be over 30%. The incidence is continuing to fall with more intensive treatment and, near the end of 1969, in most areas it was below 10%. All the figures for 1969 are not available but so far the overall incidence is 7.3%, which is roughly half that of 1968, which in turn halved the 1967 figure.

The percentage of anaemic children in various age groups shows a picture similar to the ones produced when physical parameters are related to age. The highest percentage occurs at around twelve months and is related to dietary Iron deficiency. Figure III.15 shows the in-

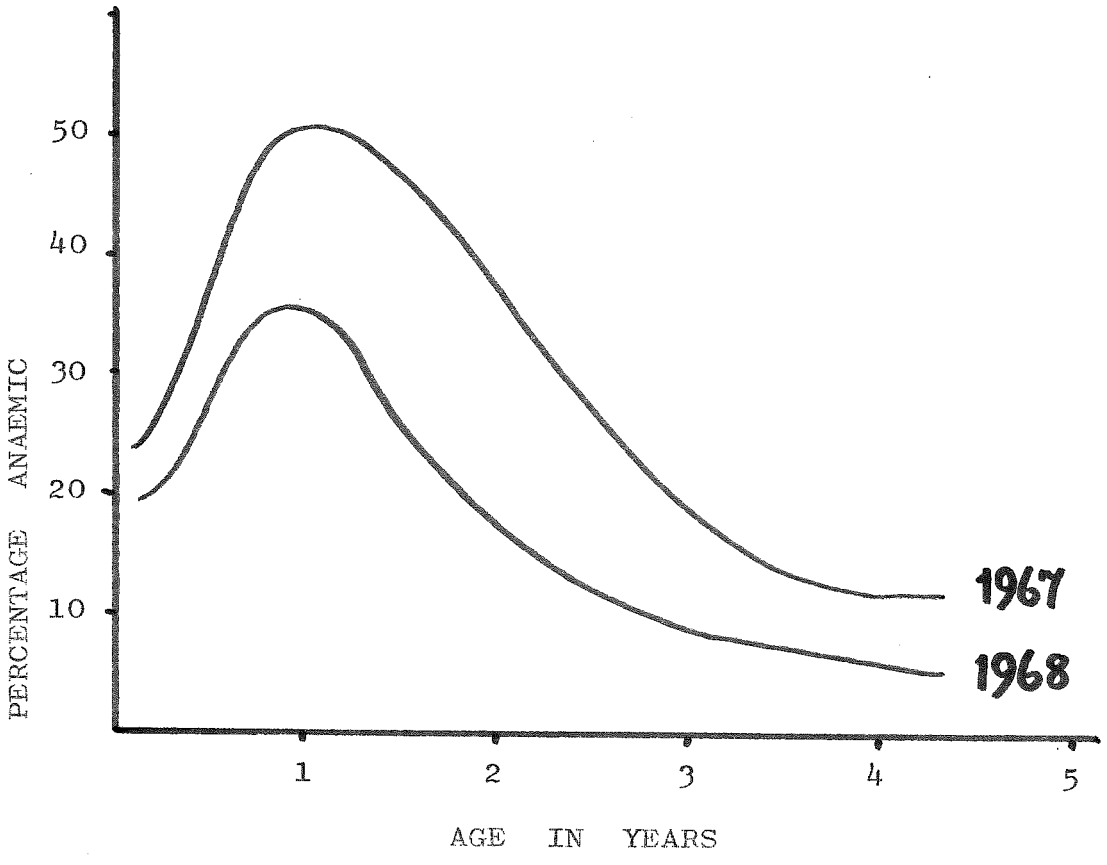

FIGURE III:15 Incidence of Anaemia (1967-1968).

cidence of anaemia during 1967 and 1968. Once again, as the individual child develops, and produces teeth, at eighteen months or two years, he is more able to eat iron containing foods, particularly red meat, and the chances of his being anaemic lessen.

Haemoglobin estimation, before the widespread use of dietary Iron supplements, was probably a worthwhile index of general undernutrition since almost all anaemia is this area is dietary in origin. Now, however, where iron is used freely, children may be grossly undernourished and yet have a normal Haemoglobin. They may become anaemic if for some reason their nutrition is improved, but for nutritional screening in remote areas, despite its simplicity, Haemoglobin estimation is of little value.

Ascorbic Acid (Vitamin C).

Serum Ascorbic Acid estimation is, with Haemoglobin, serum protein and so on, amongst the category I. tests recommended by W.H.O. for use in general nutrition surveys to supplement clinical examination and physical methods (W.H.O., 1962). Although florid Scurvy is rare amongst Central Australian Aboriginal children the clinical picture exhibited by some of these marasmic infants has much in common with this syndrome. It is well known that infection increases the body's demand for ascorbic acid and diarrhoeal disease interferes with its absorption

so that considering the frequency with which these children suffer from infection and diarrhoea some degree of deficiency would not be surprising. The possibility that Vitamin C deficiency could be related to infant mortality was publicised by Kalokerinos (1969) in Walgett and Collarenebri N.S.W., although he was unable to support his claims other than by clinical observation.

It was felt that general nutritional status, as evidenced by Vitamin C lack, and the specific deficiency warranted some investigation in Central Australia. The results of this study were reported previously in a letter published in the Med. J. Aust., Jan. 24th. 1970.

The facilities in the biochemistry laboratory at Alice Springs and the instability of Ascorbic Acid render serum assays impossible so simple saturation tests on urine were utilised. It is appreciated that this method leaves much to be desired, however the results obtained are reproduceable within limits, and apparently significant. No other available method seemed applicable to the situation.

In the latter part of 1969, ascorbic acid saturation tests were performed on 40 patients soon after their admission to the Alice Springs Hospital and prior to them receiving supplementary Vitamin C. The saturation test

using 2:6 dichlor-phenol-indophenol as the indicator (Varley, 1963), was made on freshly passed urine between four and six hours after a loading dose of Ascorbic Acid. The loading dose was calculated as 11mgm. per kg. body weight and given orally. All patients were allowed normal fluid intake throughout the test, which may have meant that the urinary ascorbic acid was less concentrated than it otherwise might have been.

The fresh urine was titrated against 5mls. of 0.2% solution of dichloro-phenol-indophenol, made up immediately prior to testing. A few drops of glacial acetic acid were added to the indicator solution, turning it from deep blue to pink. The urine was run in slowly from a burette until the indicator colour just disappeared. The Vitamin C content of the urine could then be calculated i.e.

$$\text{mg. Vitamin C per 100ml. Urine} = \frac{10}{\text{Vol. of urine required}}$$

The patients tested were mostly full blood Aboriginals under 5 years of age. In addition, a 13 year old antenatal Aboriginal girl, a 25 year old postnatal Aboriginal woman, 4 part-coloured and 3 European children were studied.

Only three patients, all Aboriginal boys in their third year, failed to excrete detectable amounts of Vitamin C following a loading dose. After fourteen days of

oral Ascorbic Acid supplements all three boys excreted at least 5mg. per 100mls. of urine, although no dramatic clinical improvement was apparent. The three boys were all grossly malnourished by anthropometric standards, but none had clinical or definite radiological evidence of Scurvy.

Thirteen of the Aboriginal children examined were malnourished andnine well nourished judging by weight and height percentiles. All of them excreted some Vitamin C but several had urinary levels suggesting a degree of desaturation. There was a marked difference between mean rates of excretion in two groups. The undersized group excreted an average of 2.6gm. of Ascorbic Acid per 100mls. of urine, whereas the better nourished children excreted 5.0gm. per 100ml.

There is some correlation between weight and height percentiles and urinary excretion of ascorbic acid following a loading dose. The results of the above study are set out in Figure III.16 as a rank order comparison, and it can be seen that a definite correlation exists, although the series is too small to make the above generalisation valid on these grounds.

On the basis of this study it is felt that various degrees of desaturation, and therefore presumably

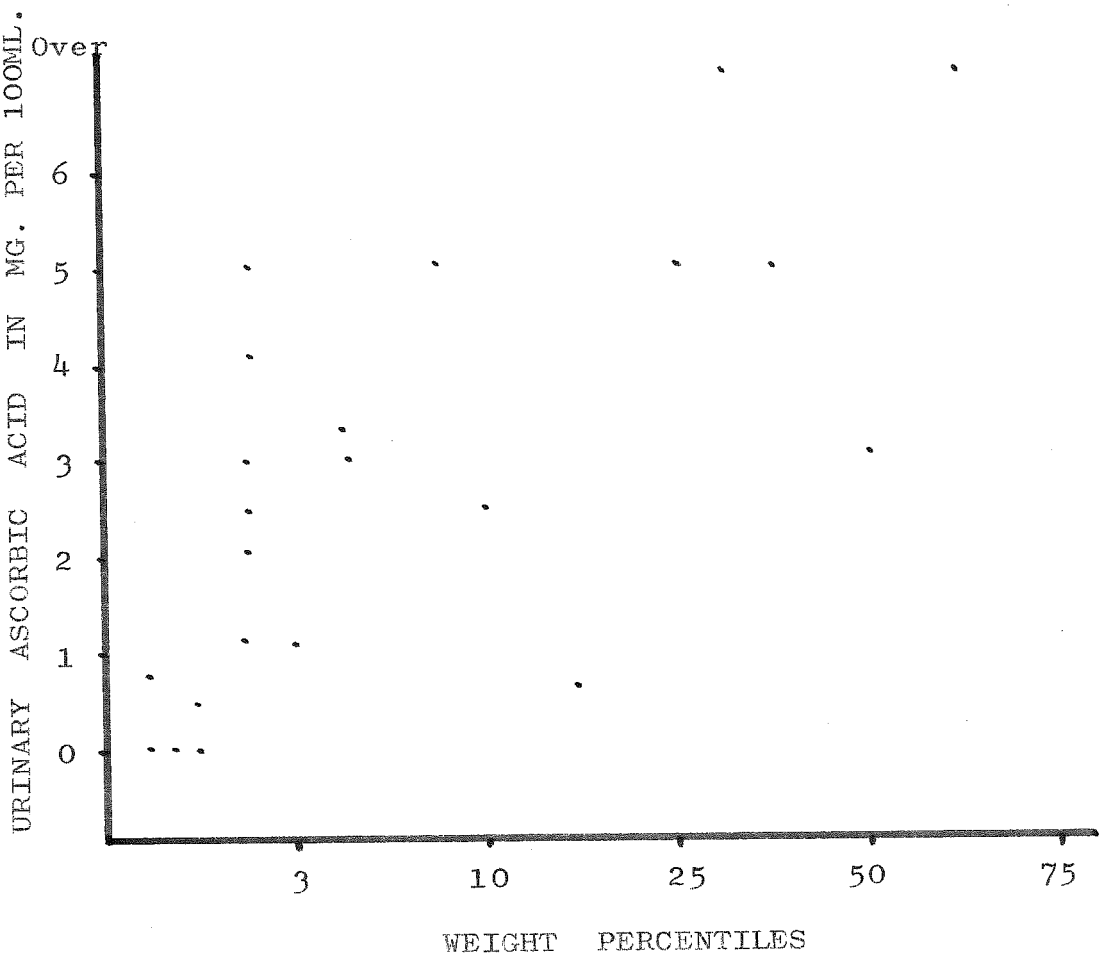

FIGURE III:16 Urinary Ascorbic Acid related to weight Percentiles.

deficiency, of Vitamin C occur in this community. Twenty-four of the forty patients tested produced less than 5mgm. of Ascorbic Acid per 100mls. of urine. This figure is given by the manufacturer of the indicator (B.D.H. Ltd.) as being that above which hypovitaminosis does not occur. The subjects in this case were all hospital inpatients and as a result tended to pass large amounts of relatively dilute urine. It is customary for them to be offered, and in many cases forced to drink, huge quantities of fluid. In some situations patients are even woken every four hours during the night in order that they might drink more. Thus it is felt that a level of, say, 2.5mg. of Vitamin C in the urine probably excludes Scurvy or significant Vitamin C deficiency at least. Taking this arbitary figure as the borderline the incidence of hypovitaminosis could be roughly 30% amongst the Aboriginal children. However one of the three Europeans would also fall into this category as would one part-coloured child and one adult.

The desaturation which occurs is part of the more general under nutrition rather than a significant single deficiency. During treatment for malnutrition, unless supplementary Vitamin C is administered, the development of Scorbutic Symptoms could be expected, but has only rarely been seen.

For the reasons rather similar to those mentioned in the section on Anaemia, Vitamin C estimation has limitations as a nutritional screening mechanism. Now that the need for extra dietary ascorbic acid is becoming more widely appreciated, many settlements and Missions make a point of providing fresh oranges or orange juice for pre-school children, and a multivatamin preparation for the infants and secotrants. The various techniques for estimating Vitamin C nutrition are not suitable for use in remote areas, so in general they must be reserved for patients already selected by another method, e.g. nutritional anthropometry.

Serum Protein.

The level of serum protein, especially Albumen, is of vital importance in areas where Kwashiorkor is prevalent, since this form of Malnutrition is primarily one of protein deficiency. Florid Kwashiorkor has not been seen in the remote areas of Central Australia, although hypoproteinaemia is quite frequently a problem during inpatient treatment of marasmic children.

In the past diets offered to small children probably contained a fair proportion of protein. Now, however, with the enforced swing towards the carbohydrate type of food provided in canteens and stores, the emerg-

ence of protein deficiency as an entity would not be surprising.

Previous studies have shown that the total serum protein levels in children at Papunya are within the normal range (Maxwell, 1964). In 1966, in the Alice Springs Hospital, abnormal serum protein levels were very uncommon, and the Albumen was almost always within the normal range. The present study confirms this, but indicates that the Albumen/Globulin ratio is often abnormal.

During 1969 blood samples were collected from 60 full-blood Aboriginal children, whose medical history was known. Their growth parameters were measured at the same time. Aliquots of blood were sent to the Australian National University in Canberra, where Mr. N. M. Blake of the Department of Human Biology carried out immunoglobulin assays. The remainder of the blood was used for estimation of total protein and serum albumen, except for about 20 from which a further aliquot was sent to the Department of Child Health in Adelaide for Transferrin assay.

The immunoglobulin investigation was undertaken initially as part of the International Biological Program, Project H.A.I.

The range of values for total protein coincided

exactly with that found by Maxwell. The mean value was 7.75g. per 100mls. of serum and the range 6.9 to 8.6. There was no correlation in this group between growth retardation, as measured by weight for age and total serum protein. The results of the basic protein estimations are shown in Table III.33.

TABLE III.33 Serum Protein Levels.

	GM. PER 100MLS.		
	MEAN	RANGE	LABORATORY NORMAL
Total Protein	7.8	6.9 - 8.6	6.3 - 7.9
Albumen	3.8	3.1 - 4.4	3.5 - 5.3
Globulin	4.0	2.8 - 4.7	1.8 - 3.6
Immuno Globulin	1.7	0.76- 3.5	0.7 - 1.2

Sixteen children (27%) had total proteins higher than the upper limit of the laboratory range. On the other hand 22 (37%) had Albumen levels below the minimum range, although the degrees of hypoalbuminaemia were relatively mild. The most remarkable variation from accepted levels appears in the Globulins. No less than 43 (72%) had some degree of hyperglobulinaemia and it appears that most of the increase is in the gamma-globulin fraction. Despite the consideration that many of the patients were young and theoretically should have had low immunoglobulin levels, 48% in fact had excessively

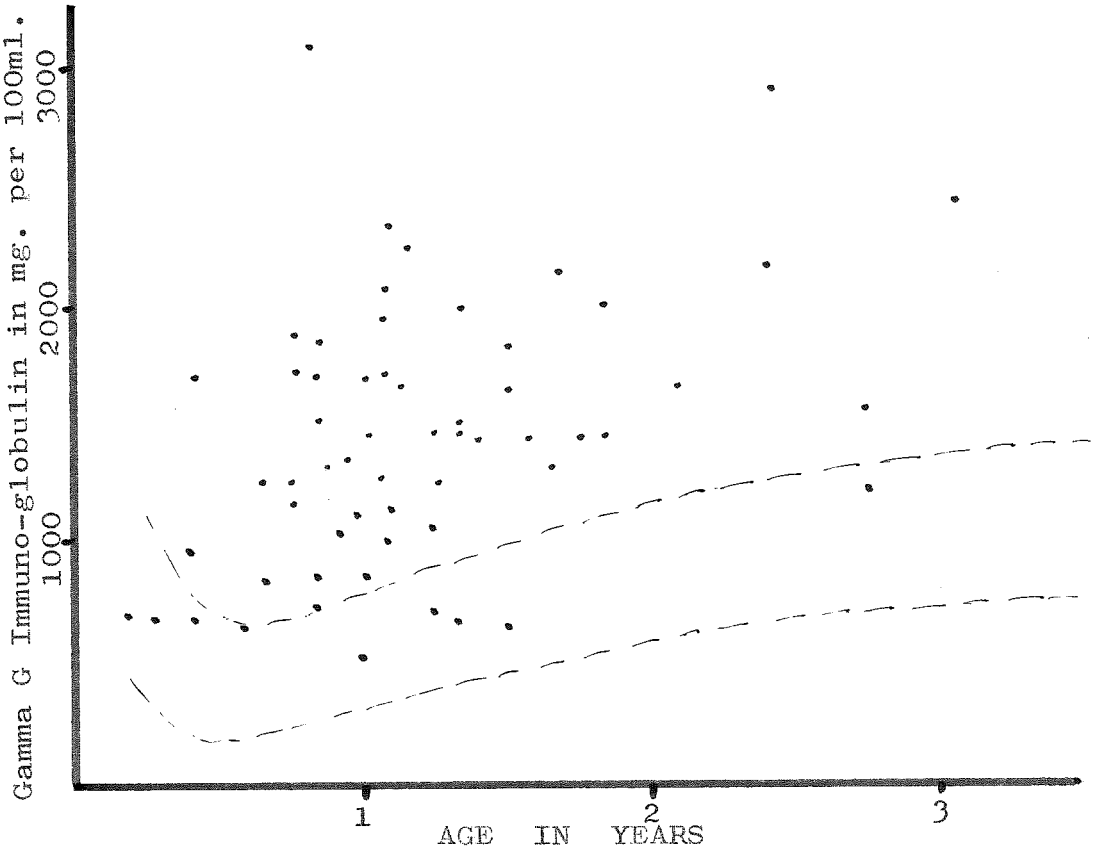

FIGURE III:17 Gamma G Immuno-globulin levels related to an approximate normal range.

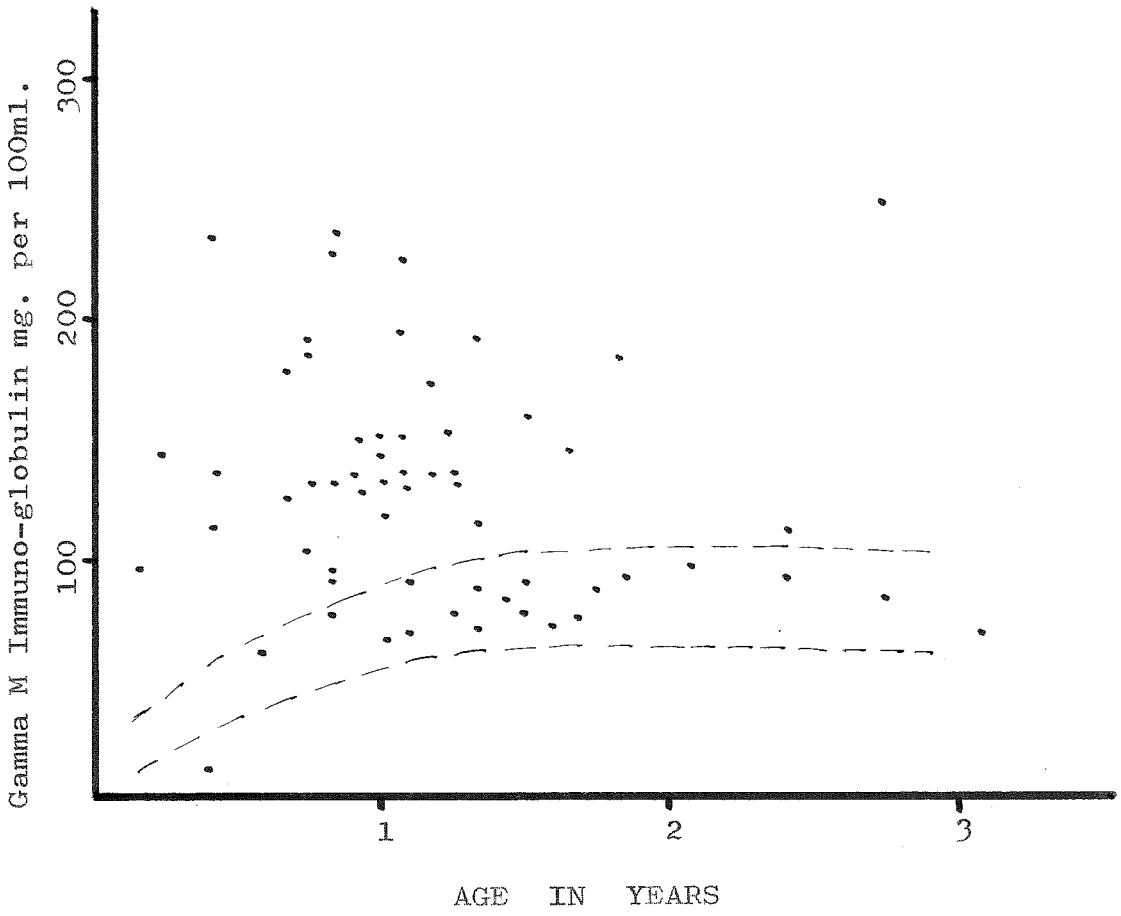

FIGURE III:18 Gamma M Immuno-globulin levels.

high values.

In order to establish the significance of age effects on the levels of gamma G and gamma M immunoglobulins, rank order comparisons were made and the results are reported in Figures III.17 and III.18. There is a relationship between age and the gammaG levels in this series, which, to some extent, nullifies the importance of other findings related to specific diseases reported later in Table III.34. However there is no such age dependence of gamma M immunoglobulin apparent.

Both gamma G and gamma M protein levels are generally very high, however, the gamma G fractions in the very young infants, especially those with no history of infection or growth retardation, tend to be much nearer to, or actually within, the normal range, expressed by the dotted lines on the figures. This is not so of gamma M.

The 60 children from whom the sera were drawn, were divided on the basic medical history, physical examination and anthropometry into four groups. These groups were chronic or recurrent respiratory infection, recurrent diarroeal disease, retarded growth or malnutrition and "normal". The mean values for the gamma G and gamma M protein for these four groups are shown in Table III.34.

TABLE III.34 Mean Immunoglobulin Levels in Major Disease Groups.

MEDICAL HISTORY	NO.	AV. AGE (MOS.)	GAMMA G MGM/100ML.	GAMMA M MGM/100ML.
Recurrent R.T.I.	25	22.0	1761	122
Diarrhoeal Dis.	13	14.7	1375	102
Malnutrition	16	14.1	1295	146
"Normal"	6	10.0	1025	134
TOTAL	60	16.6	1465	127

The great variation in gamma G levels between the various morbidity groups, as previously suggested, is partly age specific. In order to assess the significance of these variations, the mean gamma G values were plotted on a chart similar to Figure III.17 and the distance from the normal mean measured. This chart is reproduced in Figure III.19 and it suggests that the gamma G globulin is raised relatively further in children with respiratory disease than in the other groups. The "normal" children had levels nearest to the mean. It is reasonable to deduce from Figure III.19 that the high gamma G level is an acquired and progressive lesion precipitated by one of many influences, since the four points fall on a straight line. In addition, in Figure III.17, the four children under 8 months with gamma G levels within the normal range, all had "normal" medical backgrounds, had

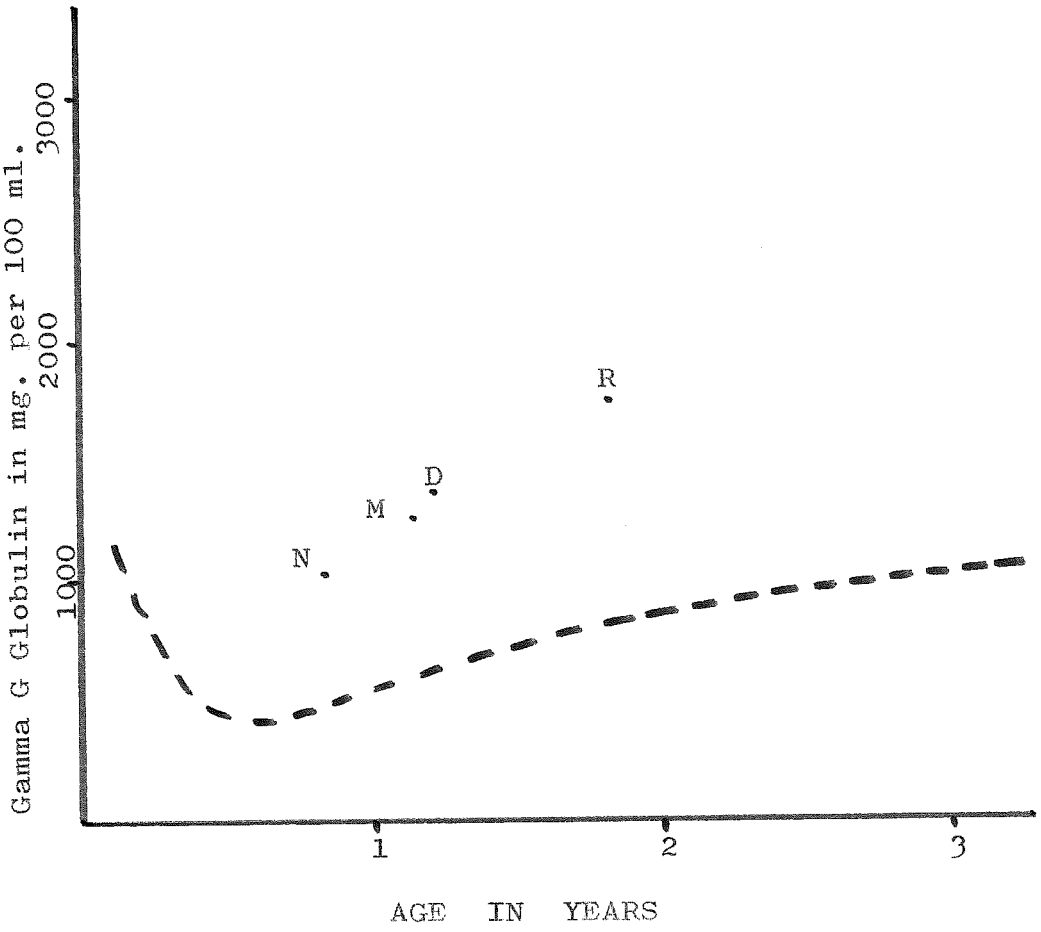

FIGURE III:19 Gamma G Immuno-globulin levels in "normal" children (N) and those with malnutrition (M), diarrhoeal disease (D) and respiratory infection (R).

been exposed to a variety of infections and for some reason, possibly better nutrition, had not come to the notice of medical record keepers.

The situation regarding gamma M immunoglobulin is less clear. Certainly the mean value for the growth retarded children was further from the laboratory mean than any other group, although the "Normal" children also deviated considerably from this mean. Figure III.18 suggests a massive rise in gamma M levels early in life which is not sustained beyond about two or two and a half years.

Probably the abnormally high levels of both of these immunoglobulin fractions are due to frequent exposure to both bacterial and viral, and in many cases parasitic, infection. Malnutrition may play a part but this is difficult to define clearly.

From the point of view of resistance to the common infections of childhood, there seems little doubt that Aboriginal children possess adequate amounts of gamma-globulin.

Gamma A immunoglobulin was estimated at the same time as the other two fractions but the marked age dependence in childhood makes the results difficult to

interpret. Presumably, since this particular globulin does not pass the placental barrier, the neonate begins manufacturing it shortly after birth, in much the same way as gamma M, and the level gradually builds up to the adult figure of around 300mg%. If this is so, the results of the assays in the 60 children in question probably represent no gross abnormality. The absolute levels for these children are shown in Figure III.20.

Magnesium Deficiency.

Occasionally during recovery from marasmus, particularly when associated with diarrhoea, these Aboriginal children exhibit for varying periods an odd syndrome of muscular tremor and hyper-reflexia amounting almost to tetany. This may last only a few hours or may be quite prolonged and continue for three or four days. It was felt at first early in 1966 that this situation represented Calcium deficiency, but serum Calcium estimations gave normal results and intravenous Calcium Gluconate failed to correct the lesion except transiently in some cases and then, presumably, in some non-specific way.

The clinical picture closely resembles the "encephalitis like syndrome" described by Kahn and Falcke (1955) and, after a time, Magnesium deficiency was inculpated (Back, Montgomery and Ward, 1962). Sera from three such

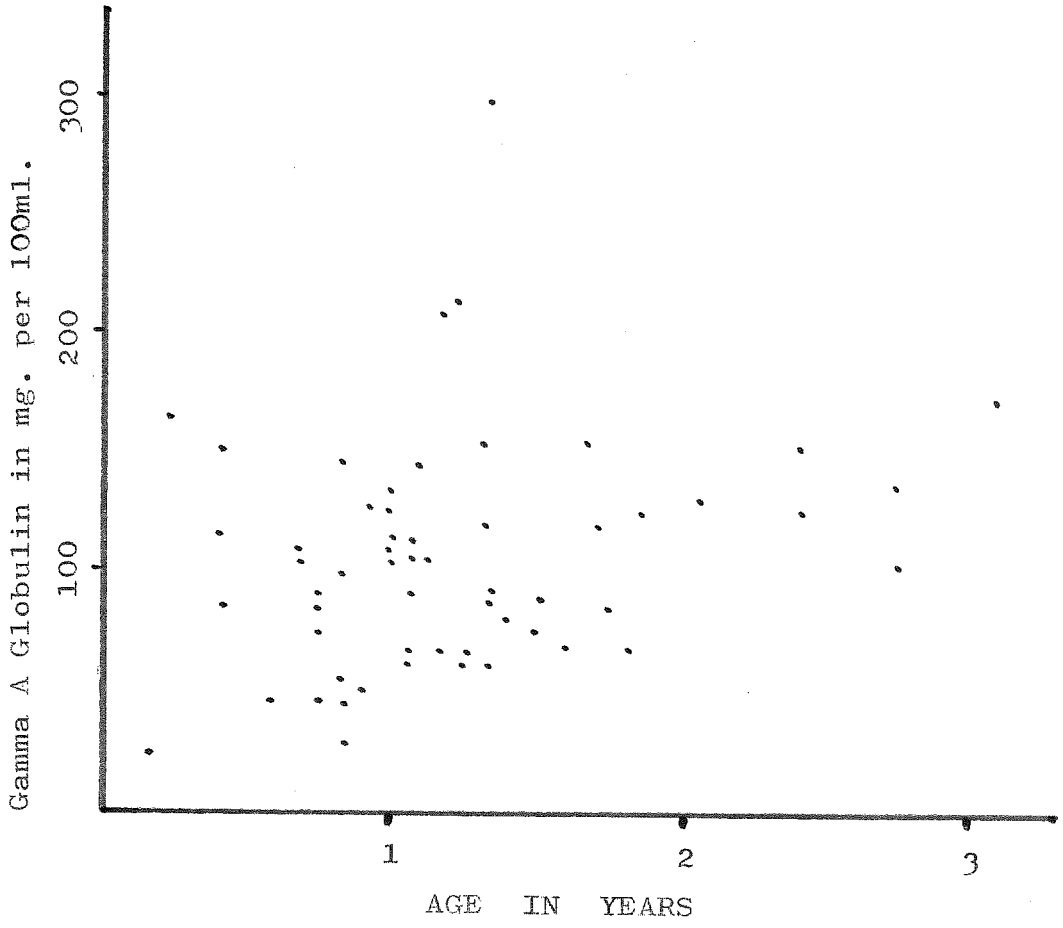

FIGURE III:20 Gamma A Immuno-globulin levels.

cases were sent to Adelaide for Magnesium estimation. All three children were between nine and eighteen months old and each of their serum Magnesium concentrations was less than 1.0meq. per litre.

A small series of ten sera were assayed from children in the same age group admitted to hospital during 1966 for reasons other than malnutrition and/or diarrhoeal disease. They were generally surgical cases or suffering from some minor respiratory problem. The mean serum Magnesium level was 1.75, which was within the normal range quoted by the laboratory in Adelaide, although rather nearer the low end than the high.

Thus it seems likely that the hypomagnesaemia which occasionally used to be seen in children recovering from malnutrition was the result of deficiency in their treatment rather than a reflection of a significant dietary lack in the rural population. Certainly since magnesium has been administered to these children routinely the problem of tetany has not occurred.

AETIOLOGY

Many of the reasons for the existance of malnutrition in the Aboriginal community have already been discussed in other parts of this thesis. It has been said that under-nutrition is inseparable from unhygienic environment

in poor communities (Gopolan, 1967). Unfavourable socio-economic considerations, language barriers and little understood cultural differences make an Aboriginal mother likely to be less skilled at infant feeding than even the worst Caucasian mother. This is especially so when it is considered that the Aboriginal has no knowledge of the sorts of food Europeans consider suitable for small children.

Breast feeding is universally practised amongst Aboriginals as in other primitive groups. Although the nutrient content of breast milk varies widely in these people, it seems adequate for normal growth during the first five or six months of life (Elliott, Maxwell, Kneebone and Kirke, 1969).

During discussion on growth rates it was pointed out that the large majority of children, who are practically all fully breast fed, develop according to accepted normal patterns for around six months. After this age, growth retardation is common since breast milk alone is no longer sufficient to maintain growth. Supplementary diet must be given after six months, and possibly even earlier, in order to avoid undernutrition.

Here then is the basic cause of malnutrition in Central Australia; lack of supplementary infant feeding,

in the second six months of life. The mothers are frequently and quite unfairly, in most cases, accused of being lazy no-hopers. Obviously in any group there will be a proportion of less able people, and this must be so of Aboriginal mothers. The women, living near towns, who drink enough alcohol to interfere with their family life must be considered poor mothers. These, fortunately form a small minority at present. The real problem is in remote areas where, although in some cases baby foods are available, the mothers still adhere to old customs and the bread winner's income is insufficient to provide for feeding and clothing the family appropriately.

The need in this situation is clearly for an extensive education programme incorporating very basic material on community life, concepts of health and infant feeding.

Other factors involved in the production, or at least aggravation, of undernutrition are primarily infective ones. Recurrent or chronic diarrhoeal disease intereferes with absorption even if theoretically enough nutrient is ingested. Chronic lung disease may increase the demand for nourishment to an extent where it may be responsible for inanition. When these two systems are diseased in the same patient the situation is practically incompatible with good nutrition.

Except in a small minority group the primary cause of malnutrition in Central Australia is simply not enough food. There are minor differences in the details of the problem in various places. In one place, for example, naturally occurring foods may be readily available and widely used so protein intake is fairly high, whereas in another situation the entire diet may consist of cheap, filling carbohydrate from a station store. Some people, responsible to and subsidised by the Government for the welfare of Aboriginals living on their properties, are conscientious. They provide and encourage the use of appropriate infant foods and vitamin supplements, and in many cases maintain their own longitudinal weight records and actively promote the health of the community. Unfortunately there are others who regard the government subsidy as personal income and spend enough on behalf of the Aboriginals only to maintain appearances. In this latter case, which is by no means uncommon, the station store is usually very poorly stocked, there is no encouragement for the people to purchase the more nutritious food types and very often the prices charged are prohibitive. One example of this is the pastoralist who charges a dollar per article, such as a loaf of bread, a tin of beef or a small tin of powdered milk, on the grounds that Aborigines do not understand money values and do not expect any change.

This type of exploitation fosters poor rapport, difficulties in education and very low socio-economic standards which reflect again in infantile malnutrition.

The problems of Vitamin C deficiency and Anaemia have been discussed. One significant aetiological factor in these cases is the widespread use of unfortified powdered milk, on account of its ease of transport and cheapness compared with more sophisticated types.

On several occasions various people have appreciated this sort of problem and have tried to introduce such things as iron-fortified flour and higher quality milk, but without a great deal of success. This lack of benefit merely underlines that it is the six to twelve months old infants who need the special attention, and this has to come from their own mothers in order to be effective. Fortified flour is not of great value to infants, and something other than milk, despite its being the best available, is necessary at this age.

There is need for a great deal of research into feeding habits, available foods and education of mothers, if this problem of malnutrition, and incidentally morbidity and mortality, is to be solved in the forseeable future.

IMPLICATIONS

A good deal has been said about the relationship of malnutrition to such things as duration of hospital stay and severity of diarrhoeal disease and chronic lung infection. These are relatively short term problems, and are closely allied to infant and toddler morbidity and mortality.

Table III.35 summarises the relationship between undernutrition as diagnosed by growth retardation and early childhood morbidity and mortality. The "at risk" population under five years was studied in 1968. The hospital inpatients were studied in 1966 and 1968. The figures for the deceased group were collected over the entire study period up to 1968, use being made of Settlement and Mission records as well as those of the N.T.M.S.

TABLE III.35 Malnutrition, Morbidity and Mortality

	GROWTH RETARDATION
At risk population (1968)	16.5%
Inpatients (1966 and 1968)	53.4%
Deceased children (1965 - 1968)	68.8%

Growth retardation was accepted as being present if the individuals weight was consistently below the 3rd. percentile, as expressed by the Caucasian Standards

(Tanner, 1958). The weights taken into account for the deceased group were those recorded prior to the terminal illness.

Undernutrition was three times more common amongst the hospital population than in the group at risk, and four times as common in the children who died. It is clear, therefore, that malnutrition is at least partly responsible for high morbidity and mortality rates.

The long term implications of growth retardation in infancy and early childhood are rather more difficult to assess. It is reasonable to assume that if physical growth, once having been retarded, never quite achieves normality unless there is some considerable delay in bony fusion and so on, that mental development may similarly be permanently affected.

Although there is some degree of accelerated growth after the age of around three years, a high proportion of children, stunted in infancy, remain significantly small for their age throughout their school going career. The children studied in 1966 were followed up in 1969. Of the 61 initially found to have retarded physical growth 43 were seen again, and all but 9, i.e. roughly 75% were still significantly undersized. Thus it was felt that most children of school going age, whose growth parameters

indicate physical retardation, were probably malnourished earlier in life.

MALNUTRITION AND EDUCABILITY

Malnutrition in infancy may retard physical growth permanently. The head circumference is affected in much the same way as height, but during recovery from infantile and toddler malnutrition, head size is much slower to show improvement. Thus the histograms in Figure III.8 for head size are quite similar in each age period from 12 months to 4 years, whereas the significant increase in heights (and weights) relative to European Standards over the same period is evident in Figures III.6 and III.7.

Head size and brain weight must be related, and it has been suggested that intellectual ability may, to some extent, be determined by the size of brain. It appears then that malnutrition in early childhood may cause retarded brain growth in some individuals. If reduced educability follows in the wake of malnutrition then a very compelling reason exists why the nutritional status of Aboriginal infants should be energetically controlled. After all most of the socio-economic and medical problems will depend upon education for their solution.

The main cause of delayed physical development in

the group of children studied was Marasmus. A school going child with physical stunting therefore had quite probably suffered from malnutrition in infancy. If the trends seen in the recovery phase up to four years of age continue thereafter, the height and weight of the child may be nearer "normal" than his head-size.

In order to test the hypothesis that malnutrition-induced stunting is in some way related to reduced educability, 209 children, 5 - 12 years old, attending schools at Hermannsburg, Amata or Yuendumu were studied near the end of the final school term in 1969. The children were selected in that only those whose ages were known and who had attended one class, with the same teacher, for the entire school year were included, and any with significant hearing loss, visual defects or a recorded history of severe illness were excluded. The final group were weighed and measured and their height, weight and head-ages calculated. These terms designate the ages at which the parameters coincide with the European medians. Thus the weight-age of 10 kg. girl would be 14 months, since it is at this age that 10 kg. falls on the 50th. percentile line.

Nutritional indices were produced by expressing the weight-age, height-age and head-age as percentages of chronological age. Hence the overall index of growth.

(N.I.) is calculated as follows :-

$$N.I. = \frac{(\text{Weight-age} + \text{Height-age} + \text{Head-age})}{\text{Chronological Age} \times 3} \times 100$$

No suitable method of testing Aboriginal childrens' intellectual ability has been devised for use in Central Australia, so the school teachers, having observed the children in this study, in relation to their peers, for three school terms were asked to make an assessment of each individual. The request stated that each child should be scored on his ability to accept new concepts rather than on some arbitrary level of attainment. The scoring system was kept rather too simple unfortunately, as the enthusiastic co-operation shown by the teachers concerned was not wholly expected. Each class acted as its own standard, since generally there is no streaming until the Senior grades are reached at the age of 14 or 15 years. Children assessed as above the class average were given "A", the biggest group of around average ability, "B", and those distinctly below par, and plainly less educable than the others, received "C". It is appreciated that a 10 point scoring system might have made the study rather less general, but possibly more teacher variation would have been introduced.

Two rank order comparisons were made, the first

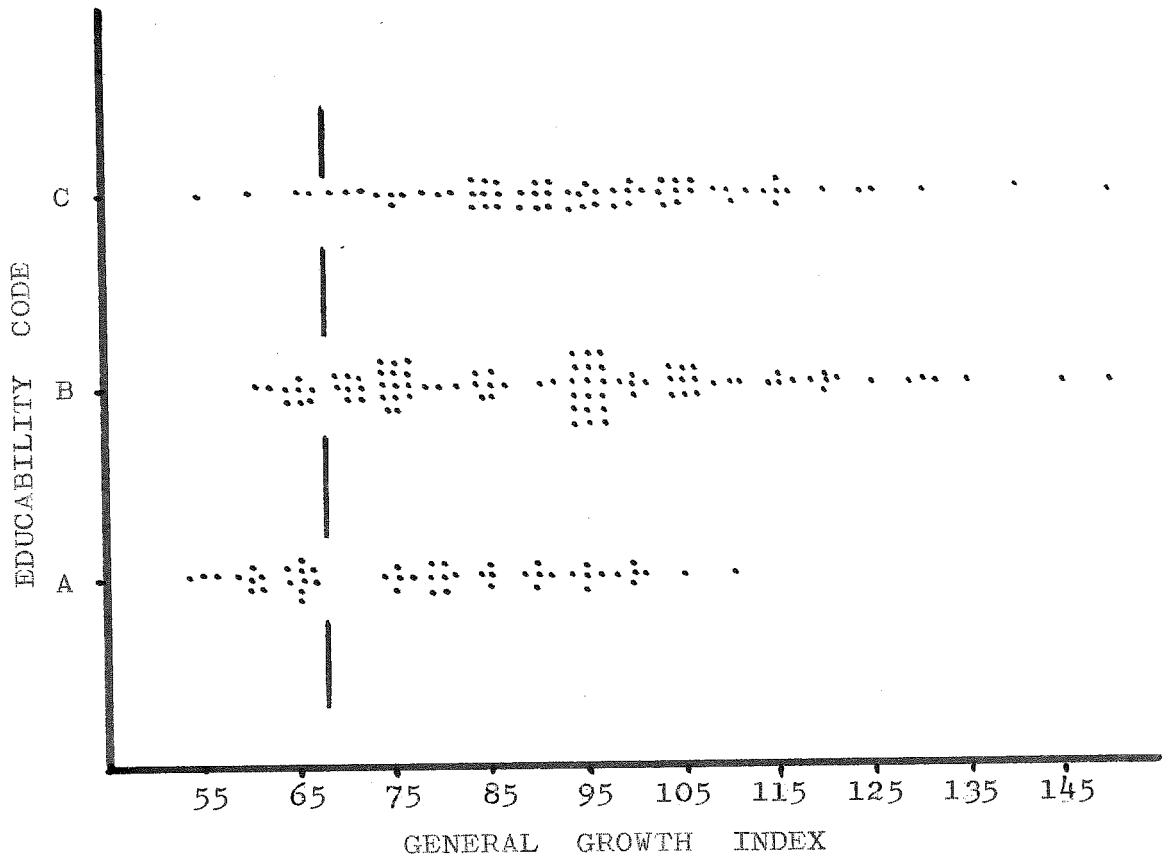

FIGURE III:21 Relationship between a Nutritional Index and school teachers' assessment of Educability.

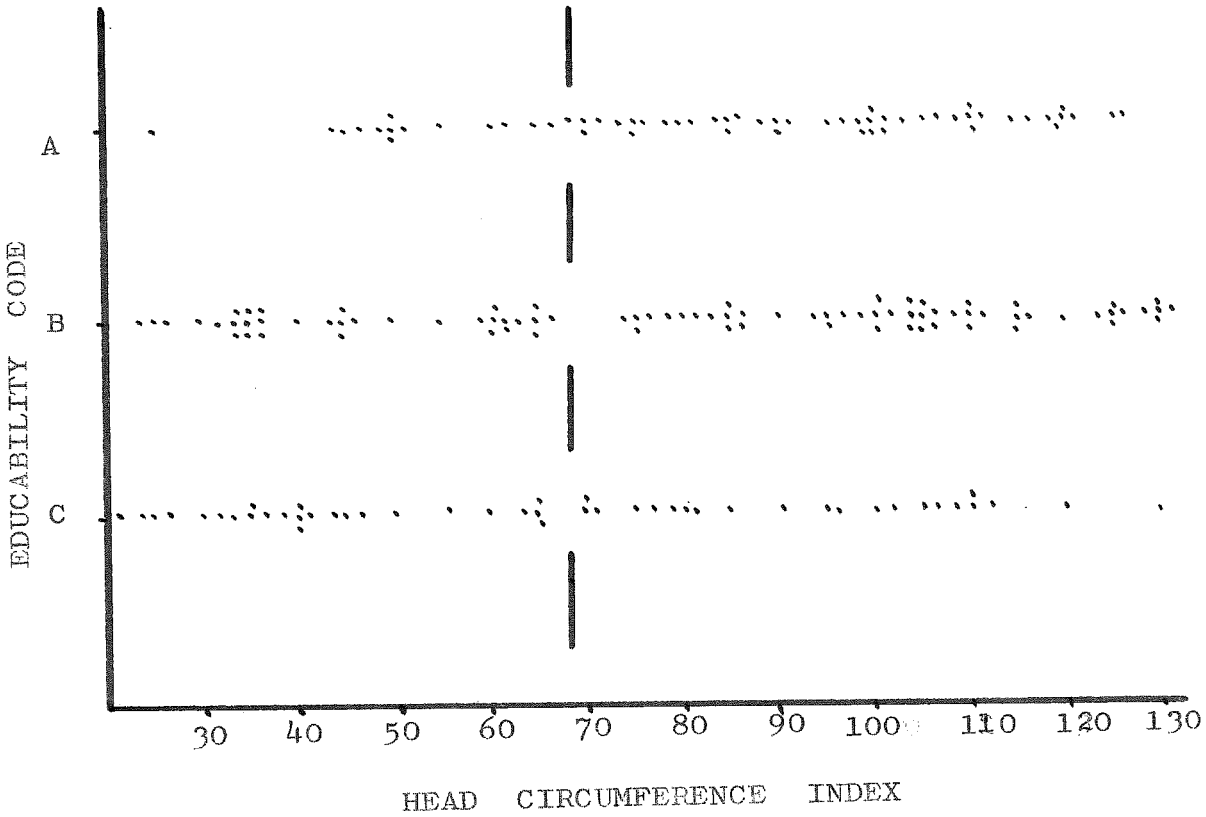

FIGURE III:22 Relationship between Head-size Index and Educability.

using the teachers' "Educability Assessment" and the general Nutritional Index, and the second comparing the same assessment and the Head Index. An arbitrary division at the Index level of 70% was made, below which was considered to be outside the "normal" growth range. Figures III.21 and III.22 show these comparisons, and there is clearly greater representation below the division at 70% in the "C" group in both charts. Table III.36 summarises the results of this study.

TABLE III.36 Malnutrition and Educability (1969)

EDUCABILITY	NO.	NUTRITIONAL INDEX BELOW 70%	HEAD-SIZE INDEX BELOW 70%
Above Average	67	4 (6%)	14 (21%)
Average	92	9 (10%)	33 (36%)
Below Average	50	17 (34%)	25 (50%)
TOTAL	209	30 (14%)	72 (34%)

The number of low general growth indices is obviously contributed to very greatly by the much higher proportion of low head-size indices. Although more of the less educable children tend to have smaller head sizes, there is a relatively large proportion of above average pupils with small heads too. This indicates that,

(i) factors other than malnutrition, such as Encephal-

itis and intra-uterine influences, which are not recorded, cause retarded head growth without necessarily interfering with educability and/or

(ii) malnutrition may produce small heads but not always retarded intellect, and/or

(iii) Aboriginals tend to have small heads anyway, and/or,

(iv) which is most likely, only malnutrition severe enough to retard permanently other parameters of physical growth, besides head circumference, is likely to interfere with educability.

The last point is supported by the relationship of the general Nutritional Index to the educability score. There is a much better correlation here than with head size alone.

It is reasonable to say that severe early childhood malnutrition causes delayed growth, and sometimes permanently retarded physical development. In cases where the growth index of school children is significantly lower than expected, there is very likely to be reduced educability. Obviously in many cases of inferior intelligence, factors other than malnutrition and/or depressed growth are responsible, and conversely very small people are not necessarily unintelligent.

The extent to which prenatal influences, such as

maternal malnutrition and infections, have a bearing on headsize and intellectual potential is difficult to assess without a closely controlled, longitudinal investigation. Considering that head-size and brain-weight are relatively much nearer adult proportions at birth than almost any other physical parameter, these early factors may be of great importance. The important point however is that widespread infant and toddler malnutrition may lead to relatively less educable second-class citizens, and should consequently be prevented at all costs.

OTHER MORBID CONDITIONS

The general situation regarding the incidence of various diseases was presented in Chapter II. It is apparent from Table III.3, that roughly 80% of the hospitalised medical problems in under five year olds stem from diarrhoeal disease, respiratory infection and/or malnutrition. The remaining 20% is made up of a heterogeneous group of conditions which will be considered briefly in this Chapter.

The vexed question of Prematurity is discussed in the chapter on Mortality. Few premature babies born in remote areas are evacuated to hospital since it has been found that they do far better if left alone and not subjected to the rigours of a long journey by road or light aircraft. The small number of low birth weight children

brought into hospital are included, in Table III.3 under the miscellaneous heading.

Infections.

It is not surprising that infections other than those of the bowel and the respiratory tract should be prominent in this discussion of morbidity. The Aboriginals' unhygienic mode of life coupled with an already immense pool of various infective agents in the community make conditions like Impetigo, Conjunctivitis and abscess-forming Adenitis extremely common. In some areas Impetigo is almost universal amongst pre-school children. The three ailments mentioned are generally treated in the rural setting and only the most severe cases are sent to hospital. It was pointed out in Chapter II that the morbidity as seen in hospital is quite different to the situation in remote areas.

The commoner infections are taken individually and their significance discussed in the following paragraphs.

Meningitis.

The purulent meningitides occur frequently and, because of delay in presentation and diagnosis, they often cause death or at least permanent brain damage. Fortunately Tuberculosis is now quite rare and consequently tuberculous meningitis is not a problem. Influenzal, meningococcal and pneumococcal infections all

occur and tend to conform to the expected age pattern.

Slightly less than 2% of Aboriginal children admitted to hospital have purulent meningitis. This figure is only a little higher than that for the part-coloured and white community, and the high incidence is clearly related to the enormous respiratory infection rate which affects all racial groups in the Alice Springs area.

It is worth noting again that several cases of meningitis occur in remote areas each year and the patient dies before reaching hospital. Sometimes the diagnosis may be made, rather tenuously, from a radio message that there was fever, vomiting and terminal convulsions etc. or it may be made at autopsy. Possibly other cases occur and are treated with antibiotics by a rural health worker who believes that she is treating a febrile respiratory illness. These meningeal infections may be aborted and never come to the notice of record keepers, despite what is probably quite inadequate treatment. This may explain, to some extent, the relatively high incidence of small head sized children of school going age who have no known history of meningitis or infantile malnutrition.

The frequency with which late presentation complicates the treatment of meningitis and adversely affects the prognosis is the most important aspect of this

disease and the one which suitable education programmes could conceivably improve.

Skin Sepsis.

School sores of both Staphylococcal and Streptococcal origin are exceedingly common in pre-school and school age children. Many of the lesions are very extensive and may cover up to 10% of the body surface. They are particularly difficult to manage when they occur in the natal cleft and on the scalp. On occasions whole communities have had to be treated simultaneously with an appropriate antibiotic in order to curtail re-infection.

In general, small localised areas of skin infection, which can be occluded fairly simply, do well with local treatment only, but the more widespread lesions, especially if there is a tendency to abscess formation, usually require both local treatment and parenteral chemotherapy. Vast quantities of antibiotics are used in this way and now that penicillin resistant Staphylococci are so common, choice of antibiotics is becoming difficult. Erythromycin has not been very successful although Lincomycin is quite useful.

Abscesses.

The commonest cause of abscess formation is Impetigo but Adenitis and wound infection are frequently problems.

These latter two are also affected by late presentation in that, in many cases, had the lesion been seen earlier, abscess formation may have been prevented.

The abscesses sometimes reach such proportions that they need formal operative drainage. Around 4% of hospital admissions are for this purpose, but many more are coped with by bush nurses and medical officers on routine visits.

Osteomyelitis.

As with most other gross bacterial infections, Osteomyelitis is very common in Aboriginal children. Over the past five years an average of three new cases have been hospitalised each year. This high incidence apparently represents a marked change from the situation in the early 1930's when no evidence of bacterial bone infection was seen (C.J. Hackett, personal communication). The infecting organism is usually Staphylococcus in the first instance but, very often, especially after surgical intervention and prolonged periods of "moist occlusion" of the wound, Pseudomonas Pyocyanaea becomes a problem.

Tetanus.

Although tetanus is uncommon because of reasonably thorough immunisation programmes, it is worth mentioning if only to say that roughly one case per year is seen in

hospital. How many neonatal deaths in rural areas are due to Tetanus Neonatorum is impossible to assess, but it seems likely that there are a few. In some places the Aboriginal midwives divide the umbilical cord by burning, rather than cutting with what could probably be a dirty knife or piece of glass. This custom almost certainly prevents a lot of infective problems.

Currently the Rural Health Staff of N.T.M.S. are administering Tetanus Toxoid to all pregnant Aboriginal women. This technique has been shown, in the Territory of Papua and New Guinea, to decrease the incidence of both neonatal and maternal tetanus quite markedly. (J.Biddulph, personal communication)

Trachoma.

A good deal has been written about the incidence of Trachoma in Central Australia. Some ophthalmologists claim it is almost 100% (G. Morlet, personal communication) and certainly a high proportion of old people have corneal lesions which could well be the Pannus of Trachoma. Without specialised knowledge and a slit-lamp the problem of Trachoma in small children nowadays is impossible to assess, but it seems relatively slight. It may be because so much Tetracycline eye ointment is used for treating simple purulent Conjunctivitis that Trachoma does not become established.

Streptococcal Infection.

In the infant age group conditions such as Nephritis, Scarlet Fever and Rheumatic Fever are not problems. During the period studied one case of Acute Nephritis was seen in a fourteen month old child from Hermannsburg and two cases in four year olds. No cases of Scarlatina or Rheumatic Fver have been diagnosed in under five year old full-blood Aboriginals, although Rheumatic Fever and Nephritis certainly are not uncommonly seen in the school going children.

Others.

Several other types of infection are seen periodically. They are in no ways different from similar infections in the European community and so warrant only a brief mention. Umbilical infection in neonates often goes unnoticed for several days since the redness is not seen against the dark skin and very often newborn babies are not produced for inspection for anything up to a week or ten days post partum. Considering the unsterile methods of severing the cord practiced by some of these people it is surprising that more serious cord infections are not seen.

Ulcerative-gingivo-stomatitis is seen not infrequently, and then usually in children debilitated by some other

condition. Monilial over-growth often characterises this lesion.

Hookworm infestation is rare in this group except in those from the extreme North end of the Central Australian area. Generally only one or two cases are seen annually in Alice Springs.

The common childhood exanthems from time to time cause a sudden rise in morbidity. Measles (Morbilli), as has already been said, is a severe and potentially lethal disease in malnourished children, and during one of the frequent epidemics is responsible for many hospital admissions. The recent introduction of "Koplivac" vaccine may serve to control measles, and the other infectious diseases are relatively mild in this age group.

Other minor problems are related to Pediculosis Capitis and Scabies. The latter is often responsible for precipitating an impetiginous illness when the scabitic lesions become infected after excoriation by scratching.

Trauma.

Four percent of Aboriginal children admitted to hospital during 1965-1969 were suffering from burns, fractured bones or foreign bodies, usually in their ears or genitalia.

The burns occurred usually at the onset of the cold weather, when sleeping children rolled into the fire. Bony injuries occurred largely in the children over two years old and were the result of accident rather than parental battery.

The foreign bodies consisted of beans and grass seeds in external auditory canals, hair pins, matches and pieces of wire in Vaginas and Urethras, small stones and flowers in nostrils and so on. A 1½" sewing needle within the Pericardium of a two year old boy had presumably gained entry via the right middle lobe bronchus.

Ingestion of Toxins.

Very few Aboriginal children ingest toxic material, because no doubt of its relative unavailability, and the fact that these children are rarely out of their mothers' sight. During 1968 only 5 out of 552 admissions were for ingestion, whereas in the part-coloured and European people 10 out of 218 admissions were for this reason. This means that ingestion was 5.3 times as commonly a reason for admission in non-aboriginals.

Almost all the poisoned Aboriginals were from Cattle Stations and had drunk kerosene or petrol. The other group, however, had swallowed a much wider range of toxins including household detergent, floor polish and white

spirit, several plants mentioned in Chapter II and such medicaments as aspirin and iron.

Congenital Anomalies.

Several of the children included under this heading in Chapter II were re-admissions for definitive surgery to their cleft lip or fitting of new splints for their talipes equino-varus. Two percent of hospital admissions were due to congenital lesions of one sort or another.

There may be some severe congenital heart lesions or multiple defects which cause early neonatal deaths and are never recorded. Thus the figures estimated for annual incidence of some conditions are likely to be inaccurate.

The commonest deformity in the Aboriginals in this area is Talipes equino-varus, and it occurs roughly once in 180 live births. Cleft palate occurs less frequently and appears once in 400 to 500 live births. Some form of heart lesion is not uncommon and such anomalies as septal defects and patent ductus arteriosus are found two or three times annually. The more severe heart lesions, multiple deformities, trisomies and meningo-myeloceles are all seen periodically, but in numbers too small to quote as incidences. There is no reason to believe

that any of these deformities except the Talipes occurs much more or less frequently than in the European community.

An occasional microphthalmos is seen and two cases of oculo-auriculo-vertebral dysplasia in full blood, female siblings have been previously described (Kirke, 1970). This latter condition is better known as Goldenhar's Syndrome.

Social Problems.

Parental neglect or incompetence, sometimes due to alcoholism but more often ignorance, is becoming a more frequent cause of morbidity. Now that infant mortality has fallen, family sizes are correspondingly much bigger and the mothers' responsibilities greater.

Since maintenance of breast feeding is so important to the well being of an infant under six months old, there is a tendency to admit a baby as a boarder if its mother needs hospitalisation. This then exposes the infant to cross infection by resistant hospital organisms.

Only one definite example of the "battered baby syndrome" has been recorded. The thirteen year old full-blood mother, from the Barkly Tableland, said she did not want her new-born baby and gave it to a group of young children to play with. On admission the child was severely

bruised, but had no fractures, and has since done well as another woman's foster child.

A good deal has already been said about the cultural and language differences which contribute to morbidity. The inability of a sick child's mother to communicate with a nurse or doctor, and vice versa, is embarrassing to both parties and detrimental to the child's health. Often the mother will delay seeking help until obvious physical signs appear, although, like most mothers, she has been aware of the illness much earlier. In the interim she may consult with the tribal medicine man who probably does no real harm other than increase the delay in presentation. It is possible that the Aboriginal practitioners have many therapeutic successes of which Europeans are totally unaware, but it is difficult to believe that they can cure severe lung infection, gastroenteritis and malnutrition in infants.

The only possible way to overcome these problems is a programme of cross-cultural education. In other words, the object would be to teach each group about the other. Until such time as free communication is possible between the different cultural groups, even basic concepts such as healthiness, the nutritional value of foods and transference of infection will remain foreign to the Aboriginal. It is difficult to motivate people to carry out

such procedures as postural drainage of chests and washing of hands, if they cannot understand the principles making them necessary.

Iatrogenic Problems.

An increasing number of children are being seen as a result of injudicious treatment. The reason for this situation is that many people, who have had little or no paediatric training or experience, are in a situation which makes them largely responsible for diagnosing and treating sick children. On a few occasions, unfortunately, inappropriate advice has been given over the radio, by inexperienced medical officers.

One of the commonest, and most severe, iatrogenic situations has arisen in malnourished children with diarrhoeal disease. The tendency has been to restrict their diet to a Dextrose/Saline mixture until the bowel becomes inactive. Very often, this approach serves only to aggravate the nutritional problems and precipitate electrolytic catastrophes. It has been found, in this area, that administration of large amounts of oral Normal Saline rapidly produces hypernatraemia, and unless Potassium supplements are given at the onset of treatment, hypokalaemia may also become a danger. Other facets of this dangerous predicament have already been discussed, and are related to hypalbuminaemia, hypomagnesaemia and so

on. Probably the most severe side effect of prolonged dietary restriction is simply acute calorie lack or starvation.

Sometimes the diarrhoeal disease itself is protracted and the health worker is in a quandary as to whether she should re-introduce some form of nutritious diet after twelve hours of saline therapy, or to wait and risk the problems mentioned above, or to ask for the patient to be evacuated to hospital.

It has proven most satisfactory to use half-Normal Saline, with sugar and Potassium added, for twelve hours, and having re-started the usual feeding, to continue the salt solution as extra fluid to avert dehydration until the diarrhoea abates.

There is an almost universal tendency to administer broad-spectrum antibiotics to children with diarrhoea. This inevitably produces problems of Monilial overgrowth and, in some cases, may increase the severity of the bowel disturbance by chemical irritation.

Some people advocate a variety of medicaments in childhood diarrhoea. Several cases of narcosis from extensive dosage with a Kaolin and Morphine mixture have been seen, and, during part of 1969, there was a vogue for administering antispasmodics which gave rise to

atropinisation.

Several infants with mild fevers and bulging fontanelles have been seen as a result of Tetracycline administration. Chloromycetin, although now not so widely used as it was several years ago, continues to give rise to occasional fatal blood dyscrasias.

The secretion-drying action of some antihistamines, has undoubtedly caused more respiratory problems than is generally appreciated. One of the greatest difficulties in managing lung infections in remote areas is the absence of adequate physiotherapy. This in itself leads to imperfect resolution of inflammatory lesions and if the bronchial mucus is rendered more tenacious than normal by antihistamines, which are commonly used non-specifically, and possibly some degree of dehydration, then chronic lung disease may well be a sequel.

Ephedrine is another drug which has widespread use, particularly in combination with an antihistamine, in respiratory infection, and periodically it causes quite severe vomiting.

Many other similar problems have been seen, and they almost always stem from lack of training on the part of health workers. One very common misconception amongst nursing staff is that virtually any antibiotic

can be used in any infection. Thus, since nurses in remote areas often initiate treatment, it happens not infrequently that inappropriate chemotherapeutic agents are chosen. For example, suspected Shigellosis may be treated with Penicillin G, or purulent Conjunctivitis with Sulphacetamide, or staphylococcic abscesses with Ampicillin and so on.

The iatrogenic morbidity is really a function of great distances and difficulty in acquiring and keeping adequately trained nursing staff on Settlements and Missions. Rapid turnover of staff in these places, particularly Settlements, renders teaching an insurmountable problem.

SUMMARY

A variety of infections cause most of the infant and toddler morbidity in Central Australia. Intestinal and respiratory infection, very often potentiated by malnutrition, make up around 80% of the problems. The remaining 20% is comprised of a multitude of other conditions including prematurity, trauma, poisoning, social and iatrogenic situations. Even in this smaller group the emphasis is on infections such as Meningitis, Impetigo, Adenitis, Osteomyelitis, Conjunctivitis, Stomatitis and so on.

The overall picture is clearly in keeping with the situation in other countries where primitive, socio-economically depressed populations are subjected to unhygienic environments which contain large amounts of endemic infection.

The rising incidence of penicillinase producing Staphylococci has caught health workers in remote areas unawares, and they have no appropriate therapeutic agents with which to treat infections caused by this organism.

Trauma and poisoning are uncommon, and congenital anomalies occur with roughly the expected frequency, although Talipes is possibly rather more common than in the white community.

Social problems related to alcoholic parents, language and cultural differences form a significant contribution to overall morbidity and, as in any community serviced by untrained or inexperienced medical workers, iatrogenic disease is not uncommon.

PART IV PROBLEMS AND SOME SOLUTIONS

The purpose of this part, and indeed of the whole investigation, is to collate the factors responsible for high mortality and morbidity levels amongst Aboriginal infants and toddlers, and to suggest some practical ways in which the current and future situations could be improved.

The biological and social elements of the general medical problems are closely integrated and inseparable. Difficulties associated with bringing about social change in a primitive group such as the Aboriginals are manifold, but the facts remain that 8-10% of the infants and toddlers are perpetually in hospital, that the Aboriginal Secotrant mortality rate is more than 20 times that of non-Aboriginals and similarly the infant mortality is six times greater. Thus prompt measures must be taken to improve these figures while appropriate gradual social changes are being wrought.

THE PROBLEM DISSECTED

Too many Aboriginal children are sick and dying. This is manifest and clearly indicative of deficiencies in their general environment, as well as being an acute challenge to the medical workers in the area.

These children die from simple diseases which, in

the non-Aboriginal community, have long since lost most of their lethal significance, except in a few small, socio-economically depressed groups. The particular ailments, which continue to decimate the infant population, are Diarrhoeal Disease and Respiratory infection. These two alone account for 80% of hospital admissions and deaths. Other infections provide a further 10% of both the morbidity load and the mortality. The only children less severely affected by the common infective conditions are the neonates.

Why do these children become sick so often and for so long, and frequently die, from simple infections of the gut or lungs? It is the answers to this question that provide the information necessary to formulate immediate life-saving programmes.

Recurrent Infection.

Overcrowded, substandard housing and total lack of understanding of personal and community hygiene combine to make the environment, in which the Aboriginals live, highly infective. Spread of organisms by direct contact, airborne droplets and flies is constant. Purulent otorrhoea and rhinorrhoea, Impetigo, Conjunctivitis, excreta, unwashed bodies and clothing all form a huge, readily available pool of infection in the community. It is small wonder that there is excessively high morbidity and

mortality from infective disease.

Response to Infection.

No genetic or physiological reason for inadequate defence against these diseases, which often reach epidemic proportions, has been discovered, and yet many children with Gastroenteritis remain in hospital for prolonged periods, and simple lower respiratory tract infections often become widespread, destructive lung disease.

Possibly the frequency with which episodes of lung or bowel infection recur leads to altered response and chronicity.

Evidence from the data collected in this investigation, however, indicates that Malnutrition is largely to blame for the severity, protracted course and, in some cases, the fatal outcome of otherwise simple infective diseases. Children, malnourished by anthropometric and other criteria, stay three times longer in hospital than their better grown peers, and stand a four times greater chance of dying.

It has been well established by workers in other countries (Scrimshaw, Taylor and Gordon, 1968) that general inanition is regularly synergistic with infection. The factors which aggravate the spread of communicable disease, also lead to marasmus in children. Lack of

maternal knowledge about food requirements and infant feeding together with very low purchasing power and language barriers set the stage for the onset of Malnutrition as soon as breast milk becomes inadequate for normal growth at six months of age.

Treatment Of Infection.

Reasons, besides malnutrition, why infective diseases are potentially fatal, stem from the scarcity of specifically trained health workers in Central Australia, delay in presentation of acutely ill patients because of a variety of cultural and other factors, immense distances to the base hospital and shortage of medical, nursing and paramedical staff in Alice Springs. These factors may lead to faulty or insufficient treatment being administered in rural areas and mediocre attention only being provided to inpatients.

The overall problem is obviously complex and the further it is dissected the more ramifications appear. Already malnutrition and several social factors have emerged as being important, besides the purely therapeutic problems associated with diarrhoea, pneumonia and other infectious diseases which in themselves are considerable.

Malnutrition and Educability.

Not only is infantile malnutrition vitally important

because it complicates the course and treatment of other diseases, but it may also retard educability, as well as physical growth, later in life. Scrimshaw et al (1968) summarised the situation as follows - "The limited physical and mental stature of adults in poor economies and their low productivity can often be traced to permanent damage in childhood that comes so largely from the synergism of malnutrition with infectious disease".

This is another compelling reason for introducing programmes to prevent malnutrition, since, if the Aboriginals are to take their place in Australian society, they must be educable.

Birth Rate.

Overcrowding is partly responsible for cross-infection. Since mortality rates have fallen, family sizes and over-crowding are increasing. The general Aboriginal population is increasing at a rate of something over 3% per annum and the birth rate is around twice that of non-Aboriginal Australians. At Hermannsburg where the mortality is relatively low, the birth rate was 60 live births per 1,000 of population in 1969. The Australian birth rate in 1966 was 19.3 (Demography, 1966).

Inadequate purchasing power and malnutrition are aggravated by rising family sizes. With the memory of

one in three young children dying still fresh in their minds, mothers are not receptive to the idea of deliberate family restriction. Nor will they be until they can be shown that the children they have will survive.

It might be thought that increased income levels and re-housing would solve much of the dilemma. Without prior re-education such programmes have been shown to fail, as has family planning without a felt need by the target community.

Communication.

Medical diagnosis and treatment can depend on meticulous history taking. Lack of communication between Aboriginal mothers and Health workers leads to poor paediatric practice. Although many Aboriginals speak some English words, very few indeed understand the spoken language and can accept a new concept put to them in English. Conversely, even fewer Europeans communicate freely with Aboriginals in any of the native dialects. Due to rapid turnover of medical personnel, except on Missions, there is little chance of a significant number learning enough Aboriginal language to be of much use.

Language barriers are the base-line of the entire medico-social problem and little progress will be made until free communication is possible.

The fact that the Aboriginals tend to live in relatively small, widely scattered language groups, often with little interchange, adds further to the obstacles in the way of general teaching programmes. Each group must be taught separately and, frequently, in different dialects.

The rapidly rising incidence of deafness also seriously aggravates the communication difficulties.

Cultural Understanding.

Without free communication it is difficult to envisage the advent of cross-cultural understanding which is necessary if black and white Australians are to co-exist happily. The non-aboriginal Australian generally feels that the Aboriginal should come all the way to white society, rather than being met half-way. This is an unreasonable attitude, if appreciable improvement is to occur in the foreseeable future.

How does a health educator explain principles of communicable disease to a group which does not understand his language and firmly believes that evil spirits are totally responsible for illness? Even at Hermannsburg, where the natives have been subjected to European ways of life for several generations, tribal medicine men are still active, and the spiritual explanation of life, sickness and death is still widely held.

Before attracting these people into European society they will have to be convinced that it offers advancement for them in all aspects of life and, in particular, hope for the future.

SOLUTIONS

Initially the morbidity, and hence, mortality, must be lowered by means of an acute rescue operation. However, unless the more basic environmental and social problems can be overcome, rapid increase in the paediatric population could make short-term difficulties grow out of all proportion.

Much has been said about the short-comings of public health and social welfare services. Upgrading of rural health facilities to improve case finding, early treatment and prevention of the infectious diseases and malnutrition is clearly necessary but the ultimate solution rests in motivated community participation in a variety of health programmes.

IMMEDIATE POSSIBILITIES

Resolution of many of the acute paediatric and other health problems in remote areas depends on providing enough suitably trained personnel in those areas. Continuous research into changing disease patterns, antibiotic sensitivities, effectiveness of various preventive programmes and so on, will be necessary to monitor

the situation.

There is no single solution to any of the questions. The answer lies in the cumulative effects of many relatively small and, in general, easily executed manoeuvres.

The first, and most obvious, constructive measures to be taken are provision of better water supplies and waste disposal, control of flies, dogs and other possible vectors of disease, and provision of adequate food supplies. It would not be unreasonable for all health and welfare personnel to be asked to learn one Aboriginal language.

Before extensive re-housing can reasonably be undertaken several considerations must be examined. Firstly have the people felt the need and are they able to make use of the facilities provided by sophisticated houses? One major task ahead of educators dealing with Aboriginals is to bring about affirmative answers to both these questions. Once satisfactorily re-housed, assuming adequate preparation, then lower mortality rates must follow. Of course there is no point in building houses at, say, Docker River, if by the time they are needed the people they were to house have moved to Adelaide.

A simple hand-book, designed with particular regard to the local conditions, could be distributed to medical

and non-medical personnel in remote areas. If such a book explained the elements of early diagnosis and treatment of the potentially lethal paediatric conditions, and its impact reinforced by relatively frequent visits from public health nurses and doctors, some benefit would follow. This sort of enthusiastic approach by government agencies would soon provoke a gratifying change in attitude by some station people.

If the Health and Welfare authorities could agree that malnutrition is a problem and that its diagnosis can best be achieved by the use of longitudinal weight charts, some standardised routine procedures could be adopted by Settlements, Missions and Stations alike. More effective maternal and child care could quite easily be established on Settlements and Missions, with minimal increase in staff and facilities. The importance of antenatal care and well-baby clinics cannot be overstated. It is in these situations that many diseases are anticipated and good rapport established.

Canteens and stores on these places could stock only foods of high nutritional value, or at least mark their prices down to make them more attractive. A good example is proprietary baby food. Some types have excessively poor nutritional content, whereas others are relatively high. Not one canteen or store manager in Central

Australia was aware of this fact, when asked during 1969. The powdered milk which is widely used in outback areas is inadequate for infants unless supplemented by Iron and Vitamin C. Once again this point is not generally appreciated. The C.S.I.R.O. Milk Biscuit has shown promise in an early trial in Alice Springs and may prove to be worthwhile introducing in rural areas. The Aboriginals cannot be expected to understand food values if their non-Aboriginal exemplars do not. Thus a case is made for health education to include all people in remote areas.

Health Education.

In order to correct faulty hygiene routines and promote improved dietary habits in remote places, teams of specially trained educators must spend considerable periods in those places. The adults in a community direct its destiny, so it is fatuous to teach children European concepts and ways of life, expecting that this will effect a substantial social change. Clearly the adults must be educated, as well as their children, if the progression through better socio-economic and physical environments to improved child health is to come about.

Educators and health workers can combine in this sort of project, since their objects are identical in

the final analysis. If teams of two women, one a social-worker and the other a public health nurse, could live in properly equipped caravans at some of the remote places for three days of each week a great deal could be accomplished. Public health routines of maternal and child health, Tuberculosis and Leprosy reviews, Audiometry, Anthropometry, Immunisations and so on could readily be performed. Sick children would be seen much sooner than the present six weekly visits allow, and morbidity due to late presentation diminished. The close and relatively continuous relationship between these teams and the Aboriginal communities would foster cross-cultural relationships and improved communication thus making health education a much easier task.

The expense of setting up this programme would be infinitesimal compared to the cost of the aerial evacuations and maintenance of acute hospital beds it would render unnecessary.

Quite often infants brought to hospital are accompanied by their mothers, who then stay around the fringes of Alice Springs, at Amoonguna or in the bed of the dry Todd River, and visit their babies during the day. This "captive" population of women could readily be housed in a hostel and taught various aspects of hygiene, baby feeding and so on. A venture similar to this has been

undertaken by the United Church in North Australia. An Institute for Aboriginal Development is being constructed in Alice Springs to consist of three units. The first, a mother training and child-care centre, is almost ready for occupation. It is intended that 30 mothers with their babies be admitted at a time, for a three week course in mothercraft, language, hygiene etc. The teaching is to take place in an open court-yard and cooking done with primitive equipment over open fires. Sleeping quarters are also designed to relate to home conditions. The teachers will be a trained social worker and a public health nurse with Aboriginal assistants. Other sections of the Institute are to include a modern language laboratory teaching the three basic Aboriginal languages to non-Aboriginals and English to Aboriginals, and a cultural centre.

This sort of project demonstrates that at least some sections of the non-Aboriginal community appreciate the need for active intervention to improve the present, unhealthy situation.

All these things so far mentioned could have a profound effect on child health in the area. However there are all the problems of poor economy, lack of employment opportunities and training for special skills, cultural and language barriers, and so on which will prevent

morbidity and mortality assuming acceptable proportions for many years yet to come.

Long-term Policies.

The future situation depends entirely on what trends are started now. If a policy of education in its widest sense, for all sections of the Aboriginal community, is initiated promptly there is hope for amelioration of all the general factors aggravating poor child health. It seems inevitable that when an improvement in socio-economic climate begins to be felt, there will be an increasing migration of young Aboriginals towards bigger centres where they feel opportunities for advancement might be available. Once these people are motivated to want proper housing, better educational and employment opportunities, and when they see security and freedom within reach, there will be little to keep them in remote areas. The common belief that an Aboriginal has such close associations with a particular piece of country that he cannot leave it has been proven false over and over again. This trend has been noticeable in South Australia for many years, with steadily increasing proportion of the State's Aboriginals actually living in Adelaide, where their early childhood mortality is similar to that of their non-Aboriginal neighbours.

As an adjunct to the gradual narrowing of the socio-

economic gap by education at all ages and levels, the non-Aboriginals must be confronted by an extensive and carefully designed programme. A series of television films could be produced to demonstrate that Aboriginals and non-Aboriginals can live in harmony and that the days of the primitive savage are over and so on. The non-Aboriginals will need to be conditioned to take in their stride this expected migration, and accept Aboriginals as they have done Italians, Greeks, Chinese and Indian Students and many others. Possibly the inevitable emergence of scores of Aboriginal sporting heroes will help assimilation.

Australia at this moment has an excellent opportunity to anticipate racial problems with regard to the minority group of Aboriginals. If enlightened, cross-cultural education is delayed the situation will become much more difficult to control, because the first generation of literate Aboriginals will be anti-white Australians.

Unless these social changes can be brought about, the childhood morbidity and mortality will remain high, no matter how excellent local health services become.

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APPENDIX - Facsimile of instruction sheet issued to nurses in remote areas.

DIARRHOEA IN CHILDHOOD

1. CAUSES:
Childhood diarrhoea, with or without vomiting, is usually caused by an infection of the bowel. The commonest cause is a virus which may also produce a sore throat, red ear drums, cough etc.

Sometimes specific germs such as Shigella, E. Coli and Salmonella may cause diarrhoea.

2. DANGERS:
Diarrhoea especially in association with vomiting can be fatal. Small babies and malnourished children are especially at risk. The inflamed bowel loses large amounts of fluid, which may be clear and watery, it may contain strings of mucus, food particles and even bright blood. The loss of fluid, unless replaced, produces "dehydration" which is the dangerous part of the illness.

Dehydration:
The fluid lost via the bowel in diarrhoea and vomiting contains many substances which are important. Besides water, the next most important is Salt. Dehydration has been called the "Low Salt Syndrome" and as it develops, the following signs appear:-

- weight loss.
- thirst, dry mouth, restlessness.
- small amounts only of dark urine.
- fever, decreased sweating.

As the situation worsens, if no treatment is given, further signs appear:-

- weakness, pallor and anxiety.
- feeble pulse.
- cold hands and feet.
- rapid breathing.
- eventual unconsciousness.

The best way to assess the severity of dehydration is to measure loss of bare body weight. Thus it is important to weight a child carefully at the first sign of diarrhoea. The table expresses severity of dehydration as a function of weight loss, (e.g. If a 20lb. child loses 1lb. the dehydration is 1/20th. or 5%)

Severity of Dehydration:-

WEIGHT LOSS	SEVERITY OF SITUATION	TYPE OF TREATMENT
1/40th or 2½%	Mild	Dietary
1/20th or 5%	Moderate	Vigorous Fluids
1/15th or 7%	Severe	Fluids & Doctor
1/10th or 10%	Very Severe	Urgent Hospital (Parenteral fluids)
1/8th or 12%	Extreme	Intravenous Fluid
1/7th or 15%	Lethal	

Prolonged diarrhoea can cause exhaustion of supplies of certain digestive enzymes, thus making re-establishment of normal diet difficult.

3. TREATMENT

The dehydration, NOT the infection itself, is dangerous and therefore treatment is aimed at preventing it. Of course, very often a co-existing respiratory infection will have to be treated as a separate entity, say, with I.M. PENICILLIN.

The simplest and most effective early treatment of infantile diarrhoea is as follows:-

i. Weigh child bare.

ii. Give NOTHING by mouth for 12-24 hours except SALT/SUGAR/WATER mixture made to this recipe.

½ TEASPOON SALT
2 TABLESPOONS OF SUGAR
1 PINT OF WATER
This is often called DEXTROSE/SALINE.

The sugar content helps to keep energy available. A small amount of orange or lemon flavouring may be added. If this mixture is flatly refused, a 1 in 16 sweetened condensed milk may be used.

If possible breast feeding should be interrupted, but this is often difficult. In any case, the DEXTROSE/SALINE must be given to make up fluid loss.

If vomiting is present, small sips of the mixture are given frequently until vomiting stops.

A guide to the amount of mixture needed can be obtained from the weight of the patient. Any weight loss is made up by increasing the amount.

For example, a weight loss of 6 ozs. means the child needs 6 ozs. of fluid replaced. It does not matter how frequently the child is weighed. Passage of light coloured urine is a good sign.

iii. After 24 hours -

a. It will be clear whether the situation is becoming worse, in which case medical advice should be sought. It may be that the bowel is so irritated that even the DEXTRO-SALINE passes through without being absorbed. If the situation does worsen and the child loses more weight despite treatment, then intraperitoneal or intravenous fluid is needed. The technique of intraperitoneal infusion has been used in remote areas in other countries and is a suitable emergency measure for use in Central Australia. It has been used successfully several times on various Missions, Settlements and Stations. The technique is described later.

b. If the patient's condition is the same or improving after 24 hours, then gradual introduction of sweetened condensed milk (1 part milk to 9 parts water) is indicated. The bigger child may be given salty biscuits with a smear of "Vegemite" (but no butter). Over the next few days, the milk is gradually increased in strength and baby rice, cereal etc., can be added.

The diarrhoea may increase again temporarily after food is started, but usually it is best to continue. It may be up to ten days or more before the child is back to normal.

iv. Antibiotics

Treatment with antibiotics is NOT indicated unless the specific germ is known and it is felt that sterilisation of the stools is necessary for the protection of others e.g. a younger sibling. Almost all cases of gastroenteritis get better without antibiotics. The untreated infection clears itself after a period.

v. Antidiarrhoeal Agents

Mixtures such as Kaolin, Kaolin and Morphia, Kaomycin, Arobon, Cornflour etc. serve only to aggravate an already irritated gut and have no place in the treatment of childhood diarrhoea. Lomotil has been used, but is of doubtful value. Antispasmodics with Atropine-like side effects such as Eumydrin, Buscopan and Merbentyl likewise are not indicated.

4. INFECTIVITY

Gastroenteritis is a highly infectious disease with a short incubation period. Anyone caring for patients should be most fastidious about hand washing before and after handling anything to do with the patients. All instruments such as thermometers and bottles should be kept in antiseptic lotions, and the patient isolated from the rest of the community.

It is important to realise that in a "gastro" ward, individuals may have different causes for their disease, and are therefore infectious to each other.

INTRAPERITONEAL INFUSION

The technique of Intraperitoneal Infusion is simple and safe. It is applicable to the treatment of severe and/or progressive dehydration in situations where Intravenous Therapy is not possible.

Indications.
Dehydration, from any cause, when oral rehydration is ineffective or impossible and when Intravenous treatment is not available.

Contraindication - Distended abdomen.

Technique.

1. The abdomen, one inch above the umbilicus, or midway between umbilicus and pubis in the midline, is swabbed with antiseptic solution.

2. Using ordinary Intravenous giving set and the wide bore needle provided (16 - 19G), either Normal Saline or Half Normal Saline in 2½% Dextrose, previously warmed to body temperature, is set up as for I.V. infusion.

3. Traction, between forefinger and thumb, at the umbilicus renders the puncture site taut, and the needle on the giving set is pushed into the subcutaneous tissue. The clip on the tubing is released and the needle advanced until the Saline flows freely. The needle is kept perpendicular to the skin. Local anaesthetic may be used.

4. An assistant should control the patient's movements by holding the ankles with one hand and the arms above the head with the other.

5. The fluid may be run in as quickly as possible and when the infusion is complete the needle is withdrawn and the puncture sealed with Collodion.

The volume of fluid infused depends on the size of the patient and the severity of the dehydration. Usually a 5 kg. child can take 250 mls. and a 10 kg. child 500 mls. at a time. Sometimes the severity dictates that the infusion be repeated several times at say four hourly intervals.

At any stage during the infusion, if the patient becomes distressed, the procedure should be stopped. This is uncommon and is usually due to the fluid being too cold.

The advantages of Intraperitoneal Infusion are that the fluid is absorbed rapidly, the procedure is quick and requires a minimum of skill and experience in the operator.