

https://dspace.org/

A review of measles notification for Soweto for the period 1984 to 1986

Hukins, Graham

1989-02-16
https://hdl.handle.net/10539/26131

Downloaded from a DSpace repository. https://dspace.org/

A REVIEW OF MEASLES NOTIFICATION FOR SOWETO FOR THE PERIOD 1984 TO 1986.

GRAHAM HUKINS.

A SHORT REPORT SUBMITTED TO THE FACULTY OF MEDICINE, UNIVERSITY OF THE WITWATERSRAND IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE MASTER OF MEDICINE IN THE DISCIPLINE OF COMMUNITY HEALTH.

I hereby declare that this short report is my own work. It is being submitted in partial fulfilment for the degree of Master in Medicine in the discipline Community Health at the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at any other university.

This study has received ethical approval from the University of Witwatersrand's ethical committee for research on human subjects. Protocol No 16/2/89

Table of Contents:

0. Summary. 1

01. Introduction.

1.1. Measles in different countries. 2
1.2. Measles in South Africa. 3
1.3. Measles and its complications. 4
1.3.1. Malnutrition. 4
1.3.2. Secondary infections. 6
1.3.3. Associated neurological conditions. 8
1.3.4. Mortality. 8
1.4. The spread of measles. 10
1.4.1. Hospitals and clinics and the spread of measles. 11
1.4.2. Social factors predisposing to the spread of measles. 11
1.5. The measurement of measles. 12
1.6. Natural and acquired immunity of measles. 12
1.6.1. The immune response to measles virus. 12
1.6.2. Variations in host immunity. 13
1.7. The prevention of measles. 14
1.7.1. The vaccine. 14
1.7.2. Passive immunity. 16
1.8. The timing of vaccination. 16
1.8.1. In developed populations. 17
1.8.2. In developing populations. 17
1.8.3. Vaccine failure. 18
1.8.4. The clinical complications of vaccination. 18
1.8.5. Contraindications and safety precautions for vaccination. 18
1.8.6. The failure to seroconvert. 19
1.9. The cost efficiency of measles vaccination. 22
1.10. The impact of the vaccine in the United States and China as an example of efficacy. 22
1.11. The promotion of measles vaccination. 23
1.11.1. Organisations and the media. 23
1.11.2. Mass campaigns. 23
1.11.3. Gaining acceptance for vaccination programmes. 24
1.11.4. Evaluating measles vaccination coverage. 25
1.12. Background information on the health services in Soweto. 26

02.The objectives of the study. 28

2.1. The annual incidence and the attack rate according to age, district, hospital and clinic. 28
2.2 Recommendations on information on notification forms and further research needs. 28

03. Methodology. 29

3.1. The study design. 29
3.2. Population. 29
3.3. Sample. 29
3.4. Measurements. 29
3.4.1. Information on measles data collection in Soweto. 29
3.4.2. Definition of rates. 30
3.4.3. The age specific attack rate. 31
3.4.4. Measles attack rate by residential district. 31
3.4.5. Measles notification rate for clinics. 31
3.5. Data analysis. 32
3.6. Implementation. 32
3.7. Limitations. 32
3.7.1. The criteria for the diagnosis of measles. 32
3.7.2. The undernotification of measles cases. 32
3.7.3. The underestimation of the population of Soweto. 33
3.7.4. Residential address. 33
3.7.5. Recording errors. 34

04. Results and discussion. 35

4.1. The Incidence of measles. 35
4.2. The Attack rate according to age. 36
4.2.1. Evaluating changes in the measles vaccination policy. 38
4.3. Attack rate according to residential district. 39
4.4. Attack rate according to hospitals and clinics. 42
4.5. Vaccination status of measles cases. 44
4.6. The seasonal variation of measles. 47

05. Recommendations. 49

5.1. The prevention of measles. 49
5.2. Notification of measles cases. 50
5.3. Research needs. 51

06. Conclusion. 52

07. Acknowledgements. 53

08. References. 55

09. Appendices 68

SUMMARY.

Prior to 1986, Sowetan children were vaccinated against measles on two occasions at 6 and 12 months of age. In order to comply with the World Health Organisation's recommendation for African countries, this schedule was changed in January 1986 to one vaccination at 9 months of age.

At the time of this change in the measles vaccination schedule, there was a reported increase in the incidence of measles at hospitals that serve the population of Soweto. This was thought to represent a general increase in the incidence of measles in Soweto.

This study set out to investigate this reported increase in the incidence of measles over the period. A retrospective record review of notification forms was undertaken.

The results showed that the incidence of notified measles cases in 1986, compared with 1984 and 1985, had not increased. However the median age at which children presented with measles had decreased from over 40 months in 1984/85 to 24 months in 1986.

In addition the peak in the age specific attack rate had changed in the period under review from over one year of age in 1984/85 to less than one year in 1986. More cases were notified from hospitals in 1986 than in 1984 and 1985.

Population migration of unvaccinated children into Soweto appeared to account for up to 100 per cent of cases in certain districts.

This emphasises the need for the implementation of a national strategy for measles prevention in order to protect all children.

Awareness programmes directed at mothers to promote measles vaccination and to remind health care professionals of the legal requirements to notify measles cases would help to control the spread of measles and would provide an assessment of the full extent of the problem.

01. INTRODUCTION.

In order to give background to the study a literature review was undertaken which encompasses measles the disease, its complications and preventive strategies. The cost efficiency of the vaccine, international experience and the optimal age of vaccination are discussed. To end the introduction the health service in Soweto is reviewed.

1.1. MEASLES IN DIFFERENT COUNTRIES.

Measles is a major global problem deserving the attention of all nations since it is an important cause of infant and child morbidity and mortality, particularly in the developing world.(1)
The measles virus is very infectious. In Greenland Christensen found that 99.9% of a previously unvaccinated, unexposed population developed measles when the wild virus was reintroduced.(2)
The impact of measles measured by surveillance data on morbidity and mortality, and range and severity of complications is extremely variable from country to country.(3,4)

Before the introduction of measles vaccine in the United States of America, the highest case-fatality rates were in the very young and in the children with low socioeconomic status.(5)
Since the introduction of measles vaccine the impact of measles has been greatly reduced, as is the case reported from North America and certain Central and South American countries such as Cuba, Chile and Costa Rica.(3)

Measles is widely recognised to be a severe illness in the developing world; Ghosh and Dhatt in Madras in 1969 considered measles to be more serious than diphtheria.(6) In developing countries, some epidemics have killed one fifth or more of the population previously unexposed.(4)

Measles is more severe in countries where childhood malnutrition is common.(7) It has been postulated that the severity in developing countries is related to both the young age at which measles is usually contracted and the high prevalence of malnutrition.(8)

In India, it is thought that "the impact of measles in the overcrowded, expanding, turbulent cities is often underassessed while the fatalistic attitude in the rural areas makes control more difficult."(3)

In Africa, measles remains a problem. It is an important cause of morbidity and mortality, particularly among socioeconomically deprived communities. Respiratory complications and diarrhoea are relatively common during and following measles infections. The case-fatality rate for all children in developing countries is estimated to be 1 to 5%. However, the true level of mortality attributable to measles in developing countries in Africa, although thought to be high, is unknown.(9,10,11)

1.2. MEASLES IN SOUTH AFRICA.

South Africa has reported severe outbreaks of measles during the past two centuries.(12,13,14) During 1985, at least 2428 patients with measles were admitted to two hospitals in Natal. The mortality rate among these patients was 14,7%.(15) At Baragwanath hospital, from January 1987 to July 1987, measles accounted for 5,8% of all paediatric admissions, compared with 1,6% in 1982. (16) Thirty six percent of these children were 9 months of age or less.(16) During the same period, from January to July 1987, measles in children 9 months or younger accounted for 27,2% of measles admissions in Cape Town(17) and between 20 and 45% of measles admissions in Natal.(18)

1.3. Measles and its complications.

In developed populations measles is often considered a mild disease of childhood.
In Europe and America a proportion of non immunised children do not appear to contract measles after exposure. It was reported from Britain that 15% of children escape attack after exposure.(19) These children may have had a subclinical attack of measles.
In East Africa 25% of unvaccinated children 1 year of age with no previous history of clinical measles had antibodies.(20) In New York, Krugman showed that while the haemagglutonin inhibition (HI) antibody level declined to 1:8 by 1 year of age, in a few infants, the HI level suddenly rose during the period 6 months to 1 year.(21) These children experienced a subclinical infection in the period of decline in the maternal antibodies.(4)
Subclinical infections occur in this age group only in areas where the disease is endemic.(4)
However, the necessity to control and eradicate measles is better appreciated when the extent and severity of the morbidity and mortality in developing populations is understood.
Many of the complications of measles eventually require hospital admission. (22,23,24) Factors which seem to underlie the unusual severity of the disease in Africa are malnutrition, a lower mean age of incidence of disease during epidemics, the extremely high rate of secondary pyogenic infections, involving the skin and the respiratory tract, and the frequency of stomatitis and diarrhoea inducing dehydration.(22)

1.3.1 Malnutrition.

Malnutrition in children can aggravate or be aggravated by measles infection.
Measles in malnourished children differs markedly from the disease as usually seen in well nourished children.(22)

Dosseter et al have said that malnutrition depresses immunity which leads to prolonged measles infection and further damage to the immune system.(25) The nutritional status of the child before and during the attack of measles is the dominant factor in producing the severe form of the disease.(4) In Nigeria measles is the acute infection most likely to precipitate acute kwashiorkor in children.(26) Following measles infection children have been reported to lose up to 10% of their body weight.(26) In Kenya, the severely malnourished patients suffered a more prolonged and complicated illness than the well nourished. These patients suffered an average of four complications, 70% had severe stomatitis and 77% had diarrhoea.(27) In India, among children hospitalised with measles, 89% of those poorly nourished had more than one complication e.g. pneumonia, otitis, stomatitis or diarrhoea, while only 20% of the well nourished had more than one complication.(28)

Measles in the poorly nourished results in severe changes of the mucous membranes of the mouth, tracheal-bronchial tree, and intestine.(22) The inflammatory process of the mucous membrane of the intestine leads to a protein-losing enteropathy.(23,24) In Guatemala, Scrimshaw et al noted that diarrhoea was three times more frequent among those poorly nourished.(29) Diarrhoea associated with measles is recognised as a danger for dehydration and increased mortality.(30) Often oral rehydration therapy is not adequate and parenteral fluid replacement is required. Death following diarrhoeal complications of measles occurs in both well and malnourished children and is associated with a variety of pathogens.(22) Measles associated with prolonged diarrhoea of more than 7 days duration significantly increases the risk of mortality.(31)

Foster reported that children below the weight for height Harvard standard who survive, recover slowly and catching up weight can take longer than 3 months.(32)

Children with more severe skin lesions tend to develop sequelae to measles. It has been suggested that this is because of the lack of some nutrient or nutrients which may cause a greater degree of perivascular extravasation, producing a darkening of the rash of the skin and exfoliation. Equivalent changes in the epithelium of other organs cause other complications.(4) This association between a darkened rash and the severity of the disease was recognised as long ago as A.D.850 by the Arabian physician Rhazes.(33)

1.3.2. Secondary infections.

The depressed cellular immune system increases patients' susceptibility to secondary infections.(34) Studies have shown that gamma interferon produced mainly by T lymphocytes is reduced in patients with measles.(35)

It has been reported that a profound lymphopenia (<2,000 lymphocytes/ ml.) during the exanthem stage distinguished those African children who subsequently died or developed chronic chest disease from those who recovered.(36) In these children an excess of HLA-AW32 (histocompatibility antigen) has been found compared with controls.(37)

For up to five weeks following measles infection, a child has an increased susceptibility to intercurrent infections with both viral and bacterial organisms.(38) Adeno type 2,3, and 7 and herpes viral infections are the most prevalent.(3)

Other viral infections are caused by cytomegalo, para influenza, respiratory syncytial and coxsackie viruses.(39) Bacterial infections are less common and therefore secondary infections that occur during the acute attack of measles are usually viral and hence not responsive to antibiotic treatment.(3)

Both laryngitis with airway obstruction and bronchial pneumonia are relatively common sequelae of measles in West Africa.(4)
The latter is a common, serious complication of measles often necessitating hospitalisation.(4)
Pneumonia has been found to be the commonest lethal complication of measles. (39) The pneumonia is usually divided into early and late onset pneumonia.(22) Giant cell pneumonia caused by the measles virus typically begins early in the course of the illness before the rash fades. More frequently, the pneumonia appears after the initial symptoms of measles have subsided.(22) This late on-set type is usually caused by viruses other than the measles virus.(22) Bacteria are seldom the cause of pneumonia.(22) At autopsy, of 18 coloured and black toddlers from South Africa who died from pneumonia following measles, it was shown that adeno virus, measles virus and herpes virus caused more than half of the late on-set pneumonias.(40)

In Columbia, Dover performed lung punctures before antibiotic therapy was commenced on 15 children with late onset pneumonia, the cultured samples yielded bacteria in only two cases.(39) Measles may also activate a primary tuberculous lesion in the lung.(41)

Conjunctivitis with involvement of the conjunctival sac is common and measles may lead to damage or destruction of one or both eyes.(4) Xerophthalmia that results in blindness occurs in areas of the world, where diets are deficient in vitamin A.(4) In these areas, for example Kenya and Indonesia, more than half of the incidence of blindness may be attributable to measles.(4,42,43)

1.3.3. Associated neurological conditions.

There is an hypothesis that a host cell-dependent lack of synthesis of M protein in the brains of patients following measles infection may be the cause of subacute sclerosing panencephalitis.(SSPE).(36) A few children (0.5-1.0 per 100 000) develop SSPE on average 7 years after clinical measles infection.(44) Another chronic neurologic disease linked largely by indirect evidence to measles virus is multiple sclerosis (MS).(45,46)
Increased levels of measles virus-specific immunoglobulin have been demonstrated in the cerebrospinal fluid (CSF) of ~50% of patients with MS, and in the CSF of a smaller percentage of patients with other neurologic disease.(47,48)
The low serum to CSF antibody ratios recorded have led to the suggestion that the antibodies in the CSF of MS patients is produced within the central nervous system.(49) Demographic and epidemiological studies have proposed an hypothetical incubation period of about 15 years for MS.(50) If infection with wild measles virus, but not with vaccine virus is in any way responsible for MS, a reduction in the incidence of this disease should occur in those countries that have promoted measles vaccination for the past decade or longer.(36)

Encephalitis and convulsions, following measles, have been described from West Africa. One and a half percent of admissions gave a history of convulsions and 2.5% developed them after admission. Mortality in these children is high.(51)

1.3.4. Mortality.

Case fatality rates vary in different parts of the world. Epidemics in the Andean Spanish colonies in South America in 1531 recorded mortalities higher than 50%.(52)

A distinction is made between death during the acute phase and deaths during the 9 months after the acute phase of measles. During the acute phase of the disease, case fatality rates in the community may range from 0.9% to 36%. (53,54) It would seem that the mortality in the period 3 to 9 months following the acute attack, is as high as during the acute phase. Some reports show that more than half the deaths after measles occurred in the period 3 to 9 months after the outbreak.(54)

Some variables that influence mortality due to measles are: The size of the family, the nutritional status of the child during the attack, the occurrence of secondary infections and the age of onset of measles.(4) In large families, mortality rates are high in young children, especially if they are secondary cases.(55) It was hypothesised that measles resulted in a significant weight loss, immunosuppression, or both, which in turn increased mortality in measles cases from undernutrition, malaria, diarrhoea, or other infectious diseases.(54)
Koster in Bangladesh found that measles and diarrhoea acted synergistically to increase mortality.(56) During the Nigerian Civil War observations made at a time of severe famine revealed that up to 15% of those in a medical centre for the treatment of kwashiorkor died.(57) In Indonesia Foster reported that only 10% of hospitalised children above the Harvard standard of weight for age died, while 18% of those below this standard died.(32)
The case fatality rate from measles was 2.3 times greater among severely malnourished children than among better nourished children, while in Bangladesh, it was 4.3 times higher.(28)

Variation in virus virulence does occur.(4) Hospital acquired measles is particularly severe with a high case fatality rate. (58,59)

1.4. THE SPREAD OF MEASLES.

Measles virus spreads within populations by the infection of a series of susceptible individuals. For the virus to remain endemic, there must be a constant influx of susceptibles, provided by birth or immigration.(10) The most common route of transmission of the virus from one individual to another involves both direct and indirect contact via respiratory droplets.(22) Those in close proximity to the source inhale the virus. It multiplies and invades the tissues of the susceptible host.

The most infectious stage of the disease is from the onset of fever, before the rash appears on the fourth day, until 2 days after the rash has appeared.

In well nourished children viral excretion continues for 3 days. In malnourished children, viral excretion can continue for up to three weeks. The persistence of the virus in lymphocytes and intestinal cells suggests that there is a delay in the production of lymphocytes that are competent to destroy these cells. As a result, the virus multiplies for a longer period, more cells are infected, and the disease is more severe. In addition, the depressed cellular immune system increases the susceptibility to spread the infection.(25)
Therefore in areas of poor nutrition, as is the case in some areas of Soweto, the likelihood of viral transmission from convalescent patients may increase.(25,56) Patients who have been admitted to hospital with poor nutrition should remain in isolation for at least three weeks following recovery.

1.4.1 Hospitals and clinics and the spread of measles.

Cross infection in hospitals and clinics is promoted by the close proximity of patients in these institutions.(60) Over a 16 month period, 77 consecutive cases of life threatening measles cases were admitted to the Red Cross War Memorial Children's Hospital intensive care unit. Twenty of these 77 probably caught the disease while in hospital.(61)
It has been suggested that, in the case of infection with measles, the crowding dose appears to be more important in promoting a severe form of the disease than the patient's resistance.(62)

1.4.2. Social factors predisposing to spread.

An outbreak of measles may be precipitated by social events, particularly after the population has congregated for some or other reason. For example, at the beginning of a school term, or for a celebration. In Nigeria during the dry season following harvests when many markets and festivals occur, transmission is greater.(63)

In large families the average age for contracting measles is lower, than in small families consisting of 1 or 2 children, probably because the opportunities for introducing the virus into the family are so much greater.(64) When the virus is introduced into a family, it spreads rapidly and almost all those not yet immune develop the disease. Hope Simpson observed a secondary attack rate of 75% among large families.(65) The large, and extended families that are common in developing populations increase the opportunity for contact and viral transmission.(22)
Similarly in cities, where contact among many children is common, the average age of acquisition of measles is generally lower than in rural areas.(22)

1.5. The measurement of measles

Under notification for measles is a universal problem.(66) Only health workers are presently allowed to document and notify measles. Alternative methods need to be considered. The initial notification of a suspected case of measles should be made simple and need not necessarily be made by health workers only. Who should be allowed to notify?; and whether telephonic notifications should be acceptable? are questions posed in the literature and may be considered for Soweto.(16) With minimal training notifications can be made by medically unskilled people. In The Gambia, use was made of village elders to report on the progression of the outbreak of measles and to ensure complete ascertainment of early cases.(9)

Within Soweto this task could be performed by child minders and teachers working in creches and at schools. Suspected cases of measles could then be referred to the local clinics, community health nurses or general practitioners for confirmation of the diagnosis.

1.6. NATURAL AND ACQUIRED IMMUNITY OF MEASLES.

1.6.1 The immune response to measles virus.

The natural immune response of people previously unexposed to measles is the production of lymphocytes that exhibit measles virus-specific antibody and cytotoxic responses.(67,68) Protection by measles specific, cell mediated response in the absence of antibody has been reported.(69) Some children with agammaglobulinemia apparently respond normally to measles infections in the absence of detectable antibody.(36)

1.6.2. Variations in host immunity.

Individual variation in host immunity does occur. This is independent of the existence of residual maternal antibody, which is known to protect young children.(4) The duration of post-vaccinal immunity beyond one year of age has not been studied, but it can reasonably be expected that immunity after one vaccination at 7.5 months will last for at least 3-5 years.(70)
The immune status in previously unexposed children that are no longer protected by maternal antibody depends upon the availability, stability and potency of the attenuated virus vaccine, route of administration, timeous age at vaccination, adequate seroconversion and any special conditions that might disrupt the health service and affect compliance, for example floods, civil unrest or an inadequate health service to vaccinate the community against measles.(4)

The immune status of the population at risk is the important factor in preventing cases. It is difficult to decide what constitutes a minimum protective level of antibody after vaccination since the laboratory tests used in the past have varied widely in sensitivity and few have directly measured neutralising antibodies by plaque inhibition(PI).(71) Krugman has argued that haemagglutonin inhibition(HI) antibody levels following vaccination may decline to less than 1:8 or even undetectable levels yet provide clinical protection, probably because of the anamnestic response following re-exposure to the wild virus.(72)

The detection of measles antibodies.

The discrepancies between various studies for the detection of residual maternal antibodies may result because of the first dilution at which sera are tested.(73)

Bass found that two thirds of sera that would have been stated to be negative if tested at 1:10 were in fact positive with plaque inhibition tests.(74) From results carried out with the classical antigen it was generally thought that passive immunity of maternal origin was depleted by 6 months of age. However, with the Tween-ether antigen, Stanford found that 35% of children in Uganda still had detectable antibodies at 6 months of age.(75) Puffer et al found that at 6 months of age, at least 20% of children will still have maternal antibodies.(23) Following vaccination only 60-80% of these children will seroconvert and develop protective immunity.(23)

In a collaborative study by the Ministry of Health of Kenya and the World Health Organisation it was found that 90% of children no longer have maternal antibodies at 7-8 months of age.(70) In this study it was precisely at that age that the incidence of measles began to rise sharply.(70)

1.7. THE PREVENTION OF MEASLES

1.7.1. The vaccine.

The vaccine is prepared by attenuation of the measles virus.

Several vaccines have been used to vaccinate against measles.

i. The Edmonston strain; First used about 20 years ago, this has undergone 40 passages in cell culture in the process of attenuation.(36) In the early 1960's, Spencer, in the Johannesburg City Health Department, conducted controlled trials with the Enders Edmonston B strain of vaccine under the auspices of the World Health Organisation. The aim of the trials was to demonstrate the clinical reactions, immunogenic response and protection when children 6 months of age were immunised against measles. Following the implementation of this programme, some children, who had been immunised at six months and were thought to be protected by the vaccine, presented with clinical measles.

This was attributed to the presence of maternal antibodies at the time that they were vaccinated which adversely effected seroconversion.

ii. The Schwartz strain; It is a derivative of the Edmonston vaccine and has undergone 77 passages.(36) It is usually used in infants older than 9 months, where optimum seroconversion rates of between 85% and 90% have been reported in Africa.(73,76)

The Schwartz strain is equally immunogenic but has less adverse clinical reactions than the Edmonston strain.

Studies with the Schwartz vaccine have shown that optimum seroconversion rates fall sharply if children are immunised earlier than 9 months because maternal plaque-neutralising (PN) antibody neutralizes the attenuated virus.(36)

iii. The Moraten and Zagreb strains; Both these vaccines are also derivatives of the Edmonston strain. Results of reduced seroconversion were reported by Parkman and Amler in 1983 for the Moraten and Zagreb strains when children less than 9 months of age were immunised.

It was shown that the failure rate was between 40-100% when infants were immunised with this vaccine at 5 months of age.(77,78) However, contrary to these findings Sabin et al, in 1984, found that an aerosol of undiluted human diploid cell Zagreb strain vaccine, produced almost 100% seroconversion rates in 4-6 month old Mexican infants.(80) Administration of this virus by the respiratory route has been suggested because the virus replicates more readily in the human lung fibroblasts than does the chick-cell-adapted Schwarz strain.(79)

In addition, administration via the respiratory route in young infants would avoid neutralisation of the vaccine by maternal measles antibodies, because maternal measles antibodies are immunoglobulin G and therefore not secreted on the surface of the infant lung.(71)

Vaccine aerosol given either by mask in a dose of 3500 or 7000 plaque forming units(PFU) or from a plastic bag at a dose of 7000 PFU raised haemagglutonin inhibiting (HI) or plaque-inhibiting measles antibodies 16 to 24 weeks after vaccination to a titre of 1:8 or greater, in all but 3 of the 51 children so vaccinated in the Gambia.(71) Aerosol inhaled from a mask or bag was generally well tolerated.(71) Children who cried did not have a lower antibody response than the others. Human diploid cell vaccines used to immunise 4-6 month old infants could thus be conveniently given at the same time as the third dose of diphtheria, pertussis, tetanus and polio vaccines.(5) This vaccine would be of particular use for vaccinating hospitalised infants who may be at high risk of developing measles.

In 1983, Sabin et al found that the Edmonston-Zagreb vaccine in a dose of 5000 plaque forming units given subcutaneously to 5 month old children also produced almost 100% seroconversion.(80) If this is so it would be of particular use in community vaccination programmes.

1.7.2. Passive immunity.

Passive immunity is available with the use of immune globulin-containing measles antibody which provides passive protection against clinical measles.(36) This should only be considered for those patients who are at risk for developing serious complications of measles.

1.8. THE TIMING OF VACCINATION.

The timing of vaccination depends upon the type of vaccine used and the method of administration. To date, a derivative of the Schwartz vaccine has been in use in Soweto. Before 1986 the policy in Soweto was to vaccinate against measles in children at both 6 and 12 months of age. In 1983 the World Health Organisation (WHO)

recommended that African countries give one dose of vaccine at the age of 9 months. In January 1986, the health authority in Soweto implemented this recommendation.

1.8.1. Developed populations.

Presently the WHO recommends that the optimal age for measles vaccination in a developed population, using the Schwartz vaccine sub cutaneously, is 15 months. This is because maternal antibodies have disappeared or are less than 1:6 by then and the population has ready access to the health service.

1.8.2. Developing populations.

The optimal age for measles vaccination in a developing population is controversial.(54) The vaccination schedule should be based upon three sets of basic data: the pattern of waning measles maternal antibodies during the first year of life; the efficacy of vaccination as determined by age specific seroconversion rates; and the age specific incidence of measles in the population. Generally, a lower age specific attack rate and greater severity of complications in a developing population require that children be vaccinated against measles at an earlier age, in fact from 6 months of age. However, measles vaccination at 6 months instead of at 9 months is considered less effective because maternal antibody is more likely to be present and this reduces seroconversion. However, even vaccination at 9 months of age is not ideal as some children vaccinated at less than 1 year of age will show no detectable haemaglutination inhibition antibody (HI) eight months after vaccination.(81)

The detection of antibody is dependent upon the method used.(73) For the detection of HI antibodies different results may be obtained according to the type of antigen used.(73) Plaque inhibition tests are 10 times more sensitive than HI tests. (73)

1.8.3. Vaccine failure.

Reasons for vaccine failure are:
i. Vaccine administration at too early an age
ii. an inappropriate anatomical site of vaccine administration or
iii the use of non viable vaccine.
The reasons for i and ii above will be discussed in the section on the failure to seroconvert.(1.8.6.)
Vaccine failure due to the inadequate maintenance of the cold chain is a problem. This is not quite as critical as it used to be. With the advent of the new stabilizers that retard heat inactivation, in field trials, >85% of children 9 months of age or older seroconverted after receiving vaccine that was stored at 23-25 degrees Celsius for 7-8 days.(82) However, once the vaccine has been reconstituted into a liquid form, it is particularly heat-labile.(10) The rehydrated vaccine remains potent for 4-6 hours. Health workers need to be continually reminded of this.
In the Soweto clinics electricity and refrigeration facilities are available to store vaccines.
Even where the supply of electricity cannot be depended upon, technological developments improving refrigeration have made the storage of vaccine possible.(83,84,85)

1.8.4. The clinical complications of vaccination.

The known side effects of measles vaccine are:
a mild fever beginning on the fifth day after vaccination and usually lasting several days; a transient rash; encephalitis and encephalopathy (in less than 1 per million doses). The latter is lower than the observed incidence of encephalitis of unknown aetiology in the general population.

1.8.5. Contraindications and safety precautions for vaccination.

There are few contraindications to vaccination other than age and pregnancy. Patients with immune deficiency or altered immune response, eg in the auto immune deficiency syndrome, should not be immunised. For intercurrent febrile illness, measles immunisation should only be postponed at the discretion of the physician.(86)

Moderately malnourished children 60%-80% of normal weight for age appear to suffer no more untoward effects and to produce as much antibody after vaccination as well nourished children.(87,88)
There is no direct evidence to implicate vaccine virus in either acute measles encephalitis or subacute sclerosing pan encephalitis (SSPE).(36) Attenuated measles virus vaccine has been widely used in the United States since 1968.(89)
Studies on patients with cystic fibrosis and with nephrosis have shown that they tolerated vaccine well and developed the usual serological response.(90)

In both these conditions low serum albumin is found which closely parallels the protein deficient Nigerian children.(91) It is important to prepare mothers for the possible side effects, at the same time ensuring them of the advantages, following measles vaccination. This would help to dispel any negative thoughts or attitudes that the community might develop towards a reaction following measles vaccination.

1.8.6. The failure to seroconvert under 1 year of age.

The initial response to the finding that some children under one year of age failed to seroconvert was a suggestion that they be revaccinated at 1 year of age. However, vaccination of children less than 8 months of age may change the immune response and leave all children so vaccinated susceptible to measles.(81)

In a study done in Los Angeles, only 49% of children responded optimally to revaccination.(81) Black et al, working in third world conditions found that children who failed to seroconvert following an initial "early" (ie. before 9 months) and second (at 9 or 12 months) dose of vaccine, had reduced levels of haemaglutination inhibition (HI) antibody titres after several months.(64)

The children were considered to be immunologically sensitized but not immune to infection. What is not clearly understood, at this stage, is what the consequences are for these children when they are exposed to natural wild measles virus. The following questions need to be answered. Are they susceptible? Do they have antibodies that are not detectable by a sensitive neutralisation test? Are the low or absent HI antibody titres fixed for life? Should these children be revaccinated? What are the implications for the immune protection of the next generation?(92) The effect immunologically sensitised but not immune patients may have on herd immunity and ultimately the intention to eradicate measles is of concern. If more than 5% of the population are susceptibility to measles then they act as a reservoir and keep measles endemic.
Vaccination at 9 months of age has been reported to protect 88% of subjects(92) In another study, immunisation at 9 months of age protected 9 out of every 10 recipients.(93)
Yet vaccination at 9 months of age is too late to protect a large proportion of those children under that age susceptible to measles. An additional problem of vaccinating at 9 months of age is the problem of failure to seroconvert in the 10% of the population who still have maternal antibodies. By 15 months they will be unprotected against measles and they will be capable of maintaining measles endemicity, even if 100% vaccine coverage has been attained.(94)

A measles vaccination policy that vaccinates after 9 months of age in a developing population not only fails to protect a large percentage of the population who are to susceptible to measles but it also is associated with failure to return for the vaccination and this may be as high as 26%, because other vaccinations have been completed.(95)
Not all communities should necessarily be vaccinated at the same age. For derivatives of the Schwartz vaccine a revised vaccination schedule for measles in developing communities should be determined by:
the concentration of maternal measles antibodies in infants 3-9 months of age; and the age specific incidence rate of measles in the population.(94) It has been suggested, though, that during epidemics children less than 9 months in high risk situations should be immunised.(96)

If the effect that the presence of maternal antibodies has on patients to seroconvert following measles vaccination could be overcome, the ideal age at which to vaccinate against measles would be at 6 months together with the diphtheria, pertussis, polio and tetanus vaccines. This could be given at maternal and child health clinics. If it is given later in life it has been shown that in the 9-23 month old age group the attendances at mother and child health clinics is rapidly falling off.
In The Gambia 15-30% fewer children attend for measles vaccine at 9 months than at 5 months, when diphtheria, pertussis, tetanus and oral polio vaccine are given.(97)
The current policy, using the Schwartz vaccine, is to vaccinate at 9 months of age based upon the WHO recommendations. However the new strains of vaccine may change this policy.

1.9. THE COST EFFICIENCY OF MEASLES VACCINATION.

In 1983, it was estimated that the cost to vaccinate against measles in cities and large towns was U.S. $2 to 6 (approximately R10) per child.(22) The cost of measles to the community has to be considered in terms of direct and indirect costs. Direct costs based on expenditure on hospitalisation and vaccination are more easily calculated. Indirect costs and benefits are less easily quantifiable and include costs of treatment of complications in out-patients clinics and of mortality. The saving of lives and the avoidance of hospitalisation are sufficient reason to have all children immunised against measles.(96)

During January to July 1987 15 patients were admitted to the Intensive Care at Baragwanath hospital. The mortality was 40%, and the financial cost of hospitalisation was calculated to be R500.000.(16) If this is compared to the estimated cost of vaccinating children against measles in 1986 then 35000 children could be immunised for this amount of money. This is approximately equivalent to twice the under 1 year old population of Soweto. Vaccination should still be considered the most effective and inexpensive of available methods for preventing and controlling measles.

1.10. THE IMPACT OF THE VACCINE IN THE U.S.A. AND CHINA.

The United States of America and China were chosen as examples of the effect of vaccine in developed and developing populations. In the United States of America prior to the introduction of live attenuated measles virus vaccine, 500 000 cases of measles occurred annually.(98) Following mass vaccination campaigns in 1966 and 1967, the number of notified cases were 67 000, and 22 000 in 1968 and 1969 respectively.(98) In 1976 and 1977, there was an

increase in the number of reported cases, ie. 40 000 and 56 000. Unvaccinated patients accounted for 90% of these cases.(98). The Chinese did a study in three selected areas with large populations. This was analyzed over 25 years. The incidence rates of measles were reduced from >1,000/ 100,000 to 333/100,000 with partial use of vaccine and to <100/ 100,000 following the intensive use of vaccine.(3)

1.11. THE PROMOTION OF MEASLES VACCINATION.

1.11.1. Organisations and the media.

It maybe necessary to go to extreme lengths to ensure compliance with a measles vaccination schedule.(4)
Radio, television and pamphlets can help to promote awareness of the need to vaccinate against measles. National leaders, community networks eg. schools, religious institutions, womans' groups, political parties, and trade unions can be used.(99)
To have children vaccinated against measles should be seen as part of the social responsibility of a community.

1.11.2. Mass campaigns.

Mass campaigns and national vaccination days help to increase vaccination coverage.(100) Unless these are sustainable or repeated they are not considered an adequate long term measure for controlling measles.

1.11.3. Gaining acceptance for the vaccination programme.

Optimal community participation is important for improving vaccination coverage.(101)

Nations which have achieved maximum coverage have used a combination of methods preferring local flexibility to rigid bureaucratic controls to encourage parents to have their children immunised against measles.(99)
The additional cost, logistical problems of administration, low rates of return for a second dose, the altered immunity in children vaccinated too early, and the loss of community support for programmes with high vaccine failure rates, are reasons to ensure effective measles vaccination during the first visit.
In South Africa measles vaccine has been available free of charge from the Department of Health and Population Development for more than 10 years.(102) Reliable delivery of viable measles vaccine supplies and a well-managed technical and administrative network are essential for optimal measles vaccination coverage.(22) Measles vaccination should be promoted at every health contact this is not possible in the Soweto community health centres at the moment due to the fragmentation of the health service.(16) Outreach services should take vaccine to those children who do not make regular use of the health service.(103)
It is usually cheaper and easier to organise vaccination programmes in the cities simply because it is physically easier to make contact with the population.(104) Local authorities have a particular responsibility to ensure that 95% measles immunisation coverage is obtained in urban areas.

On average, only 20% of a developing population enjoy the benefits of regular health care.(105) Waiting for patients to make contact with the health services therefore cannot be relied upon for 95% measles vaccination coverage. The introduction of legal requirements for evidence of adequate vaccination before being admitted to places of care or to schools would make many more parents and guardians aware of this need.

1.11.4. Evaluating measles vaccination coverage.

An evaluation of vaccination coverage establishes the success of the programme.(106,107) Epidemics can be prevented if communities with inadequate measles protection are identified and remedial action is timeously taken.

1.13. BACKGROUND INFORMATION ON THE DEVELOPMENT OF SOWETO AND ITS HEALTH SERVICES.

No report based upon measles in Soweto is complete without a brief description of the area and the development of the health services. A unique feature of the community is that developing and developed populations live in close proximity to each other. The City of Soweto resulted from an apartheid resettlement programme of the Johannesburg City Council. With the discovery of gold in 1886, white and black migrant workers flocked to the Witwatersrand. By 1899, the mines employed 112.000 black workers. In 1904, there was an outbreak of plague and the Johannesburg local authority decided to address the problem of housing. Temporary dwellings were built near Klipspruit and the first houses were built in the South West "native" Township.(SOWETO) Ground was allocated on lease hold. This restricted people from trading in real estate and resulted in the inability to form neighbourhoods based upon socio-economic class. Even today the most elaborately built modern houses, of the upper socio-economic or developed classes, which would normally be situated in an up- market neighbourhood, are found adjacent to tumbled down wood and iron shacks. Hence the peculiar situation of having developing and developed populations living in close proximity to each other.

There is both a private and a government sponsored health service in Soweto. Health services and legislation evolved from the crisis of the flu epidemic in 1918. The provision of health care became the responsibility of the 3 levels of Government, viz. Local Authority, Province and State. The Local Authority provided promotive and preventive health services; the Province, curative services; and the State, forensic, laboratory and certain other selected health services.

The community health centres are controlled by the Transvaal provincial administration, although this has not always been the case.

The first clinic in Soweto were started by the Johannesburg local authority following the appointment of Dr. Croghan, in 1927, to Pimville the oldest township in Soweto. The Johannesburg local authority was responsible for all health services in Soweto until 1939, when the University of the Witwatersrand, with the intention of using the health services for teaching medical students, asked for a meeting with all the health authorities. It was decided at this meeting that they would all co-operate under the co-ordination of the Local Authority to provide a comprehensive Health Service. The Province and State would subsidise those services which were, according to legislation, their responsibility. However, the following year, the Provincial Administration was asked to take over the curative health services in order to allow the local authority to concentrate more on preventive health services.

In 1955, the Transvaal Provincial Administration gave notice of its intention to take over all detached outpatient curative health services. This gradually occurred over the next 10 years.
In 1980, a decision was taken to make the clinics an integral part of Baragwanath Hospital with an independent administration to manage them.

At the time of this study in 1986, there were 12 provincial community health centres which supplied curative services and 12 nearby local authority clinics providing a preventive and promotive health service to the people of Soweto. There were approximately 40 private medical practitioners in or on the borders of Soweto.

2. OBJECTIVES OF THIS STUDY.

The Objectives of the study.

This study had the following objectives:

2.1 To describe the following information on measles for the Black population resident in Soweto for the period 1984 to 1986:

a) The annual incidence of reported cases.

b) The annual attack rates according to:

ii. Age;

ii. Residential district;

iii. Provincial curative clinics and hospitals in the area;

c) The vaccine coverage among children presenting with measles.

d) The seasonal variation in the incidence.

2.2. To make recommendations on :

a) additional information that should be collected on the measles notification forms.

b) further research needs on measles.

03. METHODOLOGY.

3.1. Study design.

This is a descriptive study, based on a review of measles notification records.

3.2. Population.

The study population comprised all cases of measles diagnosed in Soweto and notified to the Johannesburg/Soweto city health department in the period 1 January 1984 and 31 December 1986. (Soweto is the geographical area known as Greater Soweto and the surrounding residential environs of Deepmeadow.)

3.3. Sample.

All notified cases of measles were studied; there was no sampling.

3.4. Measurements.

3.4.1.. Information on measles data collection in Soweto.

The following information on how the data for measles cases is routinely collected will help to clarify how the data was obtained for this study. In terms of a regulation of the Health Act 63 of 1972 section 32, measles is a notifiable condition and must be reported to the Medical Officer of Health (M.O.H.) in writing on a specific form number GW 17/5. The M.O.H. then informs a community health nurse (CHN) working at the clinic closest to the residential address of the patient of the measles case. This is done with form number M1531.(Appendix A.) The CHN visits the patient, parents or guardian at the residential address with the

aim of completing form M2660 (Appendix B) which is sent back to the M.O.H. for information before being returned to clinic for filing. The information obtained on age and date of vaccination is confirmed by asking the patient or guardian of the child to produce the road to health card or any other documented proof of vaccination and date of birth.
When records are unavailable, the CHN's best estimate of the child's age is used. A child is considered vaccinated against measles only if documented evidence of the date of vaccination can be produced. A return visit to the notified address is made for children that cannot be contacted during the first visit.
In cases where personal contact with the patient is unsuccessful, neighbours or residents at the address are asked about the whereabouts of the notified measles cases. Patients who are known to be visitors, or who are unknown in the area and cannot be contacted are classified as non permanent residents of Soweto.

The names of all notified measles cases between 1 January 1984 and 31 December 1986 were taken from the form used by the community health nurse to gather data on all notified measles cases.(M2660 form) The names of the patients and the following data obtained on each patient was transferred to a data sheet. (appendix C) Date of birth, date of notification of measles, dates of vaccination against measles, residential address and name of hospital, clinic or health worker that notified the measles case.

3.4.2. Definition of rates.

To calculate the annual incidence rate of measles the total number of cases notified for each year was taken as the numerator, and the official Johannesburg/Soweto city council census of Black people resident in Soweto as the denominator. The city council calculates an annual census.

3.4.3. The age specific attack rate.

Calculated from the number of children within the age group who were notified as having measles upon the population of that age group.
Birth dates, when available, were subtracted from date of measles notification in order to calculate the age at which measles occurred. It was assumed that the date of notification of disease was equal to the date of the disease, since measles is usually a disease of short duration and it is customary for the notifying authority to notify the disease during the first visit. In cases where the birth date was unknown the estimated age of the child as recorded by the CHN or the hospital or clinic notifying the patient was used.

3.4.4. The measles attack rate by residential district.

The numerator comprised all reported measles cases < 15 years of age who had been contacted, either at home or in hospital. The < 15 year old population comprised 99 % of all the measles cases. The denominator used for each district was the official city council census for children < 15 years of age in each respective district in the years 1984 to 1986.

3.4.5. The measles notification rate for clinics.

The rate of notifications per clinic was calculated from the number of notified measles cases < 11 years of age expressed as a proportion of the total number of under 11 year old attendances for each of the clinics. The reason for using these statistics was that it was policy for the clinics to divide annual statistics for attendances into paediatric and adult attendances. Persons under 11 are regarded as paediatric, while those 11 or more are classified as adults.

The under 11 year old population accounted
for 96% of all measles cases attended to by the clinics.

3.5. DATA ANALYSIS.

All the information on the data sheet was captured on a personal computer by two persons (ie. double punched) to minimise the possibility of punching errors. The data was analyzed utilizing a SAPEC 286 personal computer and the software packages Epi-info and Lotus 123.

3.6. IMPLEMENTATION.

The research was carried out in the Soweto clinics where the necessary records were made accessible and available through the good offices of the M.O.H. A professional nurse assisted with the transfer of information to the data sheets.

3.7. LIMITATIONS.

3.7.1. The criteria for the diagnosis of measles.

All notified cases were included in this study and taken as cases. Since it was a retrospective study it was not possible to verify the diagnosis.

3.7.2. The under notification of measles cases.

The assumption made that notified and incidence are similar is probably incorrect. Measles is known to be an under notified disease.(18) No measles cases were notified by any of the private medical practitioners working in the area.
However, as there was no reason to suspect any change in notifying behaviour of health workers, it is assumed that the level of undernotification was constant during the period.

3.7.3. The under estimation of the population of Soweto.

There are two distinct sources of information required on populations before the criteria for high confidence levels and narrow confidence limits on epidemiological data can be achieved. These are:

i. an accurate enumeration of the number, age and sex of a population and

ii. the occurrence of births and deaths.

For studies of this nature such requirements raise particular problems in Soweto. Prior to 1986, de facto population census would have been difficult to do, due to the lack of resources. Prior to and after 1985 de jure population census would have been impossible as inadequate records existed. Home addresses given by patients admitted to hospital for confinements may have been for convenience to avoid being refused admission on the grounds that they did not live in Soweto. This would have resulted in an overestimation in the under 1 year of age population.

During 1986, influx control regulations which controlled the inflow of people into Soweto were relaxed. It is not possible to exclude the effect this may have had on the incidence of measles in the area or to ascertain the accuracy of the population numbers used as the denominator in many of the calculations during this period.

3.7.4. Residential address.

In this study "Soweto resident" means that the patient was traced at the address given by the patient during the first consultation at the hospital or clinic. It is not equivalent to permanent residence in Soweto. Patients who were not found on a second visit or who were unknown to neighbours, were considered to be non residents of Soweto.

3.7.5. Recording errors.

The notification forms (M2660) which were used as the data source had to be collected from the clinics. It was not possible to verify the information on the notification forms (M2660).

04. RESULTS AND DISCUSSION.

The response rate for this study was 100% as all the forms (M2660) that were collected from the clinics were used. All the forms had been correctly completed by the community health nurse (CHN). In those cases where the CHN was unable to trace the patient, the information given to the medical officer of health (MOH) during the initial notification could not be verified. This occurred in less than 15% of cases.
The results are presented according to the objectives and reference is made to paragraphs in the introduction which address the issues.

4.1. THE INCIDENCE OF MEASLES.

Table 1 shows the incidence rate of measles per 100,000 <15 year old population from 1984 to 1986. Ninety-nine percent of all measles cases notified occurred in the < 15 year old population during this period. For this reason it was decided to use the adjusted < 15 year old population to calculate the incidence. No significant difference in the incidence of measles in the Soweto area, was found for 1986 compared with 1984 and 1985. (t test p=<0.001)

TABLE 1. THE MEASLES INCIDENCE RATE PER 100.000 POPULATION < 15 YEARS OF AGE FOR THE PERIOD 1984-1986.

YEAR.	1984	1985	1986
INCIDENCE RATE	25	30	27
CASES*	N=415	N=507	N=464

* Number of cases

The true incidence of measles in Soweto is likely to be greater than the results suggested by this study because cases were only notified from clinics and hospitals. The reasons why measles cases had not been notified by general practitioners in the area needs to be investigated. Under notification for measles is not unique to this study and has been discussed in the introduction. There is a need to improve notifications so that an accurate assessment of the incidence of measles in the area can be made. This is essential for planning the adequate provision of measles vaccine to the community.

4.2 THE ATTACK RATE ACCORDING TO AGE.

In 1986, the peak in incidence of measles for under 1 year olds was significantly greater than for either 1985 or 1984. (Fig 1) (t test $p<0.001$) In fact the incidence of measles in the under 1 year old population doubled during 1986 compared to 1984 and 1985.

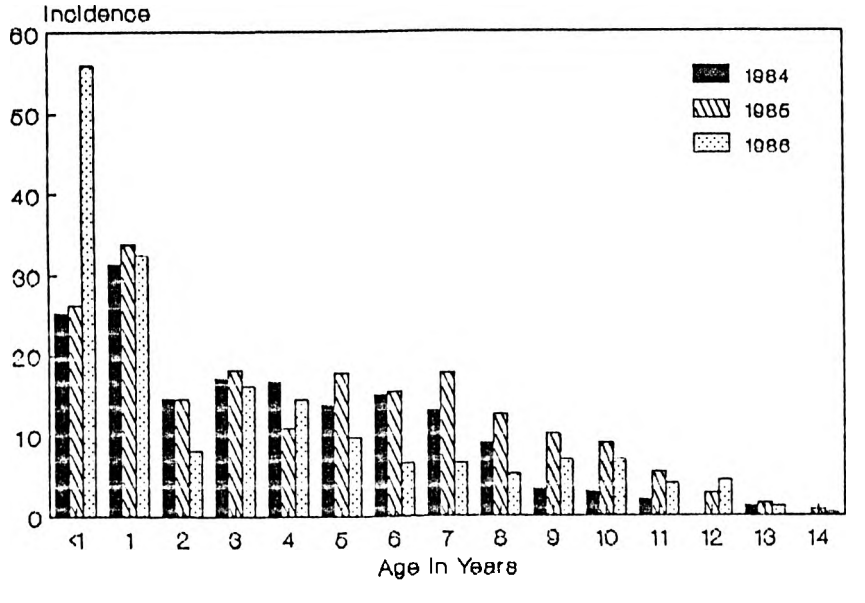

FIG.1. THE AGE SPECIFIC INCIDENCE RATE OF MEASLES IN SOWETO PER 100.000 IN THE PERIOD 1984 TO 1986.

The percentage of the total number of measles cases which were under 1 year of age was 15%, 13% and 30% respectively for these 3 years.
Gibson found that 20% of South African national notifications, from August 1979 to July 1986, were for infants less than one year of age.(107) These findings are slightly greater than the findings of this study for 1984 and 1985, but 10% less than that found for 1986, the change over period to one vaccination at 9 months of age.

This could suggest that a proportion of children between 6 and 9 months of age who were vaccinated in 1984 and 1985 were adequately protected against measles.

The change in the age specific attack rate, ie more measles in younger children, could be attributed to a greater proportion of 6 to 9 month old children who had not been vaccinated against measles because of the change in policy of age of vaccination from 6 to 9 months. They would not be protected against measles and would be more susceptible to the wild measles virus infection.

If there had been an epidemic of measles in 1986 then it could be expected that the incidence of measles in all age groups would have increased and the increase in incidence would not have been restricted to children less than one year of age. (Fig 1)
There is no reason to suspect that the ratio of children in the age groups, under one year, and 1 to under 15 years, would have changed in 1986 compared to 1984 and 1985.
The increase in the incidence of measles in the under one year olds is therefore not attributable to an increase in population in that age group but rather to an increase in the attack rate.

The percentage of children under 9 months of age who contracted measles doubled (from 7 to 14%) in 1986 compared with the two previous years. (Table 11)

TABLE 11. MEASLES CASES ACCORDING TO AGE AS A PERCENTAGE OF ALL MEASLES CASES.

Age in months	Percentage of all cases		
	1984	1985	1986
<9	7	7	14
9-15	15	13	22

The corresponding percentage increase in measles in children 9 to 15 months of age increased from 15% in 1984 and 13% in 1985 to 22% in 1986. Notwithstanding the doubling of the percentage of measles cases in the under 9 month old group for 1986 compared with 1984 and 1985 (7% to 14%), it is still less than that reported for other parts of Southern and West Africa. Loening reported that 20-45% of cases from an urban area in South Africa were less than 9 months of age while in The Gambia in 1983 measles in the under 9 month old group accounted for 18% of cases (18,71)
The increased rate in children of the age group 9 to 15 months in 1986, could be ascribed to fewer children returning for vaccination at 9 months. After 6 months of age children are less likely to attend vaccination clinics.

4.2.1. Evaluating the changes in measles immunisation policy.

Changes in vaccination schedule should not go unmonitored.
In 1974 Cameroon increased the age for vaccination against measles from 6 to 9 months.(95) A study done in Yaounde, the capital of Cameroon, in 1979 showed a 40% vaccination coverage in 12 to 23 months old children. There was a 44% decrease in reported cases of

The greatest change occurred in the reported decrease in the incidence of measles in children under one year of age in the year following the change over to the new minimum age of vaccination at 9 months of age while the smallest change was in the attack rate 24 to 47 months of age.(95) There was also a decreasing trend in the number of children who died from measles in the Yaounde Central Hospital.(109) It is necessary to monitor the effects a change in the vaccination schedule has on the incidence and the age specific attack rate for future planning. Monitoring the age specific attack rate over three years in Soweto, during which time there was a change in vaccination policy is inadequate for evaluating the age at which measles vaccine should be given.

4.3 THE ATTACK RATE ACCORDING TO RESIDENTIAL DISTRICT.

Table IV indicates the incidence of measles by residential district and status. Residential status, in this case, refers to whether the notified measles case could be traced to the home address given during the initial clinic/ hospital contact and would therefore give an indication of whether the case lived in, or was a visitor to Soweto. The issue of residential status has been addressed in the section dealing with limitations.
In some residential districts visitors to Soweto accounted for 100 percent of measles cases.

The incidence of measles from the districts of Soweto is not directly related to the incidence of measles reported in Table 1., which is calculated from the total number of measles cases that were attended to in Soweto. The latter calculation based upon all cases that were notified from the health services which included patients with residential addresses outside of Soweto.

TABLE IV. INCIDENCE RATE OF MEASLES PER 100.000 UNDER 15 YEAR OLDS BY GEOGRAPHICAL DISTRICT WITH THE PERCENTAGE OF CASES PERMANENTLY RESIDENT IN THE DISTRICT.

DISTRICTS	1984.		1985.		1986.	
	Incid.	Res.*	Incid.	Res.*	Incid.	Res.*
ZOLA	22	50	133	75	144	92
KLIPSPRUIT	171	100	196	100	288	83
SENOANE	60	75	163	82	147	91
MOLETSANE	64	50	16	0	79	60
ZONDI	66	39	130	40	220	82
ORLANDO WEST	92	50	274	72	257	53
MOFOLO	61	75	106	72	196	85
PROTEA	107	60	214	70	156	68
PHIRI	119	0	236	75	528	11
MOLAPO	120	58	273	60	124	69
NALEDI	130	61	51	82	138	83
TLADI	132	47	232	55	292	63
ORLANDO EAST	135	71	217	85	178	64
JABULANI	149	60	89	66	118	59
JABAVU	151	0	228	57	232	49
DHLAMINI	183	83	121	75	120	63
EMDENI	187	70	309	72	138	63
CHIAWILO	260	44	82	61	163	69
MOROKO	454	80	180	60	179	70
PIMVILLE	693	75	134	86	247	62

Inc = Incidence per 100.000 under 15 year old population.
Res* = The percentage of under 15 year old population that were in the district.

In order to be able to compare the districts a rate per 100000 population has been used in Table IV. The results of calculations involving small numbers of actual measles cases from each district are therefore magnified.

All the measles cases from Phiri and Jabavu in 1984 and Moletsane in 1985 could not be traced.
Sick children from country areas are brought into Soweto for diagnosis and treatment. During 1984 and 1985, the period of influx control regulations, it is unlikely that any patient presenting at either a clinic or hospital without a Soweto address would have been attended to, unless they were acutely ill or had been referred from another hospital. It could be expected therefore that false addresses would be used by patients to get medical attention.

Certain districts did have an increased incidence rate over others for particular years. The range of variation in the incidence of measles by district is from 22 to 693 per 100 000 total population. This may be a reflection of discrepancies in notification of cases from the districts. However the socio-economic status of certain districts, together with overcrowding, a large susceptible population and inadequate health services could be responsible for the range in incidence. It was beyond the scope of this study to further investigate these findings.
In 1984 Zola had the lowest and Pimville the highest incidence of measles. (Table lV.) Following visits to the area it appeared, scientifically unconfirmed, that the socio economic status of Pimville was better than Zola. There are both preventive and curative health services in these areas. Assuming that all the measles cases that presented to the clinics were notified, the number of notification of measles cases from Zola clinic appears to confirm the low incidence of measles in this district during 1984.(Fig.2.)
There was an epidemic of measles in Moroko and Pimville during 1984. No apparent changes in the socio-economic, cultural or the health service delivery could obviously be linked to this increased incidence above other districts. More than 75% of the reported measles cases in both these areas were confirmed as permanently living there.

Episodic epidemics have been described as occurring every 2 to 5 years in a developing population and may account for this finding. However, a visitor to either or both of these districts may have been responsible for the introduction of wild measles virus into the areas. During 1985, the incidence of measles in most other districts of Soweto had increased while the incidence of reported measles cases from Pimville and Moroko decreased.

Measles in Soweto cannot be seen in isolation to measles in the rest of the country. Approximately 20 % of measles cases were not resident in Soweto. It is important that all populations at risk be adequately immunised against measles.

4.4 THE ATTACK RATE ACCORDING TO HOSPITALS AND CLINICS.

The percentage of measles cases notified from hospitals increased during 1986 compared with 1985 and 1984.(Table V)

TABLE V . The percentage of measles cases that were notified from hospitals and clinics for the period 1984 to 1986.

	Percentage of cases notified 1984.	1985.	1986.
Hospital/clinics			
Baragwanath Hosp.	30	17	32
C.M.R. Hosp.*	16	16	28
Total from Hosp.	46	33	60
Total from Clinics	55	66	41

* C.M.R. MINES REGIONAL HOSPITAL

The increase in the number of notified cases from hospitals during 1986 could mean that the severity of complications among measles cases increased in that year compared with the previous two years and the complications required admission to hospital. It is known that measles in under one year olds is associated with more severe complications which require hospital admission as described in the introduction to this study.

The shift to an increased incidence among younger children in 1986 has already been discussed in the paragraph dealing with the age specific attack rate. This accounts for the increased incidence of measles notified from the hospitals in 1986 and the perceived epidemic reported by doctors working in the referral hospitals in Soweto.
Patients with measles in the clinics account for up to 2.8 per 1000 under 11 year old consultations.(Fig 2)

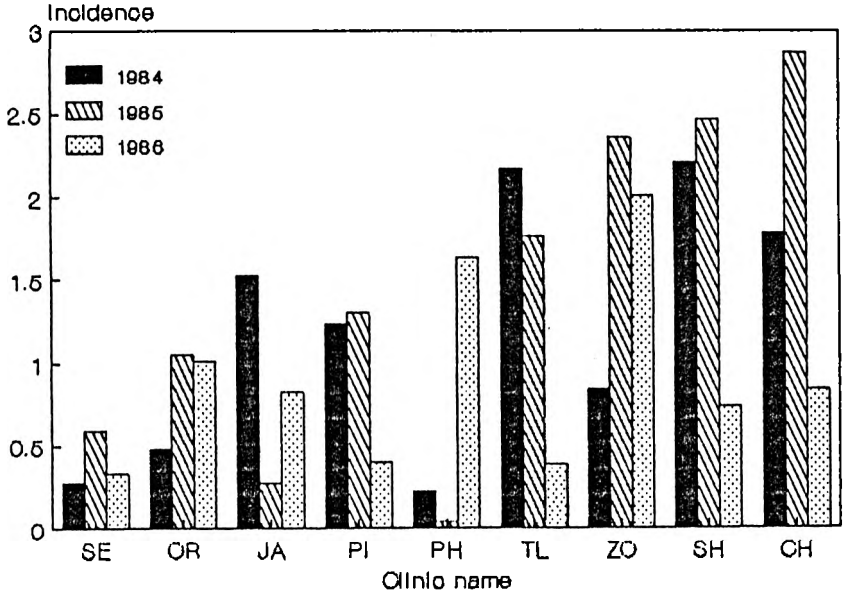

FIG.2. THE INCIDENCE RATE OF MEASLES BY CLINIC, PER 1000 UNDER 11 YEAR OLD CONSULTATIONS IN THE PERIOD 1984 TO 1986.
(SE=SENOANE, OR=ORLANDO, JA=JABAVU PI=PIMVILLE, PH=PHOMOLONG, TL=TLADI, ZO=ZOLA,SH=SHANTY,CH=CHIAWILO.)

Attendance records at the clinics are kept for under 11 year olds. Ninety four percent of measles cases occurred in the under 11 year old population. It was convenient to use the available statistics on clinic attendances for under 11 year olds to calculate the incidence of measles per 1000 consultations. Besides Phomolong and Jabavu all the other clinics reported fewer cases of measles in 1986 compared to 1985. This could mean that in 1986, measles cases were acutely ill and parents or guardians of the children decided to take their children directly to hospitals instead of going to the clinics.

4.5 THE VACCINATION STATUS OF MEASLES CASES.

More than 50% of notified measles cases had received at least one vaccination for the period 1984 to 1986.(Table Vl) Only those cases that could provide documented proof of measles vaccination were considered to be vaccinated.

TABLE Vl. The vaccination status of measles cases expressed as a percentage of the annual number of cases.

	Years.		
Vaccination status.	1984.	1985.	1986.
1 X Vaccination (only)	25	48	48
2 X Vaccinations	30	24	13
Total vaccinated	55	72	61
Total unvaccinated	45	28	39

The patients who presented with measles were divided into two groups, vaccinated and unvaccinated, for each of the three years 1984 to 1986. From 1984 to 1986, a comparison of vaccinated with unvaccinated measles patients reveals that a greater percentage of measles patients had been vaccinated for each of the three years 1984 to 1986. (Table Vl)

The present method of notification and follow up of the patient does not record the vaccinating authority or the batch number of the vaccine. In this study it was not possible to determine how many of these patients had been vaccinated by an health authority other than Soweto.

Table Vll shows the age at which vaccinated children who presented with measles received the vaccine. In 1984 and 1985, the ratio of children with measles who had been vaccinated when they were less than 9 months of age, compared with those who had been vaccinated at or after 9 months of age, was greater.(55:45) This ratio was reversed for 1986.(39:61) Vaccine failure therefore cannot be ascribed entirely to the early age at which children were vaccinated in 1984 and 1985. Assuming that the documentation on the road to health chart was correct, non viable vaccine contributed to children being inadequately protected against measles.

TABLE V11.The age of vaccination of measles cases as a percentage of all vaccinated cases.

Age in months.	The percentage of measles cases vaccinated		
	1984.	1985.	1986.
Total under 9 months	55	55	39
Total = or > 9 months	45	45	61

More than half of the notified measles cases, in this study, had received at least one measles vaccination. Reasons for vaccine failure have been discussed in the introduction (1.8.3.)

The apparent non viability of the vaccine in many of the children presenting with measles is a problem of concern. Calculating vaccine efficacy would help to determine the extent of this problem. However, it does not distinguish between vaccine failure attributable to early vaccination when maternal antibodies are present and inadequate maintenance of the cold chain.

Although a large percentage of measles cases had been vaccinated there is still an unacceptably high percentage (39%) in 1986 that had not.(Table V1) Immunity levels of greater than 90% must be achieved before transmission of measles will be interrupted.

The ideal of 100% vaccine coverage should be strived for.(54) The reasons for the apparent vaccine failure in Soweto needs to be investigated .

4.6 THE SEASONAL VARIATION OF MEASLES.

The seasonal variation was consistent over the three year period. The incidence peaks in September.(Fig.3.)

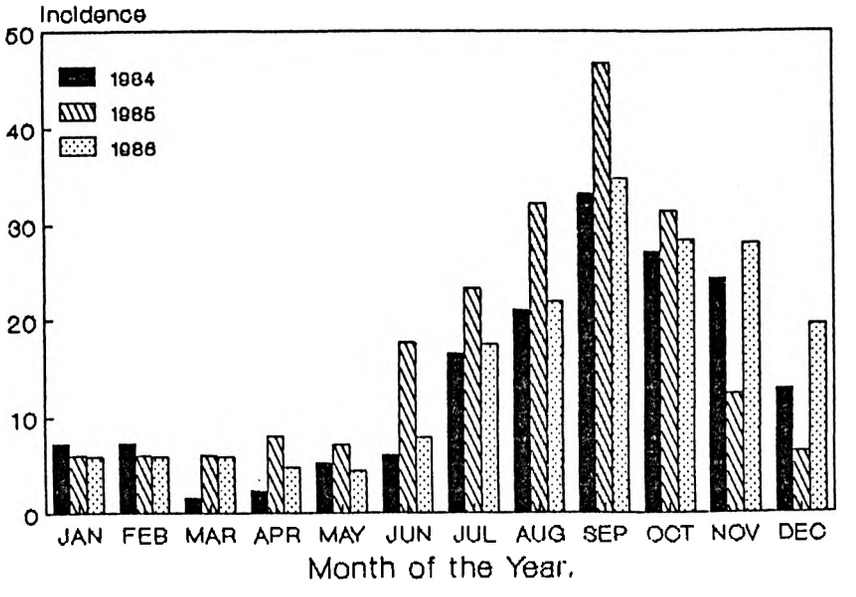

FIG.3. THE INCIDENCE RATE OF MEASLES CASES BY MONTH PER 100.000 IN THE PERIOD 1984 TO 1986.

The number of measles cases starts to increase in June and July, during the winter months, each year. At this time children are likely to spend more time indoors and are therefore in closer contact with one another.

The seasonal incidence is not uniform throughout Africa.

In Equatorial countries Uganda, Kenya and Tanzania the peak month for measles is April.(4) While in Zambia, Zimbabwe and Malawi peak months are November to January.(110)

West Africa reports measles throughout the year but most epidemics develop in the hot dry season, December to February.(4) At this time farm activities are reduced and festivals are held offering the opportunity for the spread of infection by droplets infections.(4) Some workers have related the increased incidence of measles to temperature and humidity.(111) Maximum survival of the virus does occur in the humidity found during the wet season.(111) However climatic conditions are unlikely to be an important factor in the spread of measles.

05. RECOMMENDATIONS.

5.1. The Prevention of measles.

5.1.1. In Soweto, infants from 6 months of age need to be protected against measles as shown by the increase in the incidence of measles in the under 9 month old population at the time of the change in vaccination programme from two vaccinations at 6 and 12 months to one vaccination at 9 months.

5.1.2. Community health nurses should be involved in the planning of measles vaccination programmes. This would encourage them to maintain a high level of vaccine coverage and to make additional efforts to trace measles cases in their districts, to identify sources of infection and to commence vaccination programmes during the early phase of an outbreak.

5.1.3. Measles vaccination needs to be promoted. The unacceptably high proportion of measles cases that had not been vaccinated shows that awareness programmes to promote measles vaccination should be ongoing. In addition legislation could be changed to require that parents produce evidence of measles vaccination of their children prior to their admission to schools and creches.

5.1.4. Although on-going measles vaccination programmes in the clinics should be the policy for measles prevention, mass vaccination campaigns and a national vaccination day would help to promote compliance with measles vaccination. These should be arranged in May each year before the incidence of measles starts to increase. Mass vaccination campaigns and a national measles vaccination drive should not take priority over an ongoing process to promote measles vaccination.

5.2. The notification of measles cases.

5.2.1. The notification of measles cases in Soweto needs to be improved so that adequate planning can be done for measles vaccine delivery. This could be achieved by:

i. simplifying the notification procedure by accepting telephonic notifications.
ii. regularly contacting private family practitioners in the area and asking for measles notification forms.
iii. encouraging teachers, child minders and traditional healers to report cases to the clinics.

5.2.2. Parents should be encouraged to give accurate information about their residential address. They should be informed during their initial visit to either the clinic or hospital that a community health nurse will be visiting them at home during the first and third week of the disease to clinically reexamine the child, to advise on any problems that may arise and to complete a questionnaire on the progression of the disease.

The additional information required on notification forms.

5.2.3. Follow-up forms should include the following information if the reasons for vaccine failure and the cost of measles infection are to be investigated.

i. The place and person responsible for the measles vaccination.
ii. The batch number of the vaccine.
iii. The suspected source of the infection if this is known. This is particularly important if it is suspected that the infection was acquired in hospital.

iv. The date of onset of complications and their nature.
v. The nature and quantity of medication that has been prescribed for the patient.
vi. The size of, and age of all children, in the family.

5.3. Research needs.

The following research would improve our understanding of the cost of measles and its prevention in Soweto.

1. The efficacy of the vaccine presently used in Soweto.

2. The establishment of the efficacy and cost efficiency of the aerosol of undiluted human diploid cell Zagreb strain vaccine given by inhalation to infants less than 9 months of age
or the Edmonston-Zagreb vaccine in a dose of 5000 plaque forming units given subcutaneously to 5 month old children.

3. An evaluation of the vaccination coverage in Soweto.

4. The direct costs of measles infection to the health service in Soweto is unknown. From the increase of measles admissions to hospitals in 1986 it is essential that these cost be determined. This would aid health planners to decide on the priority of improved provision of measles vaccine to the community.

06. CONCLUSIONS.

The notified incidence of measles cases in Soweto did not increase during the change over period from use of two immunisations at 6 and 12 months to one immunisation at 9 months. However, the lowering of the age specific attack rate of measles probably resulted in an increase in hospital admissions. This was attributable to the associated increase in the occurrence of severe clinical complications that require admission in children with measles who are less than 1 year of age.

Measles in children in the Soweto community presents from 6 months of age. These children need to be protected against infection from the measles virus.

The Schwartz vaccine administered intradermally in children of this age group may leave them susceptible to measles infection later in life. They would then act as a reservoir for the wild measles virus which would thwart efforts to eradicate measles.

It is necessary to consider vaccines other than the Schwartz strain given intradermally if children between the age of 5 and 9 months are to protected against the wild measles virus.

Population migration resulted in a number of cases being notified that were temporary residents of Soweto. This emphasises the importance of developing a national strategy in order to prevent wild measles virus being reintroduced into Soweto once it had been eradicated.

Measles awareness campaigns are necessary, to sensitise the population about the importance of having children immunised against measles and to remind health care providers of the need to notify all measles cases.

07. ACKNOWLEDGEMENTS.

The following persons gave approval to conduct the study.

Prof H.S. Hurwitz. Medical Officer of Health Johannesburg.

Dr. N. Padayachee. Senior Deputy Medical Officer of Health Johannesburg.

The Late Dr. W.M. Matsie Medical Officer of Health Soweto.

Prof J.S.S. Gear. Head of the Department Community Health.

The following persons have given helpful criticism, advice and help.

Dr. M. Edginton. Acting Head of the Department of Community Health, University of the Witwatersrand.

Dr. N. Padayachee. As above

Dr. H.G.V. Kustner. Director Epidemiology,Department of Health and Population Development.

Dr. H. Hull. Centre for Disease Control, Atlanta, Georgia, U.S.A.

Dr. M. Ross. Lecturer Department of Community Health, University of the Witwatersrand.

Prof. BLW. Sparks.	Head of the Department of Family Medicine University of the Witwatersrand.
Mrs. S. Pryor.	Professional nurse, Johannesburg City Health Department.
Ms A. van Middlekoop.	Deputy Director Epidemiology Department Health and Population Development.
Mr. S. Steyn.	Computer Consultant,National Centre for Occupational Health.

08. REFERENCES.

1. Assad F. Measles: Summary of Worldwide Impact. Rev Infect Dis 1983; 5:3 452-459.

2. Christensen PE, Schmidt H, Jensen O, Bang HO, Andersen V, Jordal B. An epidemic of measles in Southern Greenland,1951. Measles in Virgin soil. 11. The epidemic proper. Acta Med Scand 1952; 144: 430-449. Quoted in "Morley D. Severe Measles in the Tropics.-I Br Med J 1969; 297-300."

3. Langmuir A, Orenstein W. Current impact of measles. Rev Infect Dis 1982; 5: (3) 474-476.

4. Morley D. Severe Measles in the Tropics.-I Br Med J 1969; 297-300.

5. Barkin RM. Measles mortality: a retrospective look at the vaccine era. Am J Pediat 1965; 66: 779-86.

6. Ghosh S, Dhatt PS. Indian J Child Health. 1961; 10: 111. Quoted in Morley D. Severe Measles in the Tropics.-I Br Med J 1969; 297-300.

7. Morley DC. Proc R Soc Med 1964a; 57: 846.

8. Gordon JE, Jansen AAJ, Ascoli W. Measles in rural Guatemala. J Pediatr 1965; 66: 779-786.

9. Langmuir AD, Henderson DA, Serfling RE, Sherman IL. The importance of measles as a health problem. Am. J. Public Health 52(2[Suppl.]): 1-4 1962.

10. Aaby P, Bukh J, Lisse IM, Smits AJ. Measles vaccination and child mortality. Lancet 1981; ii: 93.
11. Kasongo Project Team. Influence of measles vaccination on survival pattern of 7-35 month old children in Kasongo, Zaire. Lancet 1981; i: 764-67.

12. Burrows EH. A History of Medicine in South Africa, 1st ed. Cape Town AA Balkema. 1958; 143.

13. Brincker JAH, A Historical, and Aetiological Study of Measles. Proc R Soc Med 1938; 31: 807-840.

14. Gear JHS, Measles in South Africa. Amer J Dis Child 1964; 103: 261-264.

15. Wittenberg DF. Measles Experience in Durban. S Afr Med J 1987; 71: 191.

16. Harrisberg J, Radcliffe M, Kala U, Hartman E. Bolton K. Measles outbreak at Baragwanath Hospital - a plea for action. S Afr Med J 1988; 73: 62.

17. Kettles AN. Difference in trends of measles notification by age and race in the western Cape, 1982-1986. S Afr Med J 1987; 72: 317-320.

18. Loening WEK, Coovadia HM: Age specific occurrence rates of measles in urban peri-urban and rural environments: implications for time of vaccine. Lancet 1983; ii: 324-326.

19. Wilson GS. Measles as a universal disease. Am J Dis Child 1962; 103: 219-223.

20. Stansfield JP, Warley MA, Kintu S. J Trop Pediatr 1966; 12 monograph 9. Quoted in "Morley D. Severe Measles in the Tropics.-I Br Med J 1969; 297-300."

21. Krugman S, Giles JP, Friedman H, Stone S. J Pediatr 66: 471. Quoted in "Morley D. Severe Measles in the Tropics.-I Br Med J 1969; 297-300."

22. Walsh JA. Selective primary health care: strategies for control of disease in the developing world. lV. measles. Rev Infect Dis 1983; 5: 330-340.

23 Puffer RR, Serrano CV. Patterns of mortality in childhood. 1973: Scientific publication no. 262. Washington D.C. Pan American Health Organisation, Pan American Sanitary Bureau, Regional Office of the World Health Organisation, 1973:

24. Axton JHM. Measles: a protein losing enteropathy. Br Med J 1975; 3: 79-80.

25. Dosseter J, Whittle HC, Greenwood BM. Persistent measles infection in malnourished children. Br Med J 1977; 1: 1633-1635.

26. Katz SL, Morley DC, Krugman S. Attenuated Measles Vaccine in Nigerian Children. Am J Dis Child 1961; 103: 402.

27. Scheifele DW, Forbes CE. Prolonged Giant cell excretion in severe African measles. Pediatrics 1972; 50: 867-873.

28. Chen LC, Chowdhury AKMA, Huffman SL. Anthropometric assessment of energy protein malnutrition and subsequent risk of mortality among preschool aged children. Am J Clin Nutr 1980; 33:1836-1845.

29. Scrimshaw NS, Salomon JB, Bruch HA, Gordon JE. Studies of diarrhoeal disease in central America. 1966 VIII. Measles diarrhoea, and nutritional deficiency in rural Guatemala. AM J Trop Med Hyg 1966; 15: 625-31.

30. Morley DC. Measles in the developing world. Proc R Soc Med 1974; 67: 1112-1115.

31. Koster FT, Curlin GC, Aziz KMA, Haque A. A Synergistic impact of measles and diarrhoea on nutrition and mortality in Bangladesh. Bull WHO 1981; 59: 901-908.

32. Foster SO. Measles in Indonesia. Field consultation to Directorate of Epidemiology and Quarantine Directorate General of Communicable Diseases, Ministry of Health, Republic of Indonesia. Centres for Disease Control, Atlanta. 1978.

33. Rhazes A. Treatise on the Smallpox and Measles (A.D. 850), translated from the original Arabic by W.A. Greenhill. Sydenham Society London.1848: Quoted in Morley D. Severe Measles in the Tropics.-I Br Med J 1969; 297-300.

34. Dossetor J, Whittle HC, Greenwood BM. Persistent measles infection in malnourished children. Br Med J 1977; 1: 1633-1635.

35. Crespi M, Struthers JK, Smith AN, Lyons SF. Interferon status after measles virus infection. S Afr Med J 1988; 73: 711-712.

36. Williams SJ. Measles Immunisation: Remaining Needs for Research. Rev Infect Dis 1983; 5: 613-617.

37. Coovadia HM. Measles in South Africa- effects of nutrition and heredity 1980; Presented at the second International Conference on Impact of viral diseases on Development of Africa and Middle East Countries. Nairobi. Quoted in "Williams SJ. Measles Immunisation: Remaining Needs for Research. Rev Infect Dis 1983; 5: 613-617."

38. Miller DL. Frequency of complications of measles, 1963. Br Med J 1964; 2: 75-78.

39. Dover AS, Escobar JA, Duenas AL, Leal EC. Virus pneumonia associated with measles. A virological and histological study of autopsy material. JAMA 1975; 234: 612-614.

40. Kipps A, Kaschula ROC. Virus pneumonia following measles. S Afr Med J 1976; 50: 1083-1088.

41. Nestadt A, Harrison I. Treatment of Progressive Primary Tuberculosis with Isoniazid Alone. Lancet 1964; 1: 1068.

42. Sauter JJM. Xeropthalmia and measles in Kenya. Kliniek voor Oogheelkunde, Rijks-Universiteit Groningen The Netherlands 1976; 51-196. Quoted in "Walsh JA. Selective primary health care: strategies for control of disease in the developing world. lV. measles. Rev Infect Dis 1983; 5: 330-340."

43. Indonesia: nutritional blindness prevention project. 1980; Helen Keller International New York. Quoted in Walsh JA. Selective primary health care: strategies for control of disease in the developing world. lV. measles. Rev Infect Dis 1983; 5: 330-340.

44. Modlin JF, Jabbour JT, Witte JJ, Halsey NA. Epidemiologic studies of measles vaccine, and subacute sclerosing panencephalitis. Pediatrics 1977; 59: 505-512.

45. Morgan EM, Rapp F. Measles virus and its associated disease Bacteriol Rev 1977; 41: 636-666.

46. Hall WW, Choppin PW. Measles virus proteins in the brain tissue of patients with sub acute sclerosing pan encephalitis. Absence of the M protein. N Engl J Med 1981; 304: 1152-1155.

47. Brown P, Cathala F, Gajdusek DC, Gibbs CJ jr. Measles antibodies in the cerebrospinal fluid of patients with multiple sclerosis. Proc Soc Exp Biol Med 1971; 137: 956-961.

48. Haire M, Millar JHD, Merrett JD, Measles virus-specific IgG in cerebrospinal fluid in multiple sclerosis. Br Med J 1974; 4:192-193.

49. Norrby E, Link H, Olsson JE, Panelius M, Salmi A, Vandik B. Comparison of antibodies against different viruses in cerebro spinal fluid and serum samples from patients with multiple sclerosis. Infect Immun 1974; 10: 688-694

50. Nathanson N, Miller A. Epidemiology of multiple sclerosis: critique of the evidence for a viral etiology. Am J Epidemiol 1978; 107: 451-561.

51. Arthur L. W Afr Med J 1961; 10: 262. Quoted in Morley D. Severe Measles in the Tropics.-I Br Med J 1969; 297-300.

52. Dobyns HF. An outline of Andean Epidemic History to 1720. Bull Hist Med 1963; 37: 493.

53. Government of the United Republic of Cameroon. National nutrition survey, final report USAID 1978; 136-42. Quoted in "Heymann D, Mayben G, Murphy K, Guyer B, Foster S. Measles Control In Yaounde: Justification of a one dose, nine month minimum age vaccination policy in Tropical Africa. Lancet 1983; 2: 1470-1472."

54. Hull HF, Williams PJ, Oldfield F. Measles mortality and Vaccine efficacy in rural West Africa. Lancet 1983; 1: 972-975.

55. Aaby P, Bukh J, Lisse ID, Smits AJ. Measles mortality, state of nutrition, and family structure. A community study from Guinea-Bissau. J Infect Dis 1983; 147: 693-701.

56. Koster FT, Curlin GC, Aziz KMA, Haque A. Synergistic impact of measles and diarrhoea on nutrition and mortality in Bangladesh. Bull WHO 1981; 59: 901-908.

57. Smith EA, Foster SO. Measles in areas of malnutrition. Journal of the Nigeria Medical Association 1970; 7: 16-18.

58. Glyn-Jones R. Measles vaccine and gammaglobulin in the prevention of cross infection with measles in an acute paediatric ward. Centr Afr J Med 1972; 18: 4-9. Quoted in "Reynolds LGvB, Klein M. The hospital as a vector of measles in the community. S Afr Med J 1987; 71: 637-638."

59. Wesley A, Coovadia HM, Watson AR. Immunisation against measles in children at risk for severe disease. Trans R Soc Trop Med Hyg 1979; 73: 710-715.

60. Beckford AP, Kaschula ROC, Stephen C. Factors associated with fatal cases of measles. S Afr Med J 1985; 68: 858-863.

61. Reynolds LGvB, Klein M. The hospital as a vector of measles in the community. S Afr Med J 1987; 71: 637-638.

62. Aaby P, Bukh J, Hoff G etal. High measles mortality in infancy related to intensity of exposure. J Pediatr 1986; 109: 40-44.

63. Morely D. Measles and whooping cough. In Cruickshank R, Standard KL, Russell HBL. ed. Epidemiology and community health in warm climate countries. New York Churchill Livingstone 1976: 63-67.

64. Black FL. Measles antibodies in the population of New Haven, Connecticut. J Immunol 1959; 83: 74-83.

65. Simpson REH. Infectiousness of communicable diseases in the household (measles, chickenpox, and mumps). Lancet 1952; 2: 549-554.

66. Martin D, Schoub BD. Measles - 1988 S Afr Med J 1988; 74: 471.

67. Graziano KD, Ruckdeschel JC, Mardiney MR. Cell associated immunity to measles (rubeola). The demonstration of in vitro lymphocyte tritiated thymidine incorporation in response to measles complement fixation antigen. Cell Immunol 1975; 15: 347-359.

68. Labowskie RJ, Edelman R, Rustigian R, Bellanti JA. Studies of cell mediated immunity to measles virus by in vitro lymphocyte-mediated cytotoxicity. J Infect Dis 1974; 129: 233-239.

69. Ruckdeschel JC, Graziano KD, Mardiney MR. Additional evidence that the cell-associated immune system is the primary host defence against measles. Cell Immunol 1975; 17: 11-18.

70. World Health Organisation Collaborative Study by the Ministry of Health of Kenya and WHO Measles immunity in the first year after birth and the optimum age for vaccination in Kenyan children Bull WHO 1977; 55: 21-29.

71. Whittle HC, Rowland MGM, Mann GF, Lamb WH, Lewis RA. Immunisation of 4-6 month old Gambian Infants with Edmonston-Zagreb Measles Vaccine. Lancet 1984; 13: 834.

72. Krugman S. Further attenuated measles vaccine: characteristics and use Rev Inf Dis 1983; 5: 477-81.

73. Ministry of Health of Kenya and the World Health Organisation. Measles immunity in the first year after birth and the optimum age for vaccination in Kenyan children. Bull WHO 1977; 55: 21-31.

74. Bass JW, et al. Booster vaccination with live attenuated measles vaccine. JAMA 1976; 235 (1): 31-34.

75. Stanfield JP, Bracken PM. Measles vaccination studies in methods and cost reduction in developing countries. Trans R Soc Trop Med Hyg 1971; 65: 620-628.

76. Halsey NA. The optimal age for administering measles vaccine in developing countries In: Recent advances in immunisation. Pan American Health Organisation 1983: publication 451. Quoted in "Whittle HC, Rowland MGM, Mann GF, Lamb WH, Lewis RA. Immunisation of 4-6 month old Gambian Infants with Edmonston-Zagreb Measles Vaccine. Lancet 1984; 13: 834."

77. World Health Organisation. Weekly Epidemiol Rev 1979; 54: 337-339.

78. King B. Measles vaccination in a rural Tanzanian community. East Afr Med J 1978; 55: 252-255.

79. Mann GF. New approaches to the practical problems of measles vaccine Proc symposium on stability and effectiveness of measles, poliomyelitis and pertussis vaccines. Yugoslav Academy of Sciences and Arts Zagreb 1976. Quoted in "Whittle HC, Rowland MGM, Mann GF, Lamb WH, Lewis RA. Immunisation of 4-6 month old Gambian Infants with Edmonston-Zagreb Measles Vaccine. Lancet 1984; 13: 834."

80. Sabin AB, Arechiga AF, de Castro JF, Sever JL, Madden DL, Shekarchi I, Albrecht P. Successful immunisation of children with and without maternal antibody by aerolised measles vaccine. JAMA 1983; 249: 2651-2662.

81. Wilkins J, Wehrle PF. Additional evidence against measles vaccine administration to infants less than 12 months of age: altered immune response following active/ passive immunisation. J Pediatr 1979; 94: 865-869.

82. Heyman DL, Nakano JH, Maben GK, Durand B. Field trial of a heat stable measles vaccine in Cameroon. Br Med J 1979; 2: 99-100.

83. World Health Organisation. Expanded programme on immunisation. Ice-lined refrigerators. Weekly Epidemiol Rec 1984; 59: 63-64.

84. World Health Organisation. Expanded programme on immunisation. The cold chain status. Geneva: World Health Organisation 1984. Quoted in "Ebrahim GJ. Immunization in Childhood-current trends and new developments. J Trop Pediatr 1987; 33: 66-67."

85. World Health Organisation. Applied research and development priorities in the Expanded Programme on Immunization. Geneva: World Health Organisation 1984. Quoted in "Ebrahim GJ. Immunization in Childhood-current trends and new developments. J Trop Pediatr 1987; 33: 66-67."

86. Katz SL. Summary of Current Status of Measles and Recommendations. Rev Infect Dis 1983; 5: 623-624.84.

87. Ifekwunigwe AE, Grasset N, Glass R, Foster S. Immune response to measles and smallpox vaccinations in malnourished children. Am J Clin Nutr 1980; 33: 621-624.

88. Mc Murray PN, Loomis SA, Casazza LJ, Rey H. Influence of moderate malnutrition on morbidity and antibody response following vaccination with attenuated measles virus vaccine. Bull Pan Am Health Organ 1979; 13: 52-57. Quoted in "Walsh JA. Selective primary health care: strategies for control of disease in the developing world. lV. measles. Rev Infect Dis 1983; 5: 330-340."

89. Modlin JF, Halsey NA, Eddins DL, Conrad JL, Jabbour JT, Chien L, Robinson H. Epidemiology of subacute sclerosing panencephalitis. J Pediatr 1979; 94: 231-236.

90. Schwachman , Kulczycki L, Katz SL. Attenuated Measles Vaccine in Children with Cystic Fibrosis. Am J Dis Child 103: 405-406.

91. Edozien JC, The serum proteins in Kwashiorkor. J Pediatr 1960; 57: 594-603.

92. Black F, Berman L, Libel M, Reichelt C, Pinheiro F, Da Rosa A, Figueira F, Gonzales E. Inadequate immunity to mmeasles in children vaccinated at an early age: effects of revaccination. Bull WHO 1984; 62: 315.

93. Ministries of Health of Brazil,Chile,Costa Rica and Ecuador, and the Pan American Health Organisation. Seroconversion rates and measles antibody titres, induced by measles vaccine in Latin American children 6-12 months of age. Bull Pan Am Health Organ 1982; 16: 272-85.

94. Black F, Berman L, Libel M, Reichelt C, Pinheiro F, Da Rosa A, Figueira F, Gonzales E. Inadequate immunity to measles in children vaccinated at an early age: effects of revaccination. Bull WHO 1984; 62: 315.

95. Heymann D, Mayben G, Murphy K, Guyer B, Foster S. Measles Control In Yaounde: Justification of a one dose, nine month minimum age vaccination policy in Tropical Africa. Lancet 1983; 2: 1470-1472.

96. Dick B. The Provision of Measles Vaccine for an Urban Population. S Afr Med J 1975; 30: 1507.

97. Williams PJ, Hull HF. Status of measles in The Gambia, 1981. Rev Infect Dis 1983; 5: 391-94.

98. Jawetz E, Melnick JL, Adelberg EA. Review of Medical Microbiology. 17th ed.Norwalk,Connecticut Los Altos,California: Appleton & Lange 481.

99. Ebrahim GJ. Immunization in Childhood-current trends and new developments. J Trop Pediatr 1987; 33: 66-67.

100. Risi JB. The control of poliomyelitis in Brazil. Rev Infect Dis 1984; 6:(2) 400-403.

101. Jordan WS. Measles immunization: Remaining Needs for Research. Rev Infect Dis 1983; 5: 613-618.

102. Reynolds LGvB. Measles in South Africa. S Afr Med J 1987;72: 507.

103. World Health Organisation. Expanded programme on immunisation: programme review: Pakistan. Weekly Epidemiol Rec 1985; 60: 253-6.

104. Ponninghaus J. The cost benefit of measles immunisation. A study from southern Zambia. Las Vegas, Verlag Peter Lang, 1979: 40-48. Quoted in "Walsh JA. Selective primary health care: strategies for control of disease in the developing world. lV. measles. Rev Infect Dis 1983; 5: 330-340."

105. Djukanovic V, Mach EP. Alternative approaches to meeting basic health needs in developing countries. Geneva 1975; World Health Organisation. Quoted in "Ebrahim GJ. Immunization in Childhood-current trends and new developments. J Trop Pediatr 1987; 33: 66-67."

106. Anonymous. Expanded programme on immunisation. Evaluation of immunisation coverage. Weekly Epidemiol Record 1982; 57:(32) 241-242.

107. Henderson RH, Sundaresan T. Cluster sampling to assess immunisation coverage: a review of experience with a simplified sampling method. Bull WHO 1982; 60: 253-260.

108. Gibson INH, Carmichael TR, Kustner HGV. Measles notifications-the first year. S Afr Med J 1982; 84: 85.

109. Cameroon. (United Republic) Measles registry, Yaounde Central Hospital 1975-1979. Quoted in "Heymann D, Mayben G, Murphy K, Guyer B, Foster S. Measles Control In Yaounde: Justification of a one dose, nine month minimum age vaccination policy in Tropical Africa. Lancet 1983; 2: 1470-1472."

110. World Health Organization Statistics Annual, 1962; 2: 113.

111. De Jong JG, Winkler KC. Survival of Measles Virus in Air. Nature 1964; 201: 1054.

Form 181 (Health) M 1531

RETURN OF ALL NOTIFIABLE DISEASES MADE TO THE CITY OF JOHANNESBURG DURING THE WEEK ENDED SATURDAY .....

Name of Person	Age	Sex	Race	Address	Disease	Date Notified	Notified by	Place, Date, Source of Infection	Action Taken

APPENDIX A

JOHANNESBURG DATE: ..... SIGNED: .....

for MEDICAL OFFICER OF HEALTH

CITY OF JOHANNESBURG - CITY HEALTH DEPARTMENT M2660

MEASLES NOTIFICATION

NAME.....

ADDRESS.....

.....

.....

PERMANENT
HOME ADDRESS.....

.....

.....

POPULATION GROUP | W | C | A | B |

SEX.....

BIRTH DATE.....

BIRTH PLACE .....

NOTIFIED ON.....

NOTIFIED BY.....

DATE OF ARRIVAL IN JOHANNESBURG.....

IMMUNISED AT CLINIC:.....

IMMUNISATION (Verified Dates)

BCG.....

DWT/DT (1).....(2).....(3).....(B).....

POLIO (1).....(2).....(3).....(B).....

MEASLES (1).....(2).....

If <u>not immunised</u> against measles state reasons:-

.....

.....

.....

.....APPENDIX B.....

Appendix C.

Data Sheet 1. Year. 19__. Sheet number.____

Pt. No.	Pt. Name.	D.O.B.	1st.V	2nd.V	Res. Address.	Not. Auth.