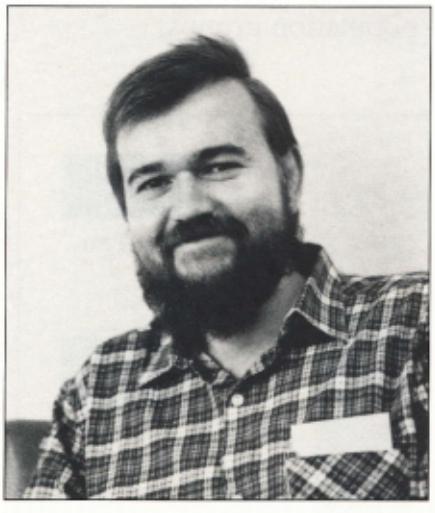
Measles in South Africa: A Comprehensive Interpretation of the Data. Part III. Other Factors influencing measles morbidity and severity in SA (Protein-energy-malnutrition, age at infection, urbanisation, socio-economic status and health care — P Ferrinho, E Buch



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Curriculum vitae

Paulo Ferrinho had his school education in Mocambique and then went on to the University of Cape Town to obtain the MBChB in 1980. He did his internship at the Groote Schuur hospital in Cape Town and then worked at the Gelukspan Community Hospital from 1982 to 1986. After that he became a registrar in Community Health at the University of the Witwatersrand. At the moment he is the Clinic Manager and Director for Research of the Alexandra Health Centre and University Clinic/Institute for Urban Primary Health Care. Although specialising in Community Health, his professional interest remains in support to primary health care.

Summary

In South Africa (SA) there is a commitment to, and indications that resources are being allocated for the eradication of measles. Still there has been no comprehensive review of the epidemiology of the disease in SA. This understanding is important to identify factors and trends to guide public health practice. This series of articles tries to cover this gap.

Part I reviews briefly the international literature on the epidemiology of the disease and describes the methodology followed, the sources of data and analysis strategy. The other articles review South African reports on morbidity — mortality and relationships to age, population group, sex and geographical distribution (Part II); Part III reviews other factors influencing measles morbidity and mortality in SA (protein-energy-malnutrition, age at infection, urbanisation, socio-economic status and health care); and Part IV contains appropriatep conclusions and recommendations.

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KEYWORDS:
Measles; Epidemiology; Age Factors; Socio-economic Factors; Urbanization; Measles Vaccine; Vitamin A

Introduction

In this article we show that measles still remains a common problem in South Africa (SA) with a high case fatality rate (CFR) and that "race", vaccination coverage, poverty, urbanisation and vitamin A deficiency are important factors in understanding its morbidity and mortality.[1,2]

There are other possible determinants of incidence and severity of measles.[1,3] Because of their importance in SA and the availability of data, we examine the influence of protein-energy-malnutrition, age at infection, urbanisation, socio-economic circumstances and availability of health care on incidence and severity for the country.

Protein-Energy-Malnutrition and Measles

There are South African reports that identify protein-energy-malnutrition as an important determinant of measles severity.[4-11] However, all these reports are hospital-based and fail to control factors like overcrowding and micro-nutrient deficiencies, which are more likely to be the relevant determinants in socio-economic environments where malnutrition is common.[12-18] As vitamin A deficiency seems to be common in SA,[8,9,19-23] the high measles CFR observed could, to some extent, be due to this.

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Morbidity - Mortality, Age Distribution and Population Group

Measles data by age and population group is analysed for case notifications, deaths notifications, certified deaths, active surveillance, outbreak investigations, hospital data and data community surveys.

Although the age structure for each population group in South Africa varies, the overall national age structure is that of a developing country with less than 5% of the population under one year of age, 20% below 5 years, 15% 5 to 9 years, 15% 10 to 14 years and 50% below 15 years.

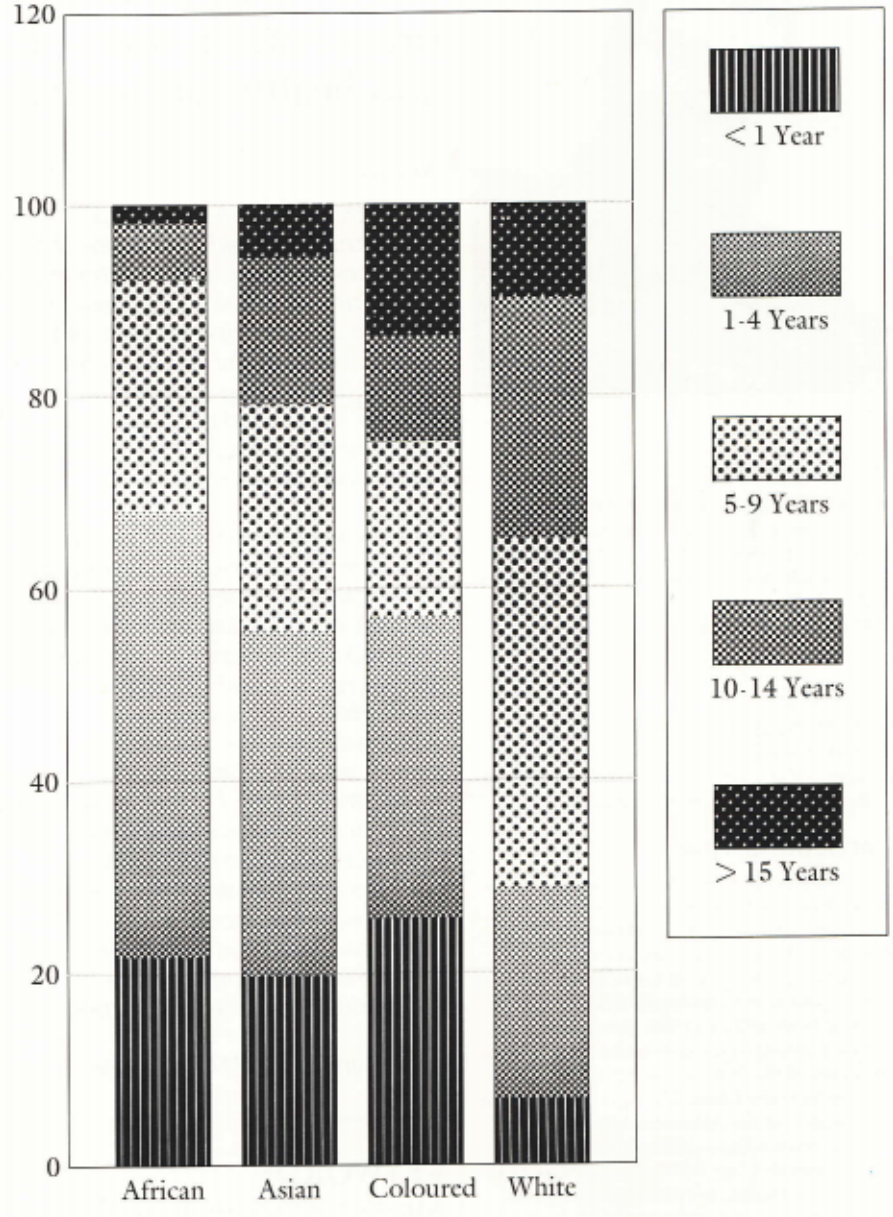
Notifications

For 1985-1987 a significant proportion (26% for Coloureds, 22% for Blacks, 20% for Asians and 7% for Whites) of notified measles occurred in children under one year of age (Fig 1). A similar age pattern can be seen for the period 1988 to July 1990. Although younger age is likely to lead to increased chances of notification, due to increased hospitalisation and mortality, this is still a very high proportion.

Children 1-4 years of age account for 46% for Blacks, 36% for Asians, 31% for Coloureds and 22% of the notified disease for Whites.

In 5-9 year olds, Blacks still have the highest age specific incidence, but Coloureds and Asians now have lower rates than Whites.[24] In children over 10 years of age Whites have the highest incidence rates.[24] Notification data also demonstrates that in the population groups with an average household size greater than five

Fig. 1 Notified measles per age and population groups, 1985-1987



(Asians, Blacks and Coloureds) the risk of measles infection amongst the under-ones is three to six times that of older children.[24]

Regional reports from the Cape indicate similar trends to national notifications. In Cape Town the notification rate was 60 per 100 000

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in 1986 with rates in children under 1 year of age of 816 per 100 000.[25]

The Cape data also suggests that urbanised children are more likely to get infected at a younger age than rural children.[25] In Kangwane and Bophuthatswana excess notifications occur in children 5-9 years of age and in all the other areas in the first year of life.[26]

There is no report available that provides an age breakdown of notified deaths.

Active surveillance data

In Johannesburg-Benoni the median age for all notified measles cases was 37 months, 10% of the cases were younger than 9 months and 18% in Johannesburg and 19% in Benoni were under 12 months.[27]

Outbreak investigations

The Port Elizabeth epidemic in 1983 showed the highest and the earliest incidence of measles in the most overcrowded shanty camps. The mortality was particularly high in the socio-economically poor squatter areas. Fifty four percent (54%) of Blacks and 62% of Coloured deaths were < 1 year of age (84% and 79% respectively were < 2 years of age). The highest age specific CFR was in children under 6 months of age (44%).[28]

Resources have been allocated to eradicate measles, but no epidemiology available

Hospital data

During 1985-1986, 72% of children admitted to the City Hospital with acute complicated measles were children aged 15 months and under.[29] Similar figures were found in 1973 where 90% of cases were below 3 years of age, 50% were younger than 15 months, and 25% were less than 10 months old.[30]

A report from the intensive care unit at the Red Cross War Memorial Children's Hospital, Cape Town, identifies the median age of children as 9 months, with 9% of the admissions in children younger than 6 months (January 1985 to April 1986).[31]

Vit A deficiency may be the reason for the high case mortality

In Clairwood and King Edward Hospitals in Natal, 28% of 111 measles cases were below 9 months (August 1986).[32] In Baragwanath (in 1987) 72% of admissions were under 2 years and 36% were 9 months or younger.[33]

Data from the City Hospital in Cape Town and from the Red Cross War Memorial Children's Hospital illustrate the high load of measles mortality in children under five years, particularly infants[29-31] while in King Edward Hospital mortality in the very young is very high with a CFR of 26% for infants under 8 months.[34]

In Letaba, Gazankulu, 41% of 109 admissions with measles (1985-1986) were below 5 years.[35]

In some rural areas there is a noteworthy trend for measles admissions to occur in older age groups, probably because of successful efforts to achieve adequate vaccination coverage. Crisp et al report that the average age of measles admissions in the Elim Health Ward of Gazankulu, increased from 29,7 months in 1976 to 60,9 months in 1986.[36] In Venda in 1985 an epidemic of measles involved children of an age group "older than normal".[37] In the Gelukspan Health Ward of Bophuthatswana measles is no longer a problem of under five children;[38-40] recent outbreaks occurring in primary school children (Sutton C: personal communication, 1988).

Miscellaneous

Loenig and Coovadia[41] used an urban community sample and samples of patients attending several health facilities (2 urban hospitals, 1 peri-urban hospital and 3 rural hospitals) to study incidence and mortality differentials for measles (1978 to 1981). Their most important finding was that over 25% of children in an urban environment get measles at a very young age, with the proportion decreasing as the population became more rural. It was unexpected to find the proportion of measles occurring at 8 months and under, to be higher in the urban community than in some

50% of our population is below 15 years

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of the hospitals, despite the urban sample, community based, being more likely to be biased to older children than the peri-urban and rural samples, both more hospital and clinic based.[41]

Certified deaths

Bradshaw et al using death certificate data supplied by the Central Statistical Services, analyzed measles share of mortality in different age groups.[42] For blacks, in all age groups, measles takes a greater share of the mortality. For all population groups, except Asians, the share is highest in the age group 1-4 years.

The age pattern of certified measles deaths is very similar to the pattern of notified deaths.

For 1985 age specific incidence for each population group were

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Very high proportion of notified measles in children under one year of age

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calculated using census data as denominators. For children younger than 15 years of age, Blacks carry the highest age specific mortalities, followed by Coloureds, Asians and Whites. From 15 to 19 years the highest mortality is for Coloureds then Whites, Blacks and Asians. Over 20 the mortality is highest in Blacks followed by Coloureds (Table I).

For Blacks and Coloureds, infants account for over 35% and under fives for more than 90% of certified deaths. For Asians, infants account for 44% of deaths, but only 80% are under-5 years of age. For Whites the infant share of mortality has been stable at 30% and up to 22% of all deaths occur after the age of 5 years (Table II). Infant share of mortality in all non-white population groups seems to be on the increase, although this is statistically significant only for Coloureds (1968-1986) ($r=0,649$, $p=0,002$).

The significant increase in Coloured infant share of measles mortality has been accompanied by a significant decrease in mortality share of children 1 to 4 years old (1968 to 1986) (the trends of the two age groups differ significantly, $p<0,001$).

The only statistically significant trend for Asians has been a decreasing

Table I. Age Specific Mortality Rate per 100 000 Population (Certified Mortality, 1985)

Age	Population Group			
Group	Asian	Black	Coloured	White
0- 4 Years	7,92	38,03	28,86	0,00
5- 9 Years	2,21	2,41	1,25	0,00
10-14 Years	0,00	1,13	0,28	0,00
15-19 Years	0,00	0,10	0,29	0,23
20 OR +	0,00	0,11	0,07	0,00

Table II. Percentage of Certified Measles' Deaths under 1 and 5 years of age for each population group

		Years			
Age	Population Group	1968- 1971	1972-1976	1977-1981	1982-1986
<1y of age	Africans*	NA#	NA	41%	43%
	Asians	20%	35%	16%	44%
	Coloureds	34%	35%	42%	43%
	Whites	24%	30%	33%	30%
<5y of age	Africans*	NA	NA	95%	92%
	Asians	94%	95%	82%	80%
	Coloureds	96%	96%	95%	95%
	Whites	77%	79%	82%	78%

* For the African Population Data is only available from 1979 to 1986. This data is therefore reported in two 4 years goups, 1979-1982 and 1983-1986.
\# NA=NOT AVAILABLE

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share of the mortality in the 1 to 4 years age group (1968-1986) (r=0,640, p=0,002) and for Blacks an increase in the mortality share in the 5 to 9 years age group (1979 to 1986, r=0,821, p=0,010) (1980-1986, r=0,778, p=0,038).

For Whites of 20 years and older there is a statistically significant decrease after 1980 (r=0,832, p=0,017).

Summary

In summary, measles occurs at younger ages for Blacks and for the urbanised, particularly those in overcrowded shanty conditions and in areas where vaccination coverage is poor.

It seems that age trends, as reflected in hospital admission figures, are conflicting. Some areas, particularly the rapidly urbanising, eg Cape Town, report a decreasing age at infection with measles. Other areas, in this case those with high vaccination coverage, report precisely the reverse.

The morbidity and mortality trends are consistent with the lower socio-economic status of the Blacks and Coloureds, an intermediary situation for the Asians and the best socio-economic indicators for the Whites.

What is apparent from comparing disease notification data with death certification data is that infants, and to a lesser extent under fives have a mortality load far in excess of their share of the disease load (Table III).

The age-specific share of mortality trends vary between population groups.

CFRs and age specific mortality rates are highest in infants.

We must remember that all data is defective in terms of underascertainment and in terms of underestimates of some of the denominators. When we consider gradients between population groupings, we must not forget that underlying them are also gradients in data quality, with the highest at risk having the worst quality data set. Therefore the differentials we are looking at are minimum estimates rather than precise values.

Table III. Proportion of Cases and Deaths, in each population group, that are either less than 1 or less than 5 years of age.

Age	Population Group	Morbidity*	Mortality#
< 1 year	Africans	23%	43%
	Asians	21%	44%
	Coloureds	31%	43%
	Whites	8%	30%
< 5 years	Africans	69%	92%
	Asians	59%	80%
	Coloureds	66%	95%
	Whites	32%	78%

* Notified Data
\# Certified Deaths

Interpretation of the Morbidity - Mortality Data

The importance of age at infection as a determinant of measles severity and mortality is not easy to interpret.[43-46] Data from other countries suggest that CFR are higher in population groups with higher mean age at infection.[45] On the other hand, age-specific CFR point to the fact that CFR are much higher in the younger infant. Also, as vaccination increases the mean age at infection increases and the CFR decreases.

A possible explanation for this conflicting information is as follows: in unvaccinated (or low vaccination coverage) populations, particularly in

Urbanised children get infected at a younger age than rural children

rural settlements, higher mean age at infection is more likely to be associated with an epidemic pattern of the disease. Here the presence of the virus in the community is short lived but in high concentration. All those susceptible, independently of age, are infected, frequently with multiple cases in the same household. The high dose of infection is likely to result in more severe infection with a high overall CFR, particularly in the young. In contrast, in the endemic situation, mostly in large, densely populated, urban areas, measles occurs the year round. As there are no epidemics, the measles virus is at a constant low level. Siblings tend to be infected in different years and there is no accumulation of susceptibles in the older ages. So, younger children

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are infected with a lower infective dose, but still with a high CFR, although lower than that in the epidemic situation, where inter alia, multiple household cases may increase mortality.[16] This explanation is still largely speculative.

To be able to advance a logical explanation of what is happening at community level in SA we need to know the mean age at infection, the overall CFR and age specific CFR, the vaccination coverage and the endemic or epidemic nature of the disease. We are not aware of any single community in SA where all of these indices are known.

Infant share of mortality in all non-white groups is on the increase

Race is also not a known determinant of measles incidence and severity. There is no reason to believe that the genetic factors associated with severity[47] have a distribution amongst the different population groups such that they make a difference to the epidemiology of the disease. Rather, race as a proxy of socio-economic circumstance, the nature of the settlement (urban or rural), household size and, vaccination coverage could be the main factors interacting to determine age at infection.

Furthermore, the epidemiological patterns present today in less developed countries are similar to those in more industrialized countries in earlier times. These patterns started to change in the more developed countries with socio-economic change[48-50] and changed radically with the introduction of vaccination.[51]

Thus, racial differences in SA are taken as a reflection of different socio-economic realities. We suggest that age at infection seems to be a dubious direct determinant of severity. Examination of the measles data in relation to differentials of urbanisation supports (but does not necessarily prove), this interpretation.

Urbanisation

Urbanisation represents all the processes of social change in the urban environment. The relationship between urbanisation and measles, is not a simple one. Whites have a lower incidence of notifications and lower CFR than both Asians, who are more urbanised, and Coloureds and Blacks, who are less urbanised. Also Kettles' analysis of notifications in the Western Cape shows that the more urbanised Blacks have a higher incidence and background rate of notifications and more frequent measles peaks than the least urbanised.[25] This work is consistent with data that showed that measles incidence was highest in urban blacks and lowest in rural Blacks.[41] In Black urban areas the mean age at infection is lower than in Black rural residents.[52] The limited data available also suggests that hospital case fatality rates are higher for urban Black residents.[53]

What seems an obvious conclusion is that, in SA, urbanisation under poor socio-economic conditions is associated with a higher incidence of measles at a younger age and with a higher fatality rate. The reverse seems to apply to urbanisation under good socio-economic conditions. Therefore, the operative factor in the

Table IV. Notified Incidence per 100 000 Population in Areas with known vaccination coverage for measles.

Area	Incidence/100 000 (year)	Vac Coverage (year)
Botshabelo	19,5 (1988)	31% (1989)
Praktiseer	88 (1988)	48% (1989)
Molopo	40,4 (1988)	50% (1989)
Nsikazi	42,5 (1988)	56% (1988)
Alexandra	30 (1988)	67% (1988)
Alexandra	25 (1988)	78% (1990)
Odi	0,0 (1988)	78% (1989)
Taung	14,5 (1988)	81% (1985)
Malamulele	121,7 (1988)	82% (1987)
Gelukspan	1,9 (1988)	84% (1988)
Elim	25,7 (1988)	85% (1985)

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Table V. Vaccination Coverage with the third dose of DWT and with the first dose of Measles.

DWT 3 coverage greater than MEASLES coverage			
Area	Year	DWT3 % Coverage	Measles % Coverage
Molopo	1984	75%	59%
Elim (Ritavi)	1985	94%	85%
Malamulele	1987	83%	58%
Eersterus	1989	92%	90%
Hillbrow	1989	75%	70%
Laudium	1989	97%	87%
Odi (12/12)	1989	81%	78%
Pretoria	1989	95%	93%
DWT3 coverage less or equal to MEASLES coverage			
Gelukspan	1985	81%	81%
Taung	1985	65%	81%
Bethesda (Ubombo)	1985	58%	58%
Gelukspan	1985/86	83%	88%
Ingwavuma (Mosvold)	1986	56%	56%
Alexandra	1988	66%	67%
Gelukspan	1988	82%	84%
Nsikazi	1988	50%	56%
Botshabelo	1989	31%	31%
Kayelitsha (Site C)	1989	57%	63%
Praktiseer	1989	46%	48%

epidemiology of measles and urbanisation is socio-economic status.

Socio-Economic Status

Socio-economic data is not available on notification forms. Still, it is well known that in SA race is closely related to socio-economic status, overcrowding, degree of urbanisation and accessibility to health care. Therefore, using race as a proxy for socio-economic status, it is apparent that in SA the poorest, least urbanised, most overcrowded group with the least access to health care, ie Blacks, have the highest incidence of and CFR for measles.

This was illustrated during the 1982-1983 measles epidemic in Port Elizabeth when 88% of notifications and 91% of deaths were in the Blacks who accounted for only 50% of the population. This was in comparison to only 1% of cases and 0,3% of deaths amongst Whites who comprised 26% of the population. The areas most affected were the most deprived socio-economically, with the most overcrowding and worst housing.[28]

In Cape Town the overall notification rate for 1986 was 60 per 100 000, but for Blacks it was 178. Seventy five per cent (75%) of cases admitted with complicated acute measles to the City Hospital were Blacks from the Peninsula.[29]

Health workers from Durban reported an average of 9,43 persons per Black household with measles, while the overall average for Blacks in SA is 5,94 and for Whites, 3,62.[54] Notification data demonstrates that in the population groups with an

Overcrowding in poor urban areas the reason for high rates of severe measles in RSA

average household size greater than 5 (Asians, Blacks and Coloureds) the risk of measles infection amongst the under ones is 3 to 6 times that of older children.[24]. Eighty per cent (80%) of cases of measles studied in Johannesburg-Benoni in 1988 stayed in houses with a crowding index of more than 2,5 persons per sleeping room.[27]

It is true that under poor socio-economic conditions, when measles is

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more common and severe, protein-energy malnutrition is also more prevalent. As discussed before, protein-energy malnutrition is now believed to be a confounder in the model that explains the epidemiology of measles. What is apparent from the literature, and SA data is compatible with such literature, is that heavy dose of infection seems to be the main determinant of measles severity. It is also suggested that the dose of infection is most commonly related to the degree of overcrowding in the community, particularly overcrowding of children. The overcrowding in the poor urban areas with poor housing and and ventilation would then contribute to a high dose of infection with the measles virus and be the reason behind high rates of severe measles in SA.

Age at infection not really a direct determinant of severity

In SA the poor and urbanised blacks would be, because of overcrowding and poor ventilation, the group at higher risk for high dose of infection. This risk could be successfully counteracted by high vaccination rates but the urban poor, particularly the Black urban poor, are also a politically deprived group with neglected provision of health care.

Health Care Provision

Health care is an important modelling factor of measles morbidity and mortality. It is discussed under 3 headings: health

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care facilities as a source of measles to the community; vaccination against measles; and care of the child with measles.

Health care facilities as sources of infection to the community

An irony is that the development of clinics and hospitals, has brought susceptible children into contact with measles cases. Thus, health care facilities have become important vectors in the spread of measles in other developing countries[55-58] and in SA.[29,31,32,59,60] Wittenberg in a survey of 111 new measles admissions found that 59 (53%) had attended a clinic or hospital 15 to 7 days before the onset of illness.[32] Reynolds reported that 25% of all cases of life threatening measles in Cape Town acquired the disease in hospital or in outpatients.[31] A survey of measles admissions to the City Hospital for Infectious Diseases over a 3-month period again identifies that 20 (32%) of 61 admissions had a contact with a health care facility in the 10-14 days preceding the onset of measles.[29]

The prognosis of hospital acquired measles is particularly severe.[56,58] Observations of children with measles, some of them dating back to the past century, relate severity of measles infection to history of or concurrent occurrence of other disease or respiratory tract infection.[49] The poorer prognosis of hospital acquired infection could be related to this phenomenon.

The above facts resulted in recommendations to protect with vaccination[10,57] or immunoglobulin,[59] children coming in contact with health facilities. The public health importance of such measures became recently apparent during a survey of health facilities in the western Cape.[60]

Preventive health care: vaccination against measles

Vaccination is essential to the control of measles. We already mentioned the falling incidence and CFR of measles in areas with successful vaccination campaigns. In SA there are vast areas in the homelands, in the "white" farm areas and in peri-urban squatter camps where health care is absent or, where preventive care is not aggressively promoted. Table IV reviews data from areas where we do have morbidity and or mortality data and data on vaccination coverage. It is apparent that, with the exception of Malamulele in Gazankulu,[61] vaccination coverage above 70%, is associated with lower incidence of measles.[42,62-70]

No obvious reason to shift age of vaccination to 6 months

The data from Malamulele[61] can be explained in 6 ways: it could be a reflection of the methodology used; high vaccination coverage in children between 12 and 23 months of age but much lower in older children with outbreaks of disease in older children; cold chain failure with concomitant failure to impart immunity to children; above average notification services when compared with other areas although this is unlikely; the prevalence of poor environmental and socio-economic conditions in Malamulele with extreme clustering of children not vaccinated, allowing for outbreaks of disease in the presence of high vaccination coverage; lastly, high density housing, would be associated with the occurrence of measles before they had the opportunity to be vaccinated.

Also apparent from the existing vaccination surveys are data that show that usually the coverage figure with measles vaccine is not higher than for DWT3[38-40,61-67,69-77] (Table V). There is therefore no operational advantage in recent recommendations to shift the age of vaccination to 6 months of age.[78,79]

Clinical care of the sick child with measles

The treatment of measles has been up to now of a supportive nature. In uncomplicated measles the emphasis has been on control of pyrexia, nutritional support and hygiene.[80] The development of complicated measles has always been followed by treatment specific to the complications. Morley has, for more than 20 years, advocated the use of vitamin A supplementation in areas known to be deficient in this nutrient, to prevent the blinding complications of measles.[80]

More recently there has been tremendous interest in the use of vitamin A to reduce mortality.[81,82] On the basis of a small randomized control trial,[83] which did not achieve statistical significance for any of its findings, the WHO has been promoting the use of vitamin A supplementation to reduce mortality in areas with CFRs equal or in excess of 1%.[81,82] This recommendation, first mentioned and ignored in 1932,[84] has not been followed in SA.

A more recent randomized control trial in Cape Town found strong

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supportive evidence for the routine use of vitamin A in children with measles complicated by pneumonia, diarrhoea or croup, admitted to hospital within 5 days of the onset of the rash. The Cape Town trial found twice as many deaths in the control group as in children given a high dose of vitamin A. The treatment group also had a more rapid recovery from pneumonia and diarrhoea, less croup and shorter admissions. Vitamin A supplementation in severe measles has therefore the potential to halve mortality and reduce morbidity by one third.[8,9]

What has not yet been proven, and requires urgent investigation, is the value of vitamin A supplementation to prevent severe measles, by supplementation before the onset of complications.

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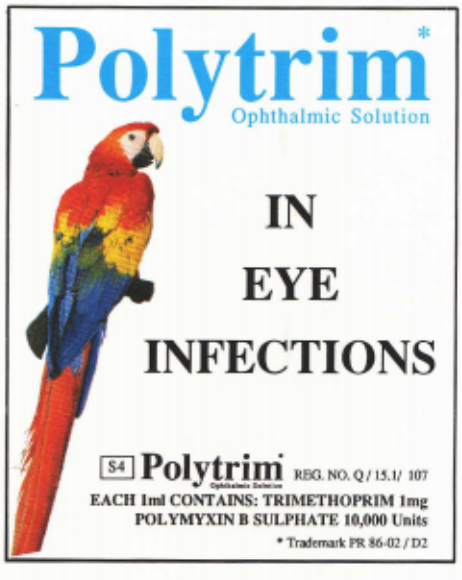
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