OBAFEMI AWOLOWO UNIVERSITY, ILE-IFE, NIGERIA.

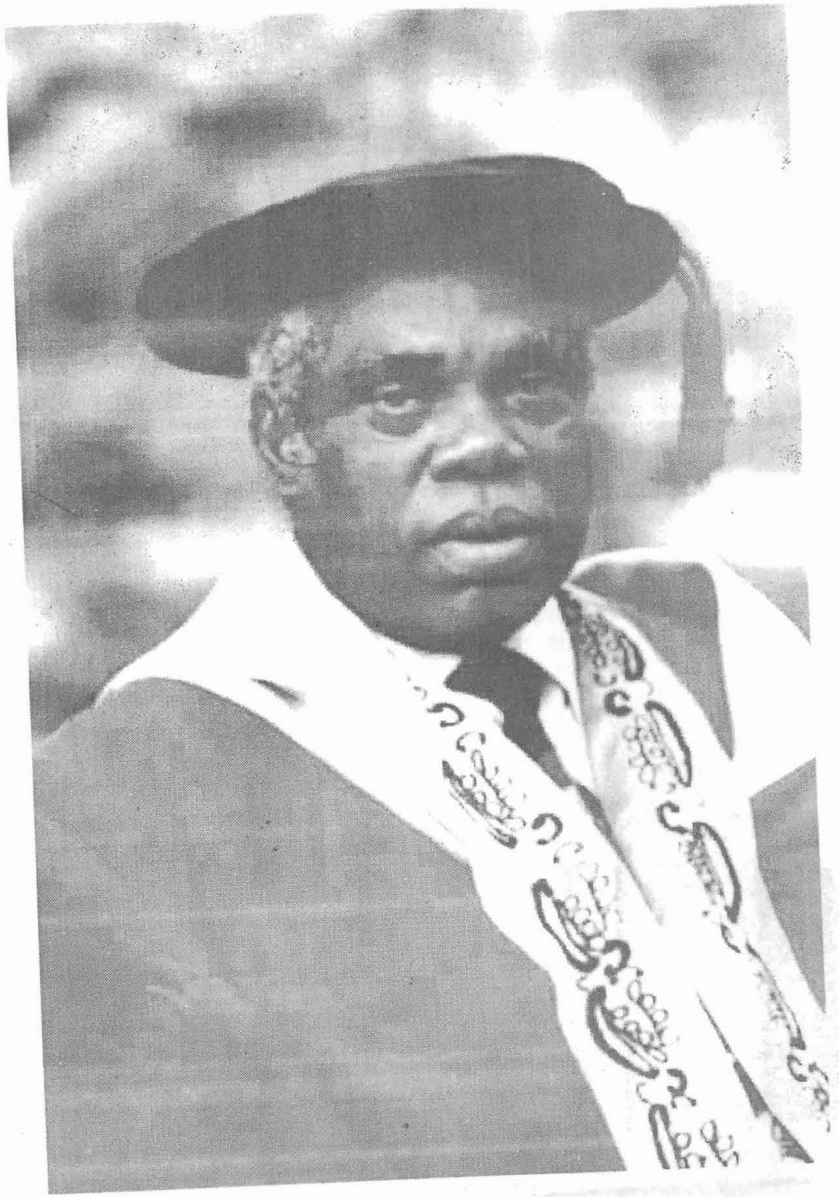
Inaugural Lecture Series 163

THE 950 LIVE BIRTHS IN NIGERIA: THE FUTURE IS NOW

By

J. Aderinsola Owa
Professor of Paediatrics

OBAFEMI AWOLOWO UNIVERSITY PRESS LIMITED



Professor Joshua Aderinsola Owa
Professor of Paediatrics

THE 950 LIVE BIRTHS IN NIGERIA: THE FUTURE IS NOW

By

J. Aderinsola Owa

Professor of Paediatrics

An Inaugural Lecture Delivered at Oduduwa Hall
Obafemi Awolowo University, Ile-Ife
On Tuesday November 4, 2003

Inaugural Lecture Series 163

Obafemi Awolowo University Press Limited
Ile-Ife, Nigeria

ISSN 0189 – 7848

Printed by

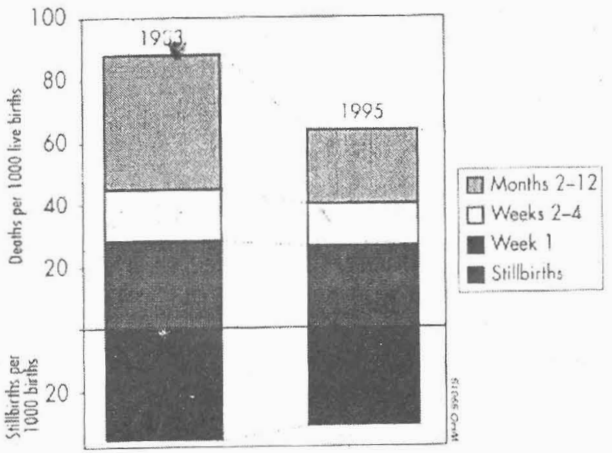
Obafemi Awolowo University Press Limited

Ile-Ife, Nigeria

INTRODUCTION

Perinatal and neonatal mortality rates have been used as indicators of health status, especially for international comparisons. They provide a measure of a country's educational, social and public health systems as well as that of nutrition, obstetric medicine and **neonatology**. Perinatal and neonatal mortality rates thus reflect standards of obstetric and paediatric care, as well as the effectiveness of social measures in general and public health actions in particular. They also provide a measure of socioeconomic development. Each year, more than 8 million infants die worldwide before they are one year, and 98% of the deaths occur in the developing countries[1], where infant mortality remains high (Figure 1).[2] And two-thirds of these deaths occur in the first 28 days of life.[3,4]

Figure 1: Stillbirths and Deaths in the First Week of Life Remain High in Developing Countries[2]



This lecture is therefore dedicated to health status of the most vulnerable children in Nigeria today: the first 28 days in the life of 950 live births per 1000 viable pregnancies. This is based on the dictum that children of today are the leaders of tomorrow. I am not sure we believe much in that saying judging from the priority we give to the survival and well-being of our newborn infants in Nigeria. We probably believe more in the dictum of the "survival of the fittest". Unfortunately, the fittest survival may never achieve his/her full potential or even half of his/her potential with levels of care available to most of our pregnant women and newborn babies today.

I have been involved in looking after this group of patients in Nigeria for over a quarter of a century. I can therefore safely say that I am part of them.

'Survival' is usually defined negatively: ***as if to survive is not to die.***[5] Infant survival is seen as not dying before the age of one. Child survival programmes place emphasis on avoiding death, usually measured by reduction in mortality rates – **I**nfant **M**ortality **R**ate (**IMR**) or **N**eonatal **M**ortality **R**ate (**NMR**) for example. While death is a dramatic and a final event and can be counted with relative ease and accuracy, death seldom occurs instantly. Most deaths follow a period of illness and deterioration that can be painfully prolonged or relatively short. Dying is a process, the end of which is death. Likewise, living is a process, the end of which is not only to survive, but to have physical, mental, and social well being.[5,6] Child survival can be defined more positively, as something more than ***avoiding death***. Surviving children fall along a continuum running from near death through sickness to a healthy state. The process of surviving then is actively seeking a healthy state, moving toward the healthy end of the ***death-sickness-health*** spectrum rather than simply preventing or arresting the process of dying.

Accepting this positive conceptualization of **neonatal survival** – as a process of seeking a healthy state at birth and early months

of life – requires looking beyond analysis of causes of mortality and beyond programmes that simply reduce mortality. Survival defined positively rather than as "not dying" should be indexed by **I**nfant (or **N**eonatal) **S**urvival **R**atio (**ISR** or **NSR**).

The success of programmes should be measured by increase in survival rates rather than decrease in mortality rates. A NSR simply turns NMR upside down. For instance, a NMR of 60/1000 live births in 1980 would give NSR of 940/1000. A NMR of 50/1000 live births (NSR of 950/1000) in 1990 means that the NSR has increased from 940 in 1980 to 950 in 1990: a mere increase of 1.0% as compared to a decrease of 16.7% using NMR.

Suggesting a survival rate (SR) in contrast to a mortality rate (MR) is more than a number game. It emphasizes living, as represented by a SR which provokes different thinking than emphasizing the avoidance of death as represented by the MR. It dramatizes the fact that more fetuses are managing to survive in spite of being "at risk." It ***also*** provokes the question about the **conditions of those who are born alive, about what is being done to cater for the health and welfare of the increasing number of those who are being born alive.**[6]

In this lecture, the term ***survival rate*** is used to emphasize living – the ***increase (or decrease) in the survival and health status of our newborn babies.*** I will start with a brief discussion on the trends in our **perinatal survival rate (PNSR), live birth rate (LBR) and neonatal survival rate (NSR)** and the factors that contribute to our respective slow increase or decrease in survival rates and health status as measured by **PNSR, LBR, NSR**. Lastly, I will make suggestions on the way forward.

PERINATAL SURVIVAL RATE, LIVE BIRTH RATE AND NEONATAL SURVIVAL RATE IN NIGERIA.

Tables I and II summarize some aspects of our perinatal and neonatal data over a ten-year (1981-1990)[7] period and latter half

of last decade (1996-2000)[8] at Wesley Guild Hospital, Ilesa. Over a ten year (1981-1990) period, there was a significant fall in total number of births in the hospital. This was associated with reduction in survival rates reaching dips in 1987 and 1988. Even in the following decade, data by our colleagues[8] showed that the situation had even worsened. While it is not possible to fully explain this, we know that in 1985, user charges were introduced into our hospitals. This coupled with the economic hardship of the time was probably a major factor.[7]

From these data, our LBR over the two decades remains about 950/1000 pregnancies and our NSR is about 980/1000 live births. These are similar to the overall data from other centres in the country.[1,9-11]

Table I: Summary of the Perinatal Data over a Ten-Year (1981-1990) Period at Wesley Guild Hospital, Ilesa[7]

Year	Total Births	Live Births	Still Birth	PNSR	LBR
1981	1846	1736	110	932	940
1982	1979	1892	87	945	956
1983	2053	1993	60	960	971
1984	1835	1786	49	961	973
1985	1350	1302	48	947	964
1986	1021	969	52	934	949
1987	**552**	**505**	**47**	**889**	**915***
1988	**637**	**597**	**40**	**910**	**937***
1989	791	749	42	933	947
1990	861	822	39	940	955
Total	**12925+**	**12351**	**574**	**942**	**956**

+ Only 18% of women who booked in WGH delivered in the same hospital.

* Years with least deliveries and lowest survival rates

The UNICEF estimated NSR in Nigeria is about 960/1000 live births. What are the reasons for our poor LBR and NSR data in Nigeria?

Table II: Perinatal Data for 1996-2000 at WGH, Ilesa (Kuti *et al* 2002)[8]

Year	Total Births	Live Births	Still Birth	PNSR	LBR
1996	970	920	50	926	949
1997	1075	1016	59	925	945
1998	1187	1125	62	923	948
1999	889	847	42	928	952
2000	929	876	53	914	943
Total	**5050**	**4784**	**266**	**923**	**947**

During the period, total number of neonatal deaths was 389; LBW 247 (63.5%), and NBW 142 (36.5%).

CAUSES OF MORBIDITY AND MORTALITY AND PLACES OF DELIVERY

Babies are normally admitted into our neonatal unit (NNU) from the hospital maternity units and outside. Tables III-VII present data on trends in admission rates and common reasons for admission into our NNU and causes of NND in relation to places of delivery. As the number of babies admitted from our labour ward into our NNU decreased, the number of babies admitted from outside units increased (Table III). With increase in the economic hardship, higher proportion of high-risk mothers were being delivered in outside units and the proportion of complicated deliveries in our hospital maternity unit also increased. The result is a worsening NSR (Table IV) even among babies delivered in WGH. Overall survival rate was significantly lower among babies brought from outside (out-born) than among babies admitted from the maternity unit of WGH (inborn)

Table V. The overall survival rate among the 3993 out-born babies was 818/1000 as compared with survival rate of 934/1000 among the 3232 admitted from our labour ward ($p< 0.001$). Survival rates were lowest among babies born at Home and Private Hospitals/Health Clinics (Table V, $p <0.001$).

Table III: Trends in Hospital Delivery and Relative Proportion of Babies Admitted Among Inborn Babies.[7,13]

Year	Live Births	Number Admitted	Admission % LB	NSR/1000 LB
1981	1736	412	23.7	986
1982	1892	420	22.2	987
1983	1993	431	21.6	986
1984	1786	418	23.4	983
1985	1302	324	24.9	982
1986	969	318	32.8	983
1987	**505**	**187**	**37.0**	**964**
1988	**597**	**214**	**35.8**	**970**
1989	749	209	27.9	982
1990	822	279	31.6	983

Our data (Tables VI and VII) and data from other centres in Nigeria[9-21] show that major causes of NND are LBW (including prematurity), infections (including neonatal tetanus [NNT]), neonatal jaundice (NNJ) and birth asphyxia (BA). While congenital anomalies may occupy a relatively more prominent position in the future, it is relatively an uncommon cause of NND in Nigeria. Major factors responsible for low neonatal survival rate among live births in Nigeria are briefly outlined below.

Table IV: Trends in Total Admission, Proportion of Babies Admitted from Outside and Overall Neonatal Survival Rate.[13]

Year	Total Admission	Number Out-born	Out-born % Total	NSR/1000 Admission
1981	733	321	43.8	888
1982	842	422	50.1	889
1983	883	452	51.2	890
1984	748	330	44.1	882
1985	686	362	52.8	885
1986	720	402	55.8	875
1987	600	413	68.8	841
1988	690	476	69.0	845
1989	618	409	66.2	829
1990	685	406	59.3	857

Table V: Survival Rates in Relation to Places of Delivery

Places of Delivery	Total Admission	Number of Deaths	NSR/1000 Admission
Wesley Guild Hospital	3232	212	934
Government Hosp/ Health Cent	1759	285	838
Home	1137	234	794
Private Hospitals/ Clinics	750	156	792
Mission Houses	347	51	853
Total	**7225**	**938**	**870**

Table VI: Diagnosis on Admission into NNU among In-born and Out-born Babies.

Diagnosis	Wesley Guild+ Hospital		From Outside	
	Number	% of total	Number	% of total
Neonatal jaundice	1474	45.6	1579	39.5
Low Birthweight	602	18.6	923	23.1
Neonatal septicaemia	300	9.3	583	14.6
Ophthalmia neonatorum	70	2.2	109	2.7
Gastroenteritis	35	1.1	91	2.3
Neonatal tetanus	4	0.1	226	5.7
Birth asphyxia	459	14.2	156	3.9
Respiratory distress	50	1.5	31	0.8
Congenital malformations	42	1.3	87	2.2
Birth trauma	45	1.4	37	0.9
Others	115	3.6	95	2.4
Total	**3232**	**100.0**	**3993**	**100.0**

+Our overall NSR during the period was 984/1000 live births among hospital deliveries.[7]

Table VII: Major Causes of Neonatal Deaths (NND)

Diagnosis	In-born (%CFR)	Out-born (%CFR)	Total (%CFR)	% of Total Deaths
Low Birthweight	602 (19.1)	932 (31.2)	1525 (26.4)	43.0
Neonatal jaundice	1474 (0.9)	1579 (7.5)	3053 (4.3)	14.0
Neonatal septicaemia	300 (3.0)	583 (18.2)	883 (13.0)	12.3
Neonatal tetanus	**4 (50.0)**	**226 (52.2)**	**230 (52.2)**	**12.8**
Birth asphyxia	459 (13.2)	156 (32.7)	615 (18.2)	11.9
Others	393 (3.1)	526 (8.4)	919 (6.1)	6.0
All low birthweight	**1078 (13.0)**	**1641 (27.1)**	**2719 (21.5)**	**62.4**

LBW + NNJ + NNS + NNT + BA accounted for 87.3% of admissions and 94.0% of neonatal deaths.

CAUSES OF LOW NEONATAL SURVIVAL RATE AMONG LIVE BIRTHS IN NIGERIA

Severe Neonatal Jaundice and Associated Brain Damage (Kernicterus) in Nigeria.

My journey into the business of neonatal jaundice (NNJ) began in 1980 during my preparation for the Final Fellowship Examination of National Postgraduate Medical College of Nigeria.[22] Figure 2 shows a jaundiced baby.

A lot has been written on neonatal jaundice (NNJ) in Nigeria.[22-31] Severe NNJ is a public health problem in Africa. It is the leading

cause of cerebral palsy (cripple), a common cause of deafness and speech delay and neonatal deaths (NND) in Nigeria. This is more so in babies admitted from home, following delivery at home, mission houses and inappropriate places. Sofoluwe and Gans (1960)[23] were the first to draw attention to the problem of NNJ in Nigeria. Since that first report, NNJ has generated a lot of research interests because of its role as major cause of neonatal morbidity and mortality. Major causes of NNJ in various parts of Nigeria are outlined in Table VIII.[22-31]

Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency, blood group ABO incompatibility, bacterial infections and low birthweight (LBW) are the major causes of severe neonatal jaundice (NNJ) in Nigeria. Bacterial infections and G-6-PD deficiency are more common as causes of NNJ in full term babies admitted from home while ABO incompatibility and LBW are more common as causes of NNJ in babies who develop jaundice before discharge from the hospital. The single most important aetiology of severe NNJ and the associated brain damage (kernicterus) is the exposure of the G-6-PD deficient neonates to agents that can cause jaundice.[22,32]

Figure 2: A Three-Day Term Baby with Severe Jaundice

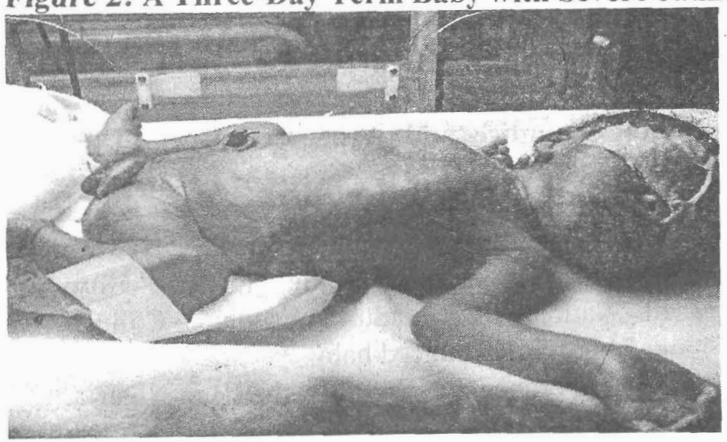


Table VIII: Major Causes of Neonatal Jaundice in Nigeria

Reference Numbers	Major Aetiological Factors in Percentages* G6PD Def.	ABO incomp	Low Birthw.	Infections	Unknown
23 λ	-	-	12.9	42.0	35.9
24 γ	21.0	28.0	-	-	30-35
25 λ	71.0	22.1	-	29.1	-
26 γ	39.2	28.8	16.0	0.8	28.0
27 λ+	58.9	27.1	26.0	59.1	9.1
28 λ+	72.7	35.0	16.7	25.0	-
29 λ	33.0	20.0	40.0	85.0	7.5
30 γ+	46.6	46.1	28.2	3.4	11.7
30 λ+	61.0	33.5	26.1	24.3	5.5
31 γ+*	33.0	29.5	27.7	2.9	-
31 λ+*	54.7	26.1	21.7	35.9	21.1
22 λ	61.5	21.1	14.7	25.7	11.9

*** Multiple factors are involved in many cases; γ = inpatients; λ = outpatients**

+ (+*) = same author(s) or institution.

Exposure to Icterogenic Agents and Severe Neonatal Jaundice in Nigeria.

Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency is the most prevalent clinically significant enzyme deficiency of man. It is the most important aetiological factor associated with severe NNJ and kernicterus in term infants in Nigeria[22,31-33] and other

parts of Africa.[34] In the US, severe NNJ is not common in the term black infants with the same variant of G-6-PD deficiency. Ifekwunigwe and Luzzatto (1966)[35] reported the first case of kernicterus in a G-6-PD deficient Nigerian baby exposed to excess dose of chloroquine and aspirin. Effiong and Laditan (1976)[27] reported that 20 of their 175 patients were exposed to possible oxidant agents. The agents included chloroquine, aspirin, naphthalene balls and vitamin K. Olowe and Ransome-Kuti (1980)[36] showed that menthol-containing dusting powder caused significant increase in serum bilirubin in G-6-PD deficient neonates. A frequent observation that NNJ was more severe and risk of kernicterus higher among infants delivered at home or following early postnatal discharge from the hospital led to the postulate that environmental factors such as infections and exposure to icterogenic agents play important roles in the higher incidence of severe NNJ and kernicterus among Nigerian neonates nursed outside hospitals.[24,37]

For the first time (1980),[2,33] we showed that over 97% of our babies with severe jaundice had been exposed to at least one of naphthalene, menthol-containing dusting powder and balms, aspirin, concoctions (***Agbo iba***) (Table IX). Exposure to these icterogenic agents was associated with severe NNJ only in the G-6-PD deficient neonates. Some of the preparations (***Agbo jedi***) contain ***Kafura*** which is related to naphthalene. Naphthalene had long been associated with severe haemolysis and kernicterus in the G-6-PD deficient infants in many parts of the world.[38-42] Inhalation of naphthalene vapour and dermal exposure to mothballs in diapers and garments (Figure 3) by neonates have led to severe haemolysis of red blood cells, and the G-6-PD neonates are particularly at risk.

Glucose-6-phosphate dehydrogenase deficiency singly or in association with other factors was the most important cause of jaundice in these babies. The relationship between exposure to

Table IX: Exposure to Icterogenic Agents in Neonates with or without Jaundice.[44]

Agents	Group A*	Group B+	X^2	P value
Naphthalene	54 (50.9)	68 (53.1)	0.1	NS
Menthol	57 (53.8)	84 (65.6)	3.4	NS
Herbs	7 (6.6)	32 (25.0)	14.1	< 0.001
Aspirin	3 (2.8)	5 (3.9)	0.2	NS
> 2 Agents	19 (17.9)	54 (42.2)	15.9	<0.001
At least one agent	86 (86.8)	109 (85.2)	0.1	NS

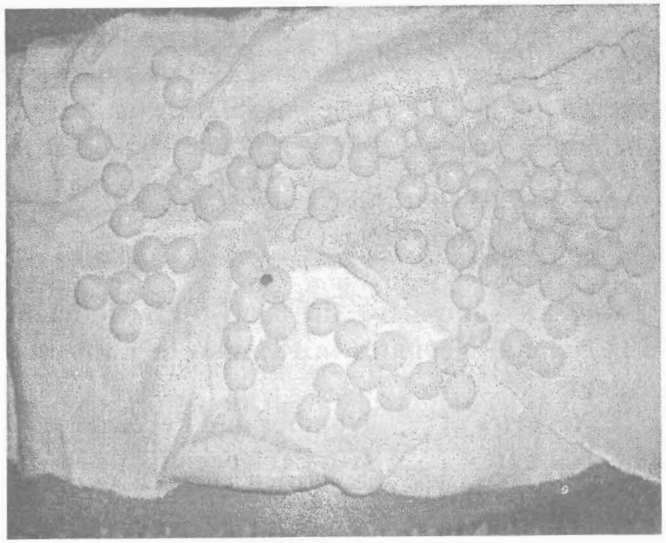
NS = P>0.05 (not statistically significant; * n = 106 with NNJ; + n = 128 without NNJ.

Table X: Comparing the 83 G-6-PD Deficient and 151 G-6-PD Normal Neonates in Relation to Exposure to Icterogenic Agents.[44]

Categories of Neonates	Number and Percentages of Neonates G-6-PD Normal	G-6-PD Deficient
NNJ + exposure	31 (20.5)	61 (73.5)
No NNJ, + exposure	99 (65.6)	10 (12.1)
NNJ, no exposure	9 (6.0)	5 (6.0)
No NNJ, no exposure	12 (7.9)	7 (8.4)
X^2	3.4	12.3
P	> 0.1	< 0.001

icterogenic agents, G-6-PD deficiency and neonatal jaundice was determined in 234 neonates admitted from home. History of exposure to icterogenic agents (Figures 3 and 4) was recorded for the two groups: 106 with jaundice and 128 without jaundice or history of jaundice as the control group. There was a strong association between NNJ and exposure to icterogenic agents in the 83 G-6-PD deficient neonates ($p<0.001$), but there was no association between NNJ and exposure to icterogenic agents in whole group of 234 or 151 G-6-PD normal neonates.[43,44]

Figure 3: Diaper Being Preserved with Mothballs

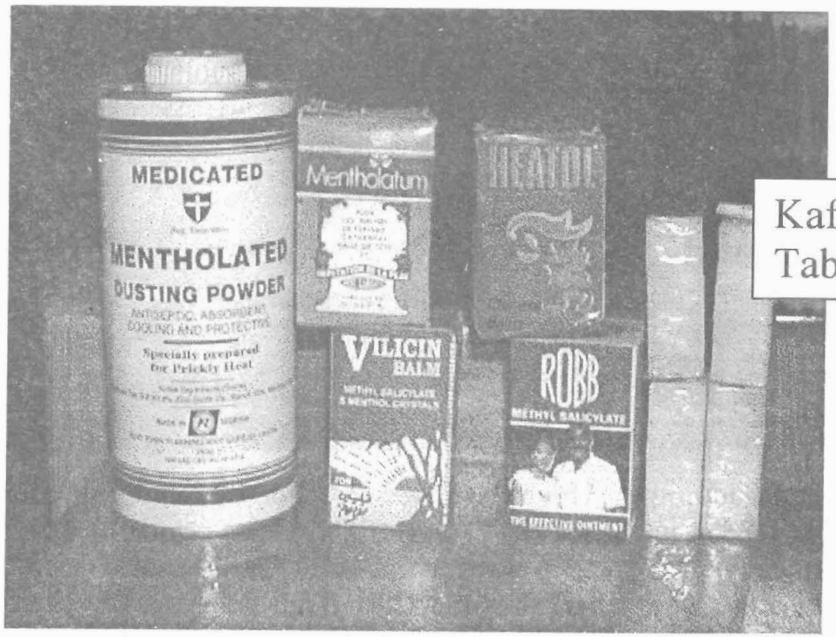


Mothballs

This has also been demonstrated in NNT patients, who normally do not have jaundice or have relatively mild jaundice as compared with neonates with significant jaundice.[45] It was therefore ***concluded*** that there is an association (cooperation) between ***genetically*** determined G-6-PD deficiency and exogenous agents in causing severe NNJ in Nigerian infants. This

is an environmental factor acting on a gene in separating G-6-PD deficient neonates for jaundice very early in neonatal period.

Figure 4: Some Preparations Containing Menthol, Camphor and Salicylates



Kernicterus (Brain Damage Secondary to High Serum Bilirubin)

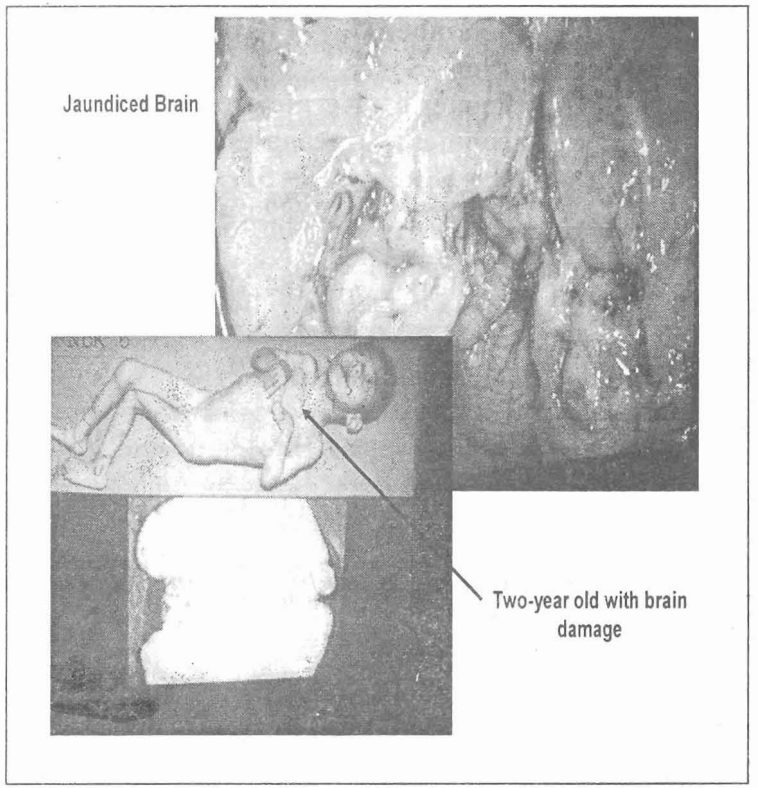
A baby with early signs of kernicterus is shown in Figure 5, while Figure 6 illustrates the irreversibility of the damage. While kernicterus is rare in the developed countries due to availability of preventive measures against Rhesus iso-immunization and effective treatment of NNJ with phototherapy and exchange blood transfusion[46], kernicterus is still a common problem in

Figure 5: A Baby Showing Early Signs of Kernicterus

Nigeria and other African countries.[31-33] For example, our data from Ibadan in early 1980s showed that 47% of the babies had kernicterus.[22,31-33] This was similar to 44% reported from Ghana[34] about the same period. Important aetiological factors of severe NNJ in babies with kernicterus are G-6-PD deficiency, ABO incompatibility, infection, and LBW. By far, G-6-PD was the most important single aetiological factor and the coexistence of G-6-PD deficiency with additional factors increased the risk of kernicterus. For example, only 11% of babies with ABO incompatibility alone had kernicterus and increased to 57% when other factors coexisted with ABO incompatibility, and the main additional factor was G-6-PD deficiency.[31]

According to western literature, kernicterus is unusual in ABO haemolytic disease of the newborn. The overall 39% prevalence of kernicterus in ABO incompatibility in our series was due to

Figure 6: Jaundiced Brain with Irreversible Damage)



the presence of multiple factors and delay in presentation in the hospital. The delay in presentation in hospital suggests a lack of awareness of jaundice by the parents. A major factor causing the delay in recognition of jaundice is probably due to the tradition in our community of keeping newborn babies indoors, in poorly lit rooms till the naming ceremony on the 8th or 9th day of life.

Table XI: Relationship between Exposure to Icterogenic Agents and Kernicterus[33]

Drugs/Chemicals	Number (%) of Patients Exposed		
	Non-kernicteric neonates (N = 58)	Kernicteric neonates (N = 51)	P Value
Naphthalene	30 (51.7)	42 (82.4)	< 0.001
Dusting powder	47 (81.0)	43 (84.3)	NS
Mentholathum	6 (10.3)	10 (19.6)	NS
Salicylates	13 (22.4)	22 43.1)	< 0.05
Herbs	8 (13.8)	18 (35.3)	<0.01

Many babies were exposed to multiple agents, particularly among kernicteric babies.

Determination of Metabolic Products of Mothballs (Naphthalene) in Urine of Jaundiced Babies.

Naphthalene is widely used as a moth-repellant in form of mothballs, flakes and deodorant cakes. Naphthalene itself is not toxic but on dermal absorption and /or inhalation exposure, its major oxidative metabolites, 1- and 2-naphthols have been implicated in neonatal jaundice. There was no report in literature for the determination of these metabolites in exposed neonates. A rapid, specific and sensitive method was developed in this University.[47] This method was applied to the urine samples of jaundiced neonates. Five of the 25 babies with history of exposure to naphthalene and none of the 25 babies without history of exposure to naphthalene had 1-naphthol in their urine. The values of 1-naphthol in the urine samples varied between

0.75 and 11.69μg/ml with mean of 5±5 μg/ml. The overall correlation coefficient (*r*) between bilirubin values and 1-naphthol was 0.1 while it was 1 in the three G-6-PD deficient infants.[48] The small number of 1-naphthol detection may be due to possible time lag between exposure, manifestation and report in the hospital as well as level of exposure.

Neonatal Jaundice among Nigerian Preterm Infants.

Neonatal jaundice is more common in premature than term infants. This is mainly due to the immaturity of the liver in premature babies. Jaundice occurs in about 71% of our preterm babies in controlled hospital environment.[49] In such babies, the most important factor is prematurity. The only factor associated with poor outcome in two of the infants was bacterial infection. Better outcome of NNJ even among Nigerian hospital-born preterm infants is probably due to early diagnosis as result of better surveillance and prompt management of jaundice among in-patients. Since premature babies are more at risk of kernicterus even at relatively low bilirubin levels, jaundice is usually treated more aggressively in them.

Jaundice Due to ABO Incompatibility

In Nigeria and many other African countries, blood group ABO incompatibility between mother and her baby is of four major factors associated severe NNJ.[22-31] These reports however showed that ABO incompatibility was often associated with other causes of jaundice. We have also shown that in jaundiced babies who are ABO incompatible with their mothers, only about 36% of the cases could jaundice be attributable to ABO incompatibility. Such babies can easily be identified by positive direct Coombs' test.[50]

Bacterial Infections (Septicaemia) and Neonatal Jaundice

Neonatal septicaemia has been emphasized as a major cause of severe NNJ in Nigeria.[22-31] Culture proven septicaemia occurs in 0.8-29% of neonates with severe NNJ and clinically suspected in up to 85% of such babies.[29] Septicaemia is predominantly found in association with severe NNJ in babies delivered at home or admitted from home after an early discharge from the hospital. We have shown that G-6-PD does not put babies at greater risk of developing septicaemia.[51,52] Of particular importance are various ways the cord are being treated at home.[51] The ways the umbilicus are treated at home are important factors in the aetiology of NNJ, septicaemia and neonatal tetanus (NNT). These three conditions are mainly found in babies admitted from home. However, when infection is present in the G-6-PD deficient neonates, NNJ tends to be more severe with resultant poorer outcome. Diagnosis of sepsis based on clinical features alone will therefore result in a higher frequency of sepsis being reported among G-6-PD deficient infants because sepsis has clinical features in common with kernicterus.

Neonatal Bacterial Infections:

Neonatal Septicaemia

Neonatal septicaemia remains a significant cause of morbidity and mortality in babies admitted into neonatal units in Nigeria. This has been abundantly demonstrated in many of our publications.[40,55-63] Common bacterial isolates are shown in Table XII. Neonatal septicaemia are of concern to clinicians because of its tendency to progress rapidly, the difficulty in making early diagnosis due to non-specific nature of its clinical presentation in affected neonates and its consequences. Incidence and mortality rates are high. Predisposing factors are low socioeconomic status, lack of antenatal care, maternal pyrexia and congenital malformations. A recent disturbing feature of neonatal septicaemia is high in-vitro resistance to commonly used

Table XII: Bacteria Associated with Septicaemia

Bacterial isolates	Number of Patients (septicaemia)[56]	Number of Patients (PROM)[58]	Number of Patients (NNT)[62]
Gram negatives			
Klebsiella spp	4	4	
Escherichia coli	4	6	4
Proteus spp		2	
Pseudomonas aeruginosa	2		
Citrobacter spp	1		
Haemophilus influenza	1		
Atypical coliform	6		
	18 (58.1%)	12 (66.7%)	4 (50.0%)
Gram positive			
Staphylococcus aureus	8	4	4
Clostridium welchi	2	1	
Streptococcus faecalis	2	1	
Staphylococcus epidermidis	1		
	13 (41.9%)	6 (33.3%)	4 (50.0%)

antibiotics[63], which has also been reported in many centres in Nigeria.

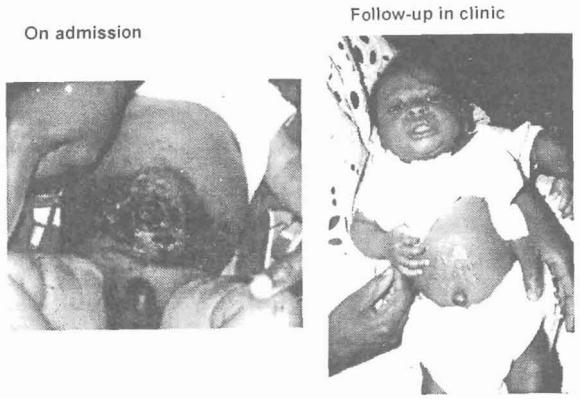
I had an opportunity to compare data between Nigeria and Saudi Arabia – two developing countries. The lower incidence of septicaemia and better outcome in Saudi Arabia were probably

due to better socio-economic conditions, personal and environmental hygiene, and better health services. For example, many of our mothers in Nigeria came from Primary Health Care (PHC) units and home after prolonged rupture of membranes (PROM) followed by multiple vaginal examinations – a practice that increases the risk of infections in PROM. Moreover, the umbilical stumps were being dressed with potentially infected materials, a practice that predisposes babies to NNT and NNJ.

Peri-umbilical Cellulitis.

Peri-umbilical cellulitis (Figure 7) has a very high mortality rate of over 70%. Major features are abdominal distention, peri-umbilical induration (hardening) and inflammation, and foul-smelling discharge. Over 90% of the mothers had antenatal care

Figure 7: Peri-umbilical Cellulitis



but 60% of the babies were delivered at home or in mission houses.[59] There was no recognizable maternal risk factor. Hot fomentation and contaminated materials applied to the cord were

Table XIII: Methods of Umbilical Cord Care Cases of Peri-umbilical Cellulitis.[59]

Methods Used	Number of Cases	Percentage of Total
Cut with:		
Razor blade	20	62.5
A pair of scissors	4	12.5
Knife	2	6.2
Not stated	6	18.8
Cleaned with:		
Hot fomentation	23	71.9
Methylated spirit	17	53.1
Dressed with:		
Dusting powder	18	56.3
Mentholated balm	6	18.8
Gentian violet	2	6.3
Others*	8	25.0

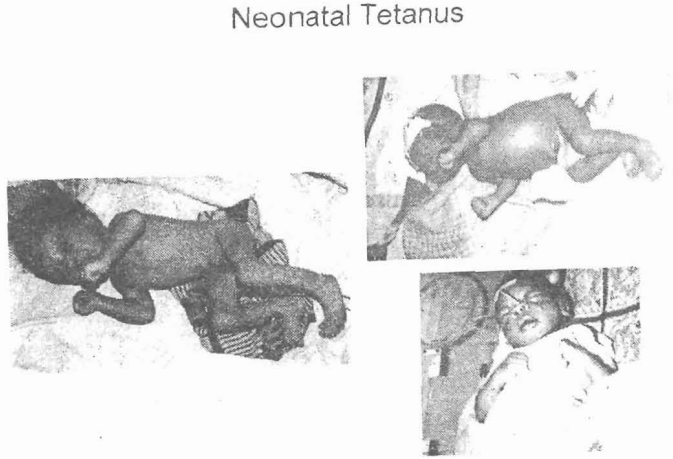
***These include traditional herbs and Vaseline cream.**
Twenty-three (71.9%) of the babies died.

the apparent causative factors (Table XIII). What is required is proper antenatal care, adequate supervision of delivery, better neonatal care and provision of better laboratory facilities to be able to reduce incidence of neonatal septicaemia, improve the management of neonatal infections and reduce morbidity and mortality from bacteria infections.

The Problem of Neonatal Tetanus (NNT) in Nigeria

Tetanus is a highly lethal disease, especially in the newborn. Neonatal tetanus is still a major public health problem in Nigeria.[64] It is among the leading causes of neonatal deaths (NND). Maternal immunization with tetanus toxoid (TT) is effective in preventing NNT. As part of preventive measure against NNT we carried out maternal TT coverage during pregnancy in Ife Central Local Government Area (ICLGA) in 1990.[65]

Figure 8: Three Different Babies with Tetanus (A Common Scene in Our Hospitals)



Out of 896 mothers interviewed, only 668 (74.6%) gave a history of receiving two doses of TT during pregnancy and in only 37 (4.1%) were records of evidence of immunization available at home. About 35% of the babies were delivered at home/mission houses. A follow up study[66] showed that the documentation of maternal immunization status can easily be improved on with the

introduction of home-based maternal records, issued to the pregnant women at booking. This also led to increase in proportion of mothers who received immunization and proportion of babies protected against NNT.

Neonatal tetanus is still very common in Nigeria today (Figure 8). The hospital admission rate for the disease is in fact increasing in many of our hospitals. The management of NNT has remained virtually the same in our institutions. Neonatal intensive care which has improved the outcome in NNT in other parts of the world is not available to our babies. What has changed is the immunization status of mothers, this has increased. A number of babies of immunized women now develop NNT.[67] Immunization against tetanus in the mothers is associated with better outcome.[67] In my two and a half years of sojourn in Saudi Arabia, I did not come across or hear about a single case of NNT. The fact that tetanus occurs in babies of immunized mothers underscores the need for other measures to combat the disease. Increased and sustained efforts must be made to ensure hygienic care during delivery and care of newborn babies after delivery. Such improved maternal and neonatal care had led to dramatic reduction to almost zero in deaths due to NNT in the USA before antenatal immunization of mothers became universally available.[68]

Community Birth Attendants (CBA) or Traditional Birth Attendants (TBA)?

A lot has been written about TBAs, particularly by non-governmental organizations (NGO). The values of their training and retraining in reducing maternal and neonatal mortality have often been over-emphasized. In ICLGA about 27-35% of the deliveries were being conducted by them.[65,69] We carried out a post-training supervision of 19 of them for one-year period after training. Our findings were not very encouraging.[70] The first problem was what to call them after the training, Community

Birth Attendants (CBA) or Traditional Birth Attendants (TBA)! The second problem was what should be their relationship with the government in terms of employment and facilities they needed to carry on their job.

Only 13 (68.4%) of the 19 CBAs had carried out some deliveries one year later and one of them only six deliveries. Six had not carried out any delivery one year after training. They needed continued support and supervision to maintain the standard expected by their training. Their training and follow up supervision could be expensive and their contribution to maternity services was small and sometimes negative. Their activities may have some limited value in the remote villages.

Table XIV: Distribution of 19 CBAs According to Their Major Groups and Location

	Where Location			
Major Groups	**Ile-Ife**	**Villages**	**Total**	**Number Practicing**
Christ Apostolic Church	8	-	8	8
Cherubim and Seraphim	1	1	2	1
The Apostolic Faith	1	-	1	1
Celestial Church	2	-	2	2
Herbalists	-	2	2	2
Others (Selected from the Community	1	3	4	1

Low Birthweight and Neonatal Survival in Nigeria.

Birthweight strongly influences the chances of the newborn to survive and thrive. Not surprisingly, LBW is the most important indirect cause of neonatal mortality and morbidity. Between 40% and 80% of neonatal deaths occur among LBW babies.[1] Low birthweight infants constitute about 16%[70] of births and they account for about 60-70% of NND in Nigeria.[1]

Figure 9: A Preterm in Intensive Care

Looking After A Low Birthweight Infant

Among inborn babies in WGH, LBW was a major cause of neonatal deaths. Most of the LBW infants who died, died within 24-48 hours of admission, from respiratory failure. [12,13] In Nigeria, there is no neonatal unit that is offering intensive care (NICU) to needy neonates. It is possible that many of the LBW

infants delivered outside WGH never reach the hospital as the illustrated case below would suggest.[72]

Saudi Arabia and Nigeria are two developing countries[73] with different levels in their medical facilities. For example, the neonatal unit (NNU) in Qatif Central Hospital (QCH), Saudi Arabia provides neonatal intensive care[74] which includes ventilator support (Figure 9). On other hand, only special baby care services are available to very ill babies at Wesley Guild (WGH) in Nigeria. We compared the outcome of management of LBW infants in the two units. The birthweight-specific and gestational age-specific mortality rates among LBW infants in two units were compared. This enabled us to assess what could be done to further improve the outcome in such infants in Nigeria. It was believed that this information would also be valuable to other developing countries in deciding on whether or not to start neonatal intensive care unit.

The overall survival rates were 845 and 812/1000 at WGH (Nigeria) and QCH (Saudi Arabia) respectively.[75] This was higher in WGH than QCH, though the difference was not statistically significant. However, when gestational age-specific and birthweight-specific survival rates are compared (Tables XV and XVI), the survival rates were significantly higher in the VLBW and infants of GA ≤ 32 weeks at QCH than WCH ($p<0.001$). On the other hand babies with GA and BW above these values survived better in WGH than in QCH.

The most common respiratory problems in the two NNU (RDS in QCH and BA in WGH) may have a common basis. The difference was probably a reflection of the differences in the available facilities in the two units. In QCH neonatal intensive care facilities like ventilators and a standby portable X-ray machine are available. More detailed evaluation of the ill VLBW infants was therefore possible. More of the VLBW infants in QCH therefore survived long enough for more accurate diagnosis to be made. Most of the VLBW babies who died in WGH died

within 24 hours of life and most of these deaths were attributed to BA, based mainly on the assessment of APGAR scores at birth.

Introduction of neonatal intensive care facilities into WGH will have a great impact in improving the very low survival rate among VLBW infants. Though the question of high cost of providing such facilities is real, it is not a justifiable reason to continue to deny thousands of such babies in Nigeria a chance to live.[75] Since good perinatal/neonatal care decreases mortality and morbidity, leading to a net-gain of surviving healthy children, with consequent socio-economic benefits, some measures still need to be undertaken in the developing countries in order to improve the care of these babies.[17] This call has been made by different workers in this country.[9-12,17-20]

Advances in neonatal intensive care has greatly contributed to the survival of such VLBW infants in advanced countries.[75-78] The better survival rate among the VLBW in QCH than WGH was largely due to better facilities at QCH. Since there are no easy ways of accurately predicting neonatal morbidity in extremely LBW infants (BW <1000 grams at birth)[78] all babies should therefore be given a chance. Especially when we know that this may be the only chance for some families to have surviving children of their own.

Such facilities also give those who survive greater chance of achieving their God given potentials. Appropriate utilisation of new technology has contributed to marked reduction of perinatal mortality even among groups of high-risk patients. Recent decline in mortality rates have been attributed to improved neonatal care. Reduction in prematurity takes longer time to achieve.[79] The higher survival rate in WGH was due largely to better survival rate in babies weighing ≥1500 grams or the GA ≥ 33 weeks. The LBW babies in WGH were relatively more mature. On the other hand, lethal congenital anomalies were more common in Saudi Arabia as a result of marriages among close relatives.[79]

Table XV: Comparison of the Main Clinical Data of the Babies.

	Hospitals		
Parameter	**WHG, Ilesa**	**QCH, Qatif**	**P value**
Total	379	373	
Discharge against medical advice	11	5	NS
Male: female ratio	1.02: 1	1: 1.08	NS
Gestational age (weeks)	34.7 (4.3)*	32.3 (3.9)	< 0.001
Birthweight (grams)	1787 (443)*	1596 (454)	< 0.001
Duration of admission (days)	12.3 (10.2)*	23.0 (22.4)	< 0.001

*Mean (standard deviation)
+NS (not statistically significant) = P value >0.05

Table XVI: Comparison of Birthweight-specific Survival Rate (NSR/1000 Admission among the Babies.

	Hospitals		
Birthweight (grams)	**WGH, Ilesa NSR/1000**	**QCH, Qatif NSR/1000**	**P value***
< 999	167 (24)	250 (44)	NS
1000-1499	607 (61)	821 (95)	< 0.001
1500-1999	929 (126)	942 (156)	NS
2000-2499	975 ((157)	863 (73)	< 0.01
< 1499	**482 (85)**	**640 (139)**	**= 0.2**
Total	845 (368)	812 (368)	NS

+NS (not statistically significant) = P value >0.05
Figures in brackets are total for each group

There are better rooms for improvement in Nigeria. We have the personnel which they do not have. What is lacking in Nigeria is the political will. Perhaps a major reason for this is that we ***collectively*** lack the intellectual capacity to appreciate that neglecting the fountain of life of nation i.e. ***neglecting her newborns is a path to national suicide***.

Severe Burns in a Preterm Baby (Figure 9): The Tragedy of Being Poor[72]

Figure 10: Severe Burns in a Preterm Infant.

The first twin had died in the private clinic where they were born. He was referred to the hospital but parents decided to keep the baby at home. He was being kept warm with a kerosene lantern when the tragedy occurred.
This case exemplifies the tragedy of being poor. This was an 11-day old, 1.1 kg baby with severe burns involving the head, the chest and hands. He was the second twin. This was a tragedy that resulted from poverty in the face of inaccessible and unaffordable health care service. **For that reason Damas bemoans "The hospital is there, but without money, you will**

die on its doorstep." Many of similarly ill babies probably die at home due to poverty.

DISCUSSION AND RECOMMENDATIONS

Neonatal mortality is an important component of infant mortality in Nigeria and many developing countries. It accounts for about two thirds of deaths in first year of life.

Major causes of neonatal deaths in Nigeria and other developing countries are asphyxia and infections. These have been eliminated in developed countries so that major causes of neonatal deaths are extreme prematurity and lethal congenital anomalies, which account for 90% of NND in such countries. Neonatal jaundice and low birth weight also play a major role in high neonatal morbidity and mortality in Nigeria. In some rapidly developing economies like Saudi Arabia, the contribution of infections and asphyxia to NND has declined due to improved care as a result of establishment of neonatal intensive care. For example, in a teaching hospital in Saudi Arabia the NSR was 991.1/1000 live births and the main causes of NND were prematurity (33%), congenital malformations (35%), birth asphyxia (13%) and infections (6%).[19]

The estimated annual live births in Nigeria are 5,082,000. With estimated NSR of about 958/1000, about 213,444 die before the age of one month, or about 585/day or 24 per hour. **That means we are losing a newborn every 3 minutes in the life of the nation. Silently! Unsung! This is unparalleled by any disaster, natural or man-made.** About two-thirds of the NND occur during the first week of life, and are largely a consequence of inadequate or inappropriate care during pregnancy, delivery or the critical hours after birth. With the first active breaths after birth, a newborn infant sets into motion a cascade of events that ends with the successful transition from an intrauterine to an extra-uterine life. Most newborn babies have no difficulty in establishing the first effective breaths which trigger this sequence

of changes. If an infant cannot initiate and sustain effective breathing after birth, or if the placenta has malfunctioned before birth, oxygen and carbon dioxide cannot be adequately exchanged resulting in a dangerous drop in the infant's blood oxygen level accompanied by an increase in the carbon dioxide level and accumulation of acid. This combination of events is called **perinatal asphyxia** (literally meaning suffocation). If not quickly corrected, the heart will weaken and the heart rate will dangerously slow down, preventing adequate amount of blood from reaching the organs, especially the brain. The organs, most importantly the brain, may become irreversibly damaged.

We have seen above, that common causes of NND in Nigeria today are preventable or treatable conditions. These include neonatal bacterial infections (including neonatal tetanus), birth asphyxia, neonatal jaundice, and preterm delivery. It was hoped that with so much emphasis being placed on Primary Health Care (PHC) the occurrence of these conditions would be reduced. However, this expectation has not been born out. The reason for this is that many babies are still being born at home or mission houses. Coupled with this is the deterioration of social services.

If the asphyxia episode is mild, infants may show no obvious evidence of permanent injury to the brain and there may be no long-term developmental problems. However, if the asphyxia episode is severe, an infant may die, or survive with life-long neurological disabilities, including cerebral palsy, mental retardation, vision and hearing impairments, and learning disabilities. Such problems may not necessarily show up until the infant is older, necessitating the need for long-term monitoring. Any infant who has experienced perinatal asphyxia should receive special developmental follow-up for the first few years of life.

Studies from around the world on the secular trends in ***survival rate*** for extremely low birthweight (ELBW <1000 gm) and very low birthweight (VLBW <1500gm) babies uniformly indicate

that there have been dramatic increases in their ***survival rates*** in the past 20 years or so. The relative proportion of moderate to severe disability has remained stable during this period of increasing survival potential. Most surviving children with birthweight (BW) less than 1500gm remain free of significant functional disabilities. Given the generally favourable prognosis for even the smallest premature infant, the clinician is probably best advised to give an individual baby the benefit of the doubt. Clearly and dramatically ***too***, neonatal intensive care has been a wonder of the twentieth century medicine. For most LBW infants and their parents, it is worth it. We must therefore find a way to protect high quality health care, *at* whatever the cost, for everyone who needs it, including the smallest premature infant.

Antenatal care has a powerful capacity to prevent fetal damage, including brain damage.

Brain damage can lead to many tragic outcomes. In recent years, important advances have been made in the state-of-the-art. Indeed, we know more than we think we know. However, the state-of-the-practice lags well behind the state-of-the-art (***knowledge***). We have the opportunity as well as the obligation to work diligently toward providing a fair start for ***all our*** children as they move from womb to the close environment of the family, from the classroom to the larger world. Survival, growth and development are simultaneous (not sequential) processes. Actions promoting survival or growth enhance development and vice versa. **The high death toll is only part of the problem. At any one time, hundreds of millions of people – mainly from developing countries are disabled. We need to remember that the primary school graduates of 2009 is now being born and already being made or maimed for future. The 950 live births of today will be dreamers, builders and leaders of this century. With good care of newborn we are aiming not for a cure of 5 years, but a survival of 65 years or more.**

They will be responsible for seeking economic and social justice, for halting devastation of our environment and for building a world in neighbours and nations that can live together in peace. They need intact brain to do this.

Most brain cells are formed before a child reaches three years old. Long before many adults realize what is happening, the brain cells of a new infant proliferate, connections are made and the patterns of a lifetime are established. A child can see and hear from birth and is born with pre-dispositions that prepare it to perceive, learn, and make demands on the external world. An infant communicates from birth by crying and through its facial expressions and movements. The implications are important. Cognitive and social developments are related to the growth of brain cells and development of neural connections. Health and nutritional conditions that damage the brain, even prenatally, when most growth is occurring will influence development. Because these early years are a time of such great change in a young life and of such long-lasting influence, ensuring the rights of the child must begin at the very start of life. Choices made and actions taken on behalf of children during this critical period affect not only how a child develops but how a country progresses. No reasonable plan for human development can wait idly for the 18 years of childhood to pass before taking measures to protect the rights of the child. Nor can it waste the most opportune period for intervening in a child's life, the years from birth to age three. **The time of early childhood should merit the highest-priority attention when responsible governments are making decisions. Yet, tragically both for children and for nations, these are the years that receive the least attention.**

When children fail to get the right start, they never catch up or reach their full potential.

We either provide the money necessary to ensure every child the best possible start in life during the early childhood years **OR** perpetuate the inequities that divide people, compromise their

well being and eventually destroy societies and nations. ...like we are experiencing in Nigeria today. The loving care and nurture children receive in their first years – or lack of these critical experiences – leave lasting imprints on young minds.

We cannot simply stand by and accept the wickedness of a system whose only miraculous achievement has been somehow to concentrate more than four-fifths of the nation's wealth is in the hands of less than a tenth of the nation's population, while millions of newborns and young children around us die unnecessarily. The case of illustrates the well known fact that when poverty engulfs a family, the youngest are most affected and most vulnerable – their rights to survival, growth and development are at risk.[6]

The care of our newborn cannot be regarded as just another task. **No endeavour is worthier of encouragement than this one. To exercise power without humanity is to engender violence of a kind that cannot be fought against with weapons. To break the cycles of poverty, violence and disease, interventions must come early in life, the earlier the better. Poor and unhealthy children make for poor and powerless nations that are then at the mercy of stronger nations. As the lives of young children are short-changed, so the fortunes of countries are lost. Choosing NOT to provide the earliest care for children is the costliest mistake a nation can make.**

All children must enjoy the highest attainable standard of health. ***Adequate neonatal health*** care is critical to survival, growth and development. Children struggle not to die; they struggle to develop mentally, socially, and emotionally. Child survival is part, not the whole picture. The question is asked: what is the quality of life for the child? How can that child realize his/her potential? Newborns that are neglected but manage to survive and then basically ignored by their society until they reach school age, frequently develop serious health and mental deficits that

may persist and which may generally impede their ability to participate productively in their society.

All our vital statistics data are at best estimates. Being registered at birth should be the first step on life's path.

I will like join my voice to those of others in Nigeria[2,11,17-20] to say that we need to start neonatal intensive care units in the country; at least at the regional levels. The availability and accessibility of modern therapies should be combined with a commitment of more resources for advanced intensive care of very LBW infants in order to improve both the short- and long-term outcome. The differences in hospital-specific mortality rates could be accounted for partly by differences in neonatal care resources. This supports the need for equal opportunity of high quality care for all high-risk infants.[19] Improvement in NSR in advanced countries has been attributed to many factors that include: better and more extensive access to ANC, a general improvement in standard of living, better nutrition, better obstetric and neonatal care and greater access to high quality medical care through perinatal regionalization.[79-82] A critically ill, VLBW infant with moderate to severe respiratory distress syndrome or severe birth asphyxia needs neonatal intensive care facilities like ventilator support to survive. Facilitated access to medical and perinatal care and improvement in neonatal care facilities would reverse the current unfavourable neonatal morbidity and mortality among the VLBW infants in Nigeria.

Additional reason why we should invest in such facilities is that we need them to train junior doctors since access to training abroad becomes more and more difficult each day. And those who train abroad do not usually return to their home countries largely because of inadequate facilities to practice what they have learned.

Neonatal jaundice is the commonest neonatal problem all over the world. It is the leading cause of brain damage and learning disability in Nigeria. Brain damage due to NNJ is very rare in

developed countries. Unfortunately NNJ is one of most badly managed condition in the country. The fact that brain damage due to NNJ is not common among neonates who remain in the hospital in first week of life means that kernicterus can be prevented too in Nigeria.

The single and most important aetiological factor is exposure of the G-6-PD deficient neonates to agents that cause jaundice. Screening newborn babies for G-6-PD deficiency and keeping those that are G-6-PD deficient in the hospital in the first weeks of life is an effective way of preventing severe jaundice in such infants. This may not be practicable in Nigeria because of the large number of babies involved: 22% of males and up to 13% of females.[55]

The alternative is health education of parents on:

- Potential dangers of severe NNJ to the newborn brain
- Importance of early recognition and treatment of NNJ
- Danger of exposing newborn babies to the icterogenic agents
- How parents can check for jaundice in their babies in brightly lit room

This should be directed to all people at risk; the entire population and most especially to women of child bearing age. Health education, which emphasizes the adverse effects of drugs to children and pregnant women now features prominently in antenatal and paediatric clinics whereas this is less so in General Outpatient Departments (GOPD). This was probably the reason why exposure to the agents was more common among women attending GOPD than women attending ANC and women who follow their children to children outpatient clinics.

The two most widely employed treatment modalities are Phototherapy (Figure 12) and Exchange Blood Transfusion (Figure 13). Jaundiced babies benefit maximally if they are treated before the onset of brain damage.

If parents seek medical help as soon as they notice jaundice in their babies, the risk associated with delay in presentation will be reduced. The health education is slowly getting to the target population.

Figure 11: A Jaundiced Baby in the Sun

Figure 12A: Two or More Babies Share a Phototherapy Unit in Nigeria.

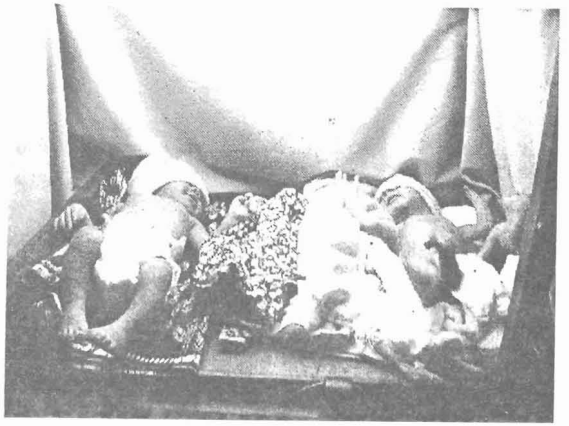


Figure 12B: Two or Phototherapy Units for One Baby in the US

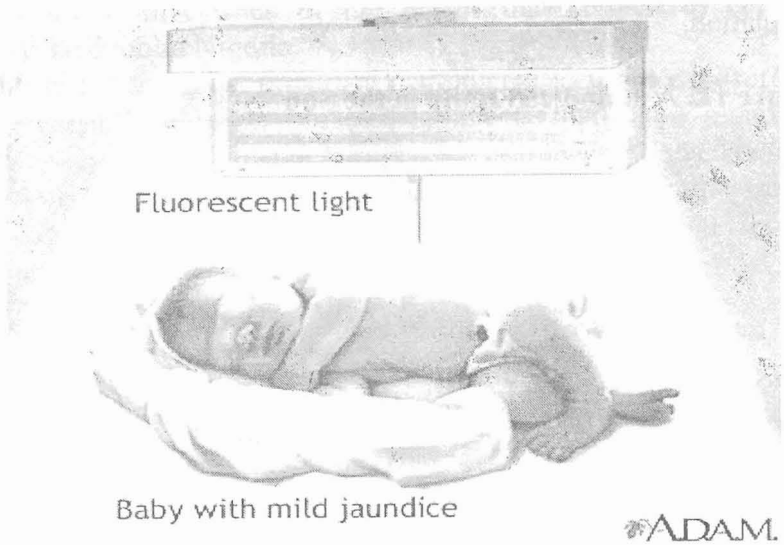
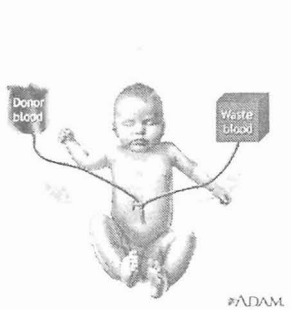
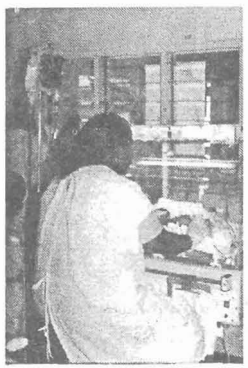


Figure 13: Exchange Blood Transfusion





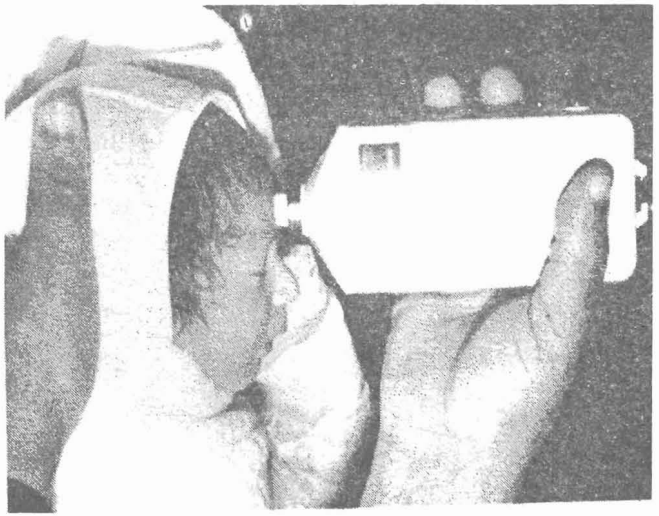
In addition, facilities for monitoring severity of jaundice in the Primary Health Care (PHC) centres and Maternity centres should be provided. There are simple instruments for doing this and they do not involve the use of reagents.

The instruments include:
a. Icterometer, b. Jaundice Meter and c. the Bilicheck.
The icterometer – reading of 3 recommended for at one serum bilirubin determination[54]

Figure 14:
An Icterometer

Jaundice meter – reading of 20 recommended for at one serum bilirubin determination[54]
The readings on them give excellent correlation coefficient with serum bilirubin values. The correlation is better for individual babies making very valuable in monitoring the progress of jaundice in individual babies. These instruments are non-invasive, simple to use and results are immediately available.

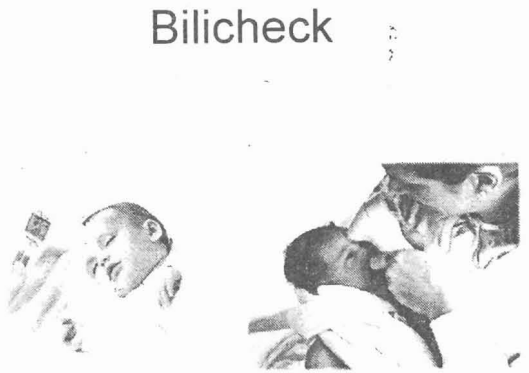
Figure 15: A The Jaundice Meter



Even mothers can use it on their babies as illustrated below. The PHC workers will be empowered to identify babies who are at risk of developing severe jaundice and refer them early for appropriate treatment. Used in tertiary units[54], the number of initial bilirubin determination can be reduced by more than 50% thereby reducing the cost of managing infants with jaundice due to saving on reagents, equipment and personnel time.

Bilicheck is as good as using the Bilirubinometer to determine the serum bilirubin.

Figure 16: The Bilicheck



Educating parents on aseptic way of caring for the umbilical cord with methylated spirit and cotton wool will also reduce the prevalence of septicaemia and NNT. This can be facilitated in the hospital environment. For this reason, pregnant women, mothers and children must be given facilitated access to medical care.

Mr. Vice-Chancellor Sir, permit me to end this lecture by quoting the Lady Nobel Prize Winner in Literature, Gabriela Mistral:

'We are guilty of many errors and many faults,
but our worst crime is abandoning the children, neglecting the fountain of life.
Many of the things we need can wait.
The children cannot.
Right now is the time bones are being formed,
his blood is being made and his senses are being developed.
To him we cannot answer "Tomorrow".

His name is "Today".'

The Future of those who are to make a difference in the life of Nigeria in the 2020s and 2030s has begun as the 950 live births of today. The choice is clear – we can make them or maim them. As the future of the Nigerian 950 live births begins NOW, let everyone of us in this audience resolve to improve the survival and well-being of Nigerian 950 live births for a stronger Nigeria.

Psalm 127: 3-5
Lo, children are a heritage of the Lord: and the fruit of the womb is his reward,
As arrows are in the hand of the a mighty man; so are children of the youth.
Happy is the man that hath his quiver full of them: they shall not be ashamed, but shall speak with the enemy in the gate.

On behalf of the Department of Paediatrics and Child Health, I will like to thank the Obafemi Awolowo University for the opportunity to develop my self over the years and the audience for attention. May the Lord bless you all. Amen

References

1. Perinatal Mortality: A Listing of Available Information. Geneva 1996. WHO/FRH/MSM/96.7
2. Reduction of Maternal Mortality: A Joint WHO/UNFPA/UNICEF/World Bank Statement. Geneva, World Health Organization 1999.
3. Why Invest in Newborn Health? Save the Children 2003. Population Reference Bureau
4. The State of the World's Child 2001. Early Childhood. New York. United Nations Children Fund 2001. www.unicef.org

5. Myers RG. *Towards a Fair Start for Children. Programming for Early Childhood Care and Development in the Developing World.* The Young Child and the Family Environment Project 1990-1996.
6. Myers RG. *The Twelve Who Survive. Strengthening Programmes of Early Childhood Development in the Third World,* 3rd Edition. Michigan, High/Scope Press, 1995.
7. Owa, JA, Osinaike AI and Makinde OO. Trends in Utilisation of Obstetric Care at Wesley Guild Hospital, Ilesa, Nigeria: effect of a depressed economy. *Trop Geogr Med* 1995; 47: 86-88.
8. Kuti O, Orji EO, and Ogunlola. Analysis of Perinatal Mortality in a Nigerian Teaching Hospital. *J Obstet Gynaecol* 2002; 23: 511-513.
9. Okolo AA, Omene JA. Trends in Neonatal Mortality in Benin City, Nigeria. *Int J Gynaecol Obstet* 1985; 191-195
10. Aisien AO, Lawson JO and Okolo A. Two Years Prospective Study of Perinatal Mortality in Jos, Nigeria. *Int J Gynecol Obstet* 2000; 171-173
11. Njokanma OF, Sule-Odu AO and Akesode FA. Perinatal Mortality at the Ogun State University Teaching Hospital, Sagamu, Nigeria. *J Trop Pediatr* 1994; 40: 78-81.
12. Owa JA and Osinaike AI. Neonatal Morbidity and Mortality in Nigeria. *Indian J Pediatr* 1998; 65: 441-449.
13. Kuti O and Owa JA. Gestational Age-specific Mortality Among preterm Singleton Births in a Nigerian Tertiary Institution. *Int J Gynecol Obstet* 2003; 80: 319-320
14. Ibrahim M, Udomah MG and Abdulwahab I. Infant Mortality at Usmanu Danfodiyo University Teaching Hospital, Sokoto. *Nig J Paediatr* 1993; 20: 17-20.
15. Rehan NE and Tafida DS. Low Birthweight in Hausa Infants. *Nig J Paediatr* 1981; 8: 35-39.
16. Adeyokunnu A, Taiwo O and Antia A. U. Childhood Mortality among 22,255 Consecutive Admissions in University College Hospital, Ibadan. *Nig J Paediatr* 1980; 7: 7-15

17. Ibe BC, Ibeziako SN and Azubuike JC. A Study of Admissions into a Newborn-Special Unit. *Nig J Paediatr* 1994; 21: 20-22
18. Njokanma OF and Olanrewaju DM. A Study of Neonatal Deaths at the Ogun State University Teaching Hospital, Sagamu, Nigeria. *J Trop Med Hyg* 1995; 98: 155-160
19. Dawodu A. Neonatology in Developing Countries: Problems, Practices and Prospects. *Ann Trop Paediatr* 1998; 18: S73-S79.
20. Njokanma OF, Egri-Okwaji MTC, Nwokoro A, Orebanjo T and Okeke CE. Birth Asphyxia, Perinatal and Maternal Mortality Associated with Caesarean Section. *Trop J Obstet Gynaecol* 2002; 19: 25-29.
21. Akang EEU, Asinobi AO, Fatunde OJ, Pindiga HU, Okpala JU, Abiola AO and Aghadiuno PU. Childhood Mortality in Ibadan: An Autopsy Study. *Nig J Paediatr* 1992; 19: 30-36.
22. Owa JA. Neonatal Jaundice in Ibadan: Observation on Aetiopathogenesis and Prevention. Dissertation presented to the National Postgraduate College of Nigeria. November 1983.
23. Sofoluwe GO, Gans B. Neonatal Jaundice in Lagos. *West Afr Med J* 1960; 9: 145-148.
24. Ransome-Kuti O. Aspects of International Perinatal Research. In: Kretchmer N, Hasselmeyer EC (editors). *Horisons in Perinatal Research: Implications for Clinical Care.* New York John Liley and Sons, 1974. Pp163-164.
25. Ransome-Kuti O. The Problems of Paediatric Emergencies in Nigeria. *Niger Med J* 1972; 2: 62-70.
26. Effiong CE, Aimakhu VE, Bienzle U, Oyedeji GA, Ikpe. Neonatal Jaundice in Ibadan: Incidence and Aetiological Factors in Babies Born in Hospitals. *J Natl Med Assoc* 1975; 67: 208-212.
27. Effiong CE, Laditan AAO. Neonatal Jaundice in Ibadan: a study of cases seen in the outpatient clinic. *Nig J Paediatr* 1976; 3: 1-10.

28. Coulter JBS, Akpabio MA, Jikeme SON, Kay T. Neonatal Jaundice in Northern Nigeria. *Nig J Paediatr* 1978; 5: 12-20.
29. Werblinska B, Stankiewicz H, Oduloju MO, Atuchuku CM, Fleming AF. Neonatal Jaundice in Zaria, Northern Nigeria. *Nig J Paediatr* 1981; 8: 11-15.
30. Azubuike JC. Neonatal Jaundice in Eastern Nigeria. *East Afr Med J* 1979; 56: 320-324.
31. Owa JA, Taiwo O, Adebiyi JAO, Dogunro SA. Neonatal Jaundice at Wesley Guild Hospital, Ilesa and Ife State Hospital, Ile-Ife. *Nig J Paediatr* 1989; 16: 23-30.
32. Animashaun A. Aetiology of Cerebral Palsy in African Children. *Afr J Med med Sci* 1971; 2:165-171.
33. Owa JA, Dawodu AH, Familusi JB. Kernicterus in Nigerian Infants. *West Afr J Med* 1987; 6: 11-20.
34. Nkrumah FKA, Neequaye J. Neonatal Hyperbilirubinaemia in Ghana. *Ghana Med J*1982; 1: 1-6.
35. Ifekwunigwe AE, Luzzatto L. Kernicterus in Glucose-6-Phosphate Dehydrogenase Deficiency. *Lancet* 1966; 1: 667-668.
36. Olowe SA, Ransome-Kuti O. The Risk of Jaundice in G-6-PD Deficient Babies Exposed to Menthol. *Acta Paediatr Scand* 1980; 69: 341-345.
37. Bienzle U, Effiong CE, Luzzatto L. Erythrocyte Glucose-6-Phosphate Dehydrogenase Deficiency (G6PD type A-) and Neonatal Jaundice. *Acta Paediatr Scand* 1976; 65: 701-703.
38. Vales T, Doxiadis SA, Fassas P. Acute Haemolysis Due to Naphthalene Inhalation. *J Pediatr* 1963; 63: 904-915.
39. Brown RW, Wong HB. Hyperbilirubinaemia and Kernicterus in Glucose-6-phosphate Dehydrogenase Deficiency in Singapore. *Pediatrics* 1968; 41: 1055-1062.
40. Dawson JP, Thayer WW, Desfeorges JB. Acute Haemolytic Anaemia in the Newborn Infant due to Naphthalene Poisoning: Report of Two Cases with Investigation into Mechanism of Disease. *Blood* 1958; 13: 1113-1124.

41. Zueler WW, Apt L. Acute Hemolytic Anemia Due to Naphthalene Poisoning. *J Am Med Ass* 1949; 141: 185-190.
42. Burka ER, Weaver Z, Marks PA. Clinical Spectrum of Haemolytic Anaemia Associated with Glucose-6-phosphate Dehydrogenase Deficiency. *Ann Int Med* 1966; 64: 817-825.
43. Familusi JB, Dawodu AH and Owa JA. Some Epidemiological Aspects on Neonatal Hyperbilirubinaemia. In: Y. Fukumaya, M. Arima, K. Maekawa and K. Yamaguchi (Eds). Child Neurology. *International Congress Series* 579. Tokyo Excerpta Medical (Publishers), 1981; pp 272-280.
44. Owa JA. The Relationship Between Exposure to Icterogenic Agents, Glucose-6-Phosphate Dehydrogenase Deficiency and Neonatal Jaundice in Nigeria. *Acta Paediatr* Scand 1989; 78: 848-852.
45. Owa JA and Olowu AO. Glucose-6-Phosphate Dehydrogenase Deficiency, Neonatal Tetanus and Neonatal Jaundice in Nigeria: Clinical Observation. *Early Child Dev and Care* 1992; 80: 91-95.
46. Harper RG, Sia CG, Kierney CM. Kernicterus 1980: Problems and Practices Viewed from the Perspective of the Practicing Clinician. *Clin Perinatol* 1980; 7: 75-92.
47. Izedonmwen EO, Ogundaini AO, Ogungbamila FO and Owa JA. A New High Performance Liquid Chromatographic Method for Simultaneous Determination of 1- and 2-naphthols in Urine. *Indian J Pharmac Sc* 1991; 53: 240-243.
48. Owa JA, Izedonmwen EO, Ogundaini AO and Ogungbamila FO. Quantitative Analysis of 1-naphthol in Urine of Neonates Exposed to Mothballs: the Value in Infants with Unexplained Anaemia. *Afr J Med and Medical Sci* 1993; 22: 71-76.
49. Owa JA and Dawodu Neonatal Jaundice Among Nigerian Preterm Infants. *East Afr Med J* 1988; 65: 552-556.
50. Owa JA, Durosinmi MA and Alabi EO. Determinants of Severity of Neonatal Hyperbilirubinaemia in ABO Incompatibility in Nigeria. *Tropical Doctor* 1991; 21: 19-22.

51. Dawodu AH, Owa JA and Familusi JB. A Prospective Study of the Role of Bacterial Infections and G-6-Pd Deficiency in Severe Neonatal Jaundice in Nigeria. *Trop Geogr Med* 1984; 36: 127-132.
52. Nasserrallah Z, Abusrair H, Al Dabbous I, Elbashier A and Owa JA. Neonatal Septicaemia and Glucose-6-Phosphate Dehydrogenase Status in Qatif, Saudi Arabia. *Saudi Med J* 1995; 16: 210-212.
53. Owa JA. Severe Neonatal Jaundice and Kernicterus in Nigeria: Causes and Prevention. *Niger Med J* 1995; 29: 3-7.
54. Owa JA, Esimai VC, Olowu WA and Jegede OA. Correlation Between Serum Bilirubin Levels, Jaundice Meter and Icterometer Readings in Nigerian Neonates. *Nig J Paediatr* 1995; 22: 24-30.
55. Luzzatto L and Allan NO. The Relationship Between the Genes of Glucose-6-Phosphate Dehydrogenase and for Haemoglobin S in a Nigerian Population. *Nature* 1968; 219: 1041-1042.
56. Owa JA and Olusanya O. Neonatal Bacteraemia in Wesley Guild Hospital, Ilesa. *Ann Trop Paediatr* 1988; 8: 80-84.
57. Olusanya O, Owa JA and Olusanya IO. The Prevalence of Urinary Tract Infection in High Risk Neonates at Wesley Guild Hospital, Ilesa, Nigeria. *Acta Paediatr Scand* 1989; 78: 94-99.
58. Owa JA, Olusanya IO, Esimai VC and Olusanya O. The Risk of Neonatal Septicaemia after Prolonged Rupture of Membranes in Nigeria. *Trop Geogr Med* 1990; 42: 217-220.
59. Owa JA, Oyelami AO and Adejuyigbe O. Periumbilical Cellulitis in Nigerian Neonates. *Central Afr J Med* 1992; 38: 40-43.
60. Abu Srair H, Owa JA, Elbashier AM and Aman H. Genital Flora, Prolonged Rupture of the Membranes and the Risk of Early Onset Neonatal Septicaemia in Qatif Central Hospital, Kingdom of Saudi Arabia. *Early Child Dev and Care* 1993; 93: 79-85.

61. Elbashier AM, Abu Srair H and Owa JA. Aetiology of Neonatal Septicaemia in Qatif, Saudi Arabia. *Early Child Dev and Care* 1994; 98: 31-38.
62. Oyelami OA, Owa J A and Olusanya IO. Septicaemia Associated with Neonatal Tetanus. *Central Afr J Med* 1995; 41: 171-173
63. Adejuyigbe, EA, Adeodu OO, Ako-Nai KA, Taiwo O, and Owa JA. Septicaemia in High Risk Neonates at a Teaching Hospital in Ile-Ife, Nigeria. *East Afr Med J* 2001; 78: 540-543.
64. Babaniyi OA and Parakoyi BD. Mortality from Neonatal Tetanus in Ilorin: Results from Community-based Survey. *J Trop Pediatr* 1989; 35: 137-138.
65. Owa JA and Makinde OO. Maternal Tetanus Toxoid Coverage During Pregnancy in Ile-Ife. *Intl J Gynecol Obstet* 1992; 39: 123-130.
66. Owa JA, Makinde OO and Awotidebe GA. Maternal Tetanus Toxoid Immunization Records. *Intl J Gynecol Obstet* 1995; 50: 295-296.
67. Owa JA and Makinde OO. Neonatal Tetanus in Babies of Immunized Mothers. *Tropical Doctor* 1990; 20: 156-157.
68. Stanfield JP and Galazka A. Neonatal Tetanus in the World Today. *Bulletin World Health Organization* 1984; 62: 647-669.
69. Low Birth Weight: A Tabulation of Available Information. WHO/MCH/92.2. Geneva 1992.
70. Makinde OO, Owa JA and Awotidebe OA. Community-based Survey of Deliveries in Ife Central Local Government of Nigeria. *Nig Med Practitioner* 1995; 30: 63-65.
71. Makinde OO, Owa JA, and Awotidebe OA. Descriptive Study of Community Birth Attendants: Training and Post Training Evaluation. *The Nigerian Medical Practitioner* 1995; 30: 39-42.
72. Owa JA, Esimai VC and Adejuyigbe EA. Severe Burns in a Nigerian Preterm Peonate: a Tragedy of Poor Access to Medical Care. *Niger Med J* 1994; 28: 127-129.

73. Hendrickse RG. Child Health in Developing Countries: an Overview. In: Hendrickse RG, Barr DGD, Matthews TS, Forfar JO, Sir McGregor IA (Eds.). *Paediatrics in the Tropics*. First Edition. London Blackwell Scientific Publications, 1991: pp 1-14.
74. Abusrair H, Owa JA and Aman HA. Cause-specific Infant Mortality rate in Qatif Area, Eastern Province, Saudi Arabia. *Ann Saudi Med* 1995; 15: 156-158.
75. Ebrahim GJ. Perinatal Priorities in Developing Countries. In: *Paediatrics Practice in Developing Countries*. London, Macmillan Press 1987;194-202.
76. Mclean F and Usher R. Measurements of Live Born Fetal Malnutrition Infants Compared with Similar Gestational and Birth Weight Controls. *Biol Neonate* 1970; 16: 215-221
77. World Health Organization. *Planning Health Survey: Teaching Health Statistics*. Lwanga SK and Cho-Yook Tye (Eds) 1986; 149-160.
78. Balazs R, Jordan T and Lewis PD. Undernutrition and Brain Development. In: Falkner F and Tanner JM (Eds). *Human Growth*. Philadelphia: WB Saunders 1986; 415-473.
79. Forsén T, Eriksson JG, Tuomilehto J, Teramo K, Osmond C and Barker DJP. Mother's Weight in Pregnancy and Coronary Heart Disease in Cohort of Finnish Men. Follow-up Study. *Brit Med J* 1997; 315: 837-40.
80. Neer NR and Alvarez JO. Risk Factors of Fetal Malnutrition in a Group of Guatemalan Mothers and Neonates. *Boletin de la Officina Sanataria Panamericana* 1991; 110: 93-107.
81. Gozal D, Ndombo PK, Ze-minkinde J, Kago I, Tetanye E and Mbede J. Anthropometrics Measurements in a Newborn Population in West Africa. A Reliable and Simple Tool for the Identification of Infants at Risk for Early Postnatal Mortality. *J Pediatr* 1991; 118: 800-5.
82. Owa JA, Al-Dabbous IA and Owoeye AA. Comparison of Morbidity Mortality Rates among Low Birthweight Infants in

Two Neonatal Units from Developing Countries. Proceedings of 7th World IAMANEH Conference, Cape Town, South Africa 2000; 202-207.
83. State of the World's Newborns: A Report from Saving Newborn Lives. London. Save the Children 2001.
84. Dobbing J. Vulnerable Periods in Brain Growth and Somatic Growth. In: Wright EV, Roberts DF, Thomson AM (Eds). *The Biology of Human Fetal Growth.* London: Taylor and Francis, 1976.
85. Hamburg DA. A Developmental Strategy to Prevent Lifelong Damage. Reprinted from *1995 Annual Report of Carnegie Corporation of New York.*