Changing Patterns in Neonatal Care*

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All over the world, today, there has been considerable improvement in neonatal mortality rates. In the United States, the rate was 20/1000 in 1950, and was down to 11.6/1000 in 1975. (1) In the United Kingdom, the British Perinatal Mortality Survey revealed a lowering of mortality rates even in the low weight live births between 1958 and 1969. (2) The trend towards a substantial decline in mortality rates is in the developed countries, and to a lesser extent in the developing ones. The latest W.H.O. statistics, published in 1979. (3) gives the status of different

countries as shown in Figure 1. The mortality rates in India, Fiji, and the Philippines (4), (5) were added to the list. Perinatal mortality rates followed the same trend, that is, it is highest in those countries with very high neonatal mortality rates; thus, Mozambique has a perinatal mortality rate of 80.9/1000, while Denmark has 10.7/1000 births including late foetal deaths from at least 28 weeks of gestation. (3).

FIGURE 1: Neonatal Mortality Rates in Representative Countries of the World.

NEONATAL MORTALITY RATES IN REPRESENTATIVE COUNTRIES OF THE WORLD 1979

PER 1000 LIVE BIRTHS

AFRIC/	ASIA	OCEANIA	AMERICAS	EUROPE	
9 MOZAMBIQUE MADAEASCAR	INDIA PHILIPPINES MALAYSIA SINGAPORE HONGKONG JAPAN	FIJI COOK ISLAND NEW CALEDONIA AMERICAN SAMOA AUSTRALIA NEW ZEALAND	ARGENTINA BARBADOS CHILE DOMINICAN REP GREENLAND MEXICO PANAMA COSTA RICA TRINIDAD & TOBACCO U.S.A	HUNGRY YUGOSLAVIA GREECE POLAND ITALY NORTH IRELAND BULGARIA AUSTRIA ROMANIA SCOTLAND BELGIUM GERMAN DEM. RED. U.K. (ENG. & WALES) FRANCE FINLAND NORWAY SWEDEN SWITZERLAND DENMARK	60
50					50
40	-				40
30	14	Π			30
20					20
10					10

An analysis of the recent improvement in neonatal mortality rates (1950-1975) by the Division of Neonatology of the Albert Einstein College of Medicine (1) in August 1979, has shown that there has been no improvement in the weight distribution of U.S. live births during this 25 year period. In fact, the nonwhite low birth weight rate was 20% higher in 1975 than in 1950, and the very low birthweight babies was 27% higher. Despite this large increase in the delivery of high risk low birthweight babies, neonatal mortality has declined substantially in the U.S. nonwhite population. Although it would seem that one ideal way to reduce neonatal mortality would be to improve on these maternal socio-demographic and biologic conditions that are associated with high rates of low birth weight infants, nevertheless, for whatever reason, there has not been any favourable change in birthweight distribution of live births in the past 25 years.

Furthermore, although many studies have associated maternal education, income, age parity, race, prepregnant weight, and smoking status with neonatal mortality; the bulk of evidence suggests that once one controls birthweight, socio-demographic factors are of little importance. (6) Kessner, (7) found in an analysis of over 100,000 live births that when factors specifying maternal medical risks, social class and antepartum care were added to a regression equation correlating birthweight and infant mortality, the correlation coefficient between birthweight and mortality remained unchanged. Shah, et al (8) reported similar findings.

It is believed, therefore, that the most plausible explanation for the declining mortality rates in the neonatal age group in these countries is improved perinatal care. Hence it is propsed that high quality perinatal medical care is the hope of the developing countries, as well, for improving the survival of infants of low birthweight, and lowering neonatal mortality rates.

The purpose of this paper is to review recent literature on current practices that may have contributed to the present high quality perinatal medical care in most countries, and see how the Fiji situation compares and how this can be improved.

REVIEW OF LITERATURE

1. Intrauterine Diagnosis consists of diagnostic procedures employed for the identification of disease in the foetus in instances in which direct treatment of the foetus is possible, or even in some instances when interruption of the pregnancy is under consideration for definite indications.

The indications for prenatal genetic studies are:

— advanced maternal age

- a previous child with a serious or fatal genetic or congenital defect
- a family history of genetic disorder or mental retardation
- one or both parents are known or suspected carries of a genetic disorder
- exposure during pregnancy to drugs, viruses, xray, etc.
- parents concern about the risk of having a child with a major defect, mental retardation or genetic disorder.

Continuous wave ultrasound (9), (10) has become an accepted part of medical care for mother and child where resources permit it. It is used for automatic simultaneous counting of foetal heart rate and uterine contractions to determine their relationship to each other. Deceleration in foetal heart rate which occurs after a peak of uterine contraction measured by amniotic fluid pressures, and a pattern of deceleration which occurs irregularly both before uterine contraction starts and after it stops correlate with foetal distress, low Apgar score and foetal death. No injury to maternal or foetal tissues from pulsed or continuous ultrasound has been demonstrated to date.

Ultrasonography (9), (10) employs pulsed sound of high resolution and of short wave lengths above the audible limit for man to obtain serial accurately measurable images of the foetus. Foetal gestational age can be predicted between 20 weeks (biparietal diameter-5cm) and 34 weeks (8.7cm) with an accuracy of $\pm 8-10$ days in 95% of instances. It has been used successfully to diagnose cranial malformations, multiple pregnancies after the 16th week, foetal death in utero, hydatidiform mole, and placenta praevia. It has also been used to locate the placenta prior to amniocentesis.

Amniocentesis (9), (10), (11) which has been shown to be of value in many ways in intrauterine diagnosis can be carried out with little discomfort to the mother and little risk when performed by an expert. It is recommended between 14 and 16 weeks and is best preceded by ultrasound to localise the placenta, assess gestational age accurately, and for the recognition of multiple pregnancy. It has been found useful to determine the need for foetal transfusion or timing the delivery of the foetus with erythroblatosis foetalis. It has also been used to determine the sex of the foetus when mother is known to carry a severe x-linked recessive trait such as hemophilia or progressive muscular dystrophy, where the cells are stained for the presence or absence of Barr bodies. The absence of Barr bodies signifies a male foetus and a 50% chance of having the disease in question. The best of chemical indices for foetal maturation are provided by the determination of amniotic fluid creatinine and lecithin which reflect the maturity of foetal kidney and lung respectively. After 36 weeks of gestation, the creatinine concentration is at least 1.8mg/dl of amniotic fluid in 98% of pregnancies with an amniotic fluid/maternal plasma creatinine ratio of 3:1 or greater. Lecithin concentration is usually at least 2mg/dl with an amniotic fluid lecithin/sphingomyelin ratio of at least 2:0.

Golbus, (12) in an analysis of 3000 consecutive amniocentesis for prenatal diagnosis showed the diagnostic accuracy and the safety of the procedure Under inexperienced hands, however, it is not without risks.

The risks of amniocentesis are as follows: (13)

- hemorrhage
- introducing infection
- inflicting a puncture wound or laceration
- causing sudden foetal death
- precipitating premature labor resulting in foetal loss
- maternal sensitization to foetal blood
- maternal hemorrhage
- maternal peritoneal or intrauterine sepsis
- maternal abdominal pain
- maternal death

The foetal loss rate has been reported as follows: (14), (15).

 U.S. (NICHD)
 below 0.5%

 U.K. (MRC)
 1.0-1.5%

 Canada (MRCS)
 below 0.5%

Estriol in Maternal Urine (9), (10) usually rises to 12—50mg/24hrs during the third trimester of pregnancy. Serial values between 4 and 12mg per 24 hours may indicate foetal jeopardy in cases of maternal diabetes, hypertension, or toxaemia.

Values below 4mg have usually been associated with foetal death particularly when they represent a documented fall from the normal range.

It has been found to be low (below 10mg/24 hrs) in: anencephaly

congenital adrenal hypoplasia small for dates infants severe toxaemia antepartum hemorrhage diabetes threatened abortion and extremely low (below 4mg/24hrs) in foetal death.

It is falsely low when certain drugs have been taken like Ampicillin, Meprobomate, corticosteriods, mandelamine, phenolphthalein, and cascara. It is high in congenital adrenal hyperplasia.

Alphafetoproteins (10), (16), (17) are synthesized in foetal liver and yolk sac reaching the amniotic fluid by some unknown route. The upper limit of normal values are:

10-18 weeks — 50ug/ml 19-26 weeks — 25ug/ml over 26 weeks — 10ug/ml

Elevated levels in maternal serum has served as clues to indicate the presence of foetal neural defects, as in spina bifida and anencephaly. However, it is also high in —

placental injury Turner's syndrome congenital nephrosis omphalocoele duodenal atresia multiple pregnancy foetal death

The American Academy of Pediatrics committee on Fetus and the Newborn to the present exerts no great pressure to make this screening test routine.

2. Intravenous Alimentation (9), (18) is done by the introduction of a silastic catheter into the internal or external jugular vein so that the tip is proximal to the right atrium. It is secured in place and tunneled to exit in the posterior occipital region to avoid dislodgement and minimize the hazard of infection. A millipore filter is interposed to minimize contamination from micro-organisms or particulate matter. The catheter is attached to an infusion pump for the accurate control of the flow of the hypertonic acid infusate which contains a protein equivalent (beef fibrin and casein hydrolysate and synthetic amino acids) of 2.5gm/dl and hypertonic glucose in the range of 10-25gm/dl in addition to appropriate quantities of electrolytes and vitamins.

This has been found to be most useful in the feeding of neonates where some surgical procedure has limited the absorptive surface of the GI tract.

3. Phototherapy (19), (20) has been used widely for the treatment of severe neonatal jaundice since the landmark controlled trial of Lucey, et al despite a lack of understanding concerning its mechanism of action or potential toxicity.

New information indicates that the configurational photoisomerization of bilirubin at the 5 and 15 carbon bridges is the major mechanism of bilirubin photocatabolism in vivo, and that singlet oxygen plays only a minor role. It has also been stated that intermittent as compared to continuous therapy might cause greater damage to skin DNA (carcinogenesis). Human KB cells in culture, on illumination with phototherapy lamp exhibit a decrease in the size of their isolated DNA which is reparable during a dark period. DNA repair enzymes are error prone, and such error may play a role in carcinogenesis.

To date, however the question of potential long term side effects have not been evaluated by long-term follow-up and there is no anecdotal evidence of such sequelae as carcinogenesis or effects on sexual maturation.

- 4. Prophylaxis and Treatment of GC Infection (21)

 The prevalence of asymptomatic genital gonorrheal infection in pregnant women and the occurrence of GC ophthalmia in untreated infants born to infected women indicate the need for continued prophylaxis for all newborn infants. The AAP Committees of Drugs, on the Fetus and the Newborn, and on Infectious Diseases recommend the use of opthalmic ointment or drops containing tetracylicline or erythromycin or a 1% silver nitrate solution as effective and acceptable.
- 5. Effect of position on mean transcutanceous PO2 (22) — The search for any recent or not so new practices that may improve the care of the neonate especially without any additional cost led to the choice of this article to be included in this review. A study at the department of pediatrics Case Western Reserve University School of Medicine and the Rainbow Babies and Children's Hospital, Cleveland, showed that when infants were prone, Pa02 rose by a mean of 7.4m Hg (P greater than 0.001) an increase of 15% for the whole group and a 25% increase in Pa02 for 5 infants with residual cardiopulmonary disease. This improved oxygenation in the prone position appears to be the result of enhanced ventilation-perfusion ratios and not merely secondary to an alternation in sleep state with positioning of the infant. In the management of small preterm infants, the maintenance of an optimal Pa02 is a critical aspect of their care. The 25% increases in arterial oxygen observed in this study by merely placing such infants in the prone position may significantly reduce the morbidity of these low birthweight infants.
- 6. On drug therapy in the neonate (23), (24), two

articles are included in this review for what they are worth. First, on the use of aminoglycosides which are used to a large extent in the nursery for coverage of gram negative organisms, it is good to note that a 4 year follow-up in 1970 on the long term effects of gentamycin and kanamycin revealed that no sensorineural hearing loss or vestibular dysfunction was identified. Audiometric, vestibular and psychometric evaluations were performed in this study on both treated neonates and matched controls.

The second article supports the favourable effect of aminophylline on the respiratory center activity and metabolic rate in premature infants with idiopathic apnea. At a dose of 2mg/Kg IV every 6 hours for 48 hours the incidence of apnea decreased from 29.7 to 4.4 episode per 24 hour period in the neonates observed. Oxygen consumption increased 20% after 24 hours. The action of aminophylline seems to be to increase respiratory center output by lowering the threshold of the respiratory center to CO2. thus increasing ventilation.

- 7. On idiopathic respiratory distress syndrome, (25), (26) the first demonstration in human of the use of surfactant by Fujiwara with consistent and dramatic success after a single instillation by endotracheal tube is reported. The problem of a widely patent persisting ductus arterious becoming symptomatic between 21 and 96 hours of life was explained on the basis of the prompt improvement in ventilation leading to a fill in pulmonary vascular resistance thus leading to a significant left to right shunting.
- 8. Neonatal signs as predictors of cerebral passy (27) become important as the survival rate of low birthweight and very low birthweight infants improves. This article reviewed 40,000 infants prospectively for the purpose of identifying those at markedly heightened risk of motor handicap or early death.

Tenfold to 33-fold increases in risk of cerebral palsy were observed in surviving children with any of the following:

- birthweight below 2000gms.
- head circumference larger than 3 standard deviations above or below the mean.
- 5 minutes Apgar score of 3 or less
- diminished actively or diminished cry lasting for more than 1 day thermal instability
- need for gavage feeding
- hypo- or hypertonia
- single or multiple apneic episodes
- hematocrit less than 40%
- neonatal seizures

Apgar score below 3 at 10 minutes or later
 The last two factors supposedly carried over 50% risk.

9. Protective Factors in Human Milk (28) — This review would not be complete without including an article which adds to our knowledge about human milk, since this is something we are continuously recommending as part of good medical neonatal

care. There is no question that a significantly lower incidence of respiratory and diarrheal diseases has been demonstrated in breast fed infants as compared to those fed cow's milk formulas.

Table 1. shows the multiple components present in breat milk that protect The neonate from the pathogenic microorganisms in his environment.

TABLE 1. PROTECTIVE FACTORS IN HUMAN MILK (28)

ELEMENT	FUNCTION	ANTIMICROBIAL ACTION
Cells Macrophages (90% of leukocytes)	Phagocytosis Production of lysozyme Complement components (C3-C4) Lactoferrin Transport of Secretary IgA	Phagocytes bacteria, virus Cleaves bacterial cell wall Opsonizing properties when activated via C3 preactivator Iron binding protein Deprives certain bacteria of iron Protection of mucus membrances from many different virus and bacteria Activites alternate complement pathways
B lymphocytes (20-50% of lymph)	Production of	Diminishes exposure to food antigens
T lymphocytes	Potential augmentation of local immune response Stimulus to cellular immune system	
Bifidus factor	Promotes the growth of Lactobacillus bifidus which maintains an acid milieu within the GI tract.	Acid environment inhibits the growth of pathogenic bacteria milieu.
Lactoperoxidase		Destroys cell wall of bacteria
Anti-staphylococcus factor		Inhibits growth of staphylococcus.

10. Recommendations of the Committee on Fetus and Newborn of the American Academy of Pediatrics (29) Some recommendations of this committee are presented here to show what they are practising in neonatal units in that country.

A. Neonatal Areas

- Observation or transition
 30 square feet per infant
- Intensive care
 60 square feet per infant
- Normal

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20 square feet per infant or rooming-in

B. Nursing Staff

1 nursing staff per 4 infants

1:1 or 1:2 ratio per shift

1 nursing staff per 8 infants

- C. Candidates for admission to Intensive Care Area are:
- infants with respiratory insufficiency
- infants with in-dwelling umbilical catheters

- infants weighing 1500gms or less
- There infants to be transferred to recovery area when condition stabilizes
 - with significant anomalies
 - with frequent episodes of apnea
 - with sepsis, meningitis, etc.
 - who require exchange transfusion
 - pre- and post-operative
- D. Medical Staff require sleeping quarters close to nursery complex for physician on call.

Neonatologist (30) The need for these specialists in the United States is based on a rough figure of 132,000 based on 3,300,000 births per year out of which 40 per 1000 neonates will require intensive care.

Thus they recommend that the training of subspecialist neonatologists should continue. They estimated that 1005 sub-board neonatologists are needed and that in 1980, there is a shortfall of 275.

E. Transfer Facilities - Ideal is an ambulance to permit experienced personnel to stand or sit by the infant with the infant connected to appropriate cardiac and respiratory monitoring equipment, and an air ambulance as needed.

FIJI STATUS

How do we stand in Fiji in regard the neonatal mortality and perinatal medical care? The following statements and figures are based only on Suva statistics and therefore does not represent national statistics.

- 1. Low birthweight We have a very high incidence of low birthweight. 1975 - 747 of 4299 deliveries weight 2500gms and below 1979 - 551 of 4413, a rate of 12.5% of total live births. The incidence in the U.S. and U.K. are shown below. Britain 7-8% U.S.A. 6-9%.
- 2. Maternal Anaemia The incidence of maternal anaemia is high. 15-20% of women delivered each year have haemoglobins below 11.5Gms.
- 3. Rhesus Factor Contrary to previous belief, the incidence of Rn negative blood is 8.4% in half the population delivering in this hospital.
- 4. Mortality Rates of Low Birthweight Neonates In 1979, the mortality rates according to weight groups were as follows:

Weight	No. Delivered	Deaths		
Up to 1Kg.	22	18		
Over 1 — 1.5Kg	65	35		
Over $1.5 - 2.0$ Kg.	102	23		
Over 2.0 — 2.5Kg	362	10		
Total	551	86		

This represented a rate of 30.6/1000 live low weight births.

These mortality rates compared favorably with Lubchenko's (31) mortality risks of low brithweight infants as shown below. It should be noted, however, that these are percent mortality risks of 10 years ago.

1955-1969

Percent Mortality Risks of LBW Infants, Lubchenko (U. of Colorado) (

PERCENT MORTALITY RISK

Weight												
2500	29	24	20	17	14	12	10	8	7	6	3	5
2000	44	36	28	24	21	18	15	13	11			
1750	58	49	40	35	30	25	23	21	19			
1500	72	63	57	48	42	37	33	30	27	22		
1250	85	76	68	61	55	50	45	40	36	33	29	26
1000	94	88	82	76	70	64	58	53	49	45	42	
750	99	96	90	85	80	75	69	65				
500	100 100 100 100 100											
Meeks												

Gestation 26 27 28 29 30 31 32 33 34 35 36 37

SUMMARY AND CONCLUSIONS

Although our perinatal and neonatal mortality rates are not the worst, they are not anywhere close to the best either, and therefore, stand to be improved.

The recent transfer of the newborn nursery to the new hospital building is a big step in this direction. The staffing, however, is very far below recommended figure. There is no neonatologist, but the paediatric staff of the hospital are all doing their best.

With regard to transfer facilities, the best that we can recommend considering the difficulties in transport and the physical nature of the islands, is for the doctors in the periphery to be aware of and refer their high risk pregnancies early. The best transport for the fetus under the present circumstances is in it mother's womb!

Realizing that the improvement in survival rates for the low birthweight neonates may mean a question of neurodevelopmental handicaps becoming a problem among the survivors, an attempt in prevention has been initiated. There is enough evidence in medical literature of the considerably high incidence of neurological sequelae or neurodevelopmental handicaps in this group of infants. (27). These infants are therefore kept under close surveillance at our neonatal clinics for the purpose of determining:

- the incidence of neurological sequelae or handicaps
- the earliest these can be detected
- whether early intervention can alter the course of these handicaps.

For this purpose, the low birthweight neonates are followed up at monthly or two-weekly intervals by the paediatrician and a physiotherapist, assessing their neuromuscular status and development using a combination of recommended procedures. Infants showing any neurological abnormality are seen more often and mothers are instructed on physical exercises to perform at home.

closely for early signs of anaemia and treated accordingly.

In conclusion one might quote that "the first minutes of life may determine the quality of that life. Prompt, organized and skilled response to emergencies in this period requires not only written policies but qualified personnel in the proper techniques 24 hours a day."

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The newborns of anaemic mothers are also followed

COVER PICTURE STORY

FOR THE FIRST TIME, GUEST SPEAKERS FROM U.S.A. PARTICIPATED IN THE CONFERENCE

Professor Kurt Benirschke

Dr. Benirschke is a pathologist and educator. He was born in Glueckstadt, Germany on May 26, 1924. He attended various universities in Germany from 1942-1948, receiving his MD from the University of Hamburg (Germany).

Dr. Benirschke is married and has three children.

Dr. Benirschke completed his residency in Teaneck, New Jersey, various Boston Hospitals during period 1950-1953. He became pathologist at Boston Lying-In Hospital in 1955 and a teaching fellow associate at Harvard Medical School from 1954-1960.

Dr. Benirschke became a Professor of Pathology and Chairman of the Department of Pathology at the Darmouth New Hampshire Medical School in 1960 and served in that capacity for ten years.

Dr. Benirschke presently is (since 1970) Professor of Reproductive Biology and Pathology, University of California at San Diego, Chairman, School of Medicine, La Jolla, California (since 1976), Director, Research at San Diego Zoo (since 1975).

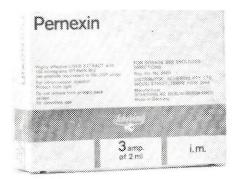
Dr. Benirschke also served as a consultant to the National Institute of Health from 1957-1970.

He is a member of the Ameircan Medical Association, International Academy of Pathology, American College of Pathology, New York Academy of Science, and Animal Care Panel.

He resides in La Jolla, California.

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REFERENCES

- Lee KS, Paneth N, Gartner LM, et al: Neonatal mortality: and analysis of the recent improvement in the United States, Am. J. Public Health, Vol. 70, No. 1, 1980.
- Forfar Jo & Arneil GC: Text Book of Paediatrics 2nd Ed. Churchill Livingston, Edinburgh, London, New York, 1978.
- 3. WHO Statistics Annual, 1979 Geneue, 1979.
- Cresse VM & Hill EE: The preterm Baby; 8th Ed. Churchill Livingstone, Edinburgh London & New York, 1975.
- 5. Philippine Health Statistics, 1974 Dept. of Health
- Lee K, Tseng PI, Eidelman AI, et al: Determinants of neonatal mortality, Am, J Dis Child 130: 842-5, 1976.
- Kessner DM, Singer J, Kalk CE, et al: Infant death: an analysis by maternal risk and health care. Washington DC, Institute of Medicine, 1978.
- Shah FK, Abbey H: Effects of some factors on neonatal and postneonatal mortality. Analysis by a binary variable multiple regression method. Milbank Mem Fund Q 49:33-57, 1971.
- Vaughan VC McKay JR: Nelson Textbook of Pediatrics, Tenth Ed. wB Saunders Co., Philadelphia, London, Toronto.
- Milunsky A: Prenatal Genetic Diagnosis., Ped in Review AAP, 1:9, March 1980.
- NICHD National Registry for Amniocentesis Study Group: Midtrimester amniocentesis of prenatal diagnosis: Safety and accuracy. JAMA 236-1471, 1976.
- Golbus MS, Loughman WD, et al: Prenatal 1 genetic diagnosis in 3000 amniocentesis, N. Eng. J Med 300-157, 1979.
- Medical Research Council Working Party on Amniocentesis: An assessment of the hazards of amniocentesis, Br J. Obstet Gynacol 85 (suppl 2) 1978.
- Lowe CU, Alexander D, Dryla C, et al: The safety and accuracy of midtrimester amniocentesis, US Dept of Health, Education and Welfare, Publ. No. (NIH) 78-190.
- Canadian Medical Research Council Report Number 5: Diagnosis of genetic disease by amniocentesis during the second trimester of pregnancy. Ottawa, Canada, 1977.
- 16. UK Collaborative study on alpha-fetoprotein in relation to neural defects: Maternal serum alphafetoprotein measurements in antenatal screening for anecephaly and spina bifida in early preg-

- nany. Lancet 1:1324, 1977.
- Ferguson-Smith MA, Rawlinson MA, May HM, et al: Avoidance of anencephalic and spina bifida births by maternal serum-alpha-fetoprotein screening, Lancet 1:1330, 1978.
- 18. Klaus mH & Fanaroff AA: Care of the high-risk neonate; 1st Ed, WB Saunders Company, Philadelphia, England, Canada.
- Lucey JF, Ferreiro M, HewittJ: Prevention of hyperbilirubinemia of prematurity by phototherapy, Pediatrics 41:1047, 1968.
 Cohen AN & Ostrow JD:
- 20. New concepts in phototherapy, Pediatrics 65:4:740, Apr. 1980.
- American Academy of Pediatrics Committee on Drugs, Committee on Fetus and Newborn, Committee on Infectious Diseases: Prophylaxis and treatment of neonatal gonococcal infections, Pediatrics 65:5:1047, May 1980.
- Martins RJ, Rerrell N, Rubin D, et al: Effect of spine and prone positions on arterial oxygen tension in the preterm infant, Pediatrics 63:4:528 Apr. 1979.
- 24. Gerhardt T, McCarthy J, & Bancalari/E: Effect of aminophylline on respiratory center activity and metabolic rate in premature infants with idiopathici aphea, Pediatrics 63:4:537, Apr 1969.
- Finitzo-Hicher T, McCracken GH, Roeser R, et al: Ototoxicity in neonates treated with gentamycin and kanamycin: Results of a 4 year controlled follow-up study; Pediatrics 63:3:443, March 1979.
- 25. Avery ME: On replacing the surfactant; pediatrics 65:6:1176, June 1980.
- 26. Fujiwara T, Maeta H, Chida S, et al: Artificial surfactant therapy in hyaline membrane disease: Lancet 1:55, 1980.
- Nelson KB & Ellenberg JH: Neonatal signs as predictors of cerebral palsy; Pediatrics 64:2:225, Aug. 1979.
- 28. Klaus MH & Diaz-Roselle J: Breast-feeding 1980. Pediatrics in Review 1:9:289, March 1980.
- 29. Recommendations of the Committee on Fetus and Newborn, American Academy of Pediatrics, 3rd Ed. 1977.
- 30. Committee on Fetus and Newborn Section of Perinatal Pediatrics: Estimates of need and recommendations for personnel in neonatal pediatrics; Pediatrics 65:4:850, Apr, 1980.