CEREBRAL PALSY IN SINGAPORE CHILDREN

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SYNOPSIS

Kernicterus was the major cause of cerebral palsy in 1965. The aetiology of kernicterus in Singapore differs from western countries in that glucose-six-phosphate dehydrogenase deficiency accounted for 43% of the children with kernicterus as shown by Wong (1). The other causes include liver immaturity, ABO incompatibility, sepsis, prematurity and Rhesus incompatibility. In 1968 Tan studied 577 cerebal palsied children and found that 31.5% were of the athetoid variety (2). The high incidence of athetosis compared to western countries is attributed to kernicterus in 56% in the neonatal period. Glucose-six-phosphate dehydrogenase is necessary in the hexose-mono-phosphate shunt of glycolysis in the erythrocyte. Environmental factors responsible for triggering off haemolysis are herbs taken traditionally by nursing mothers and also exposure to napthalene used for storing the newborn babies clothes.

Preventive measures directed towards kernicterus are the screening of cord blood for glucose-six-phosphate dehydrogenase at all government hospitals. All trigger factors, like herbs, are prevented from reaching the newborn baby and the baby is kept in the nursery from 21 days. In the event of neonatal jaundice occurring, a timely exchange transfusion is done. During the past 18 years, the number of kernicteric infants has been reduced to negligible numbers.

This paper also describes the clinical features, the developmental milestones, the incidence of deafness, speech development and the hand skills of 92 kernicteric children.

Other preventive measures directed towards tuberculous meningitis and Japanese B encephalitis have reduced the number of cerebal palsy and mental subnormality to very few cases annually. Immunication of young adolescent females against rubella will reduce the incidence of mental subnormality and cerebral palsy. While prenatal, natal and post natal causes have reduced the incidence of mental subnormality from 20% in 1963 to 11% in 1977 and 5% in 1978, we will still be left with the survivors of low birth weight babies and small premature babies who, in future, will need long-term assessment.

INTRODUCTION

Singapore with a population of 2.1 million is an example of a South-East Asian country where the problem of cerebal palsy and mental subnormality are coming to the forefront. Prevention of cerebral palsy has taken a very important part in Singapore and thus is linked with the general health measures of the country as a whole. In order to prevent cerebal palsy one must study the causes of cerebral palsy in Singapore

KERNICTERUS IN SINGAPORE CHILDREN

The aetiology of kernicterus in Singapore differs from western countries in that glucose-six-phosphate dehydrogenase deficiency accounted for 43% of the children with kernicterus (1). The other causes included liver immaturity, ABO incompatibility, sepsis, prematurity and Rhesus incompatibility.

Action of G6PD in the Hexose-monophosphate shunt of glycolysis in erythrocyte

Glucose-6-phosphate		6-phosphogluconate
N.A.D.P.H.	G.S.H. Reductase	N.A.D.P.
G.S.S.G.	G.S.H. Peroxidase	G.SH.
H_2O		H_2O_2

N.A.D.P. = Nicotinamide Adenine Dinucleotide Phosphate

G.S.H. = Reduced Glutathionine.

G.S.S.G. = Oxidised Glutathione

Infants with neonatal jaundice due to glucose-six-phosphate dehydrogenase deficiency produce jaundice on the 5th day of neonatal life leading to kernicterus. Glucose-six-phosphate dehychogenase is an intraerythrocytic enzyme necessary for the integrity of the red blood cell and is necessary in the hexose-monophosphate shunt of glycolysis in the erythrocyte, as shown in the diagram. Haemolysis is triggered off by an oxidant drug which would tip the balance in the economy of the hexosemonophosphate shunt. It is the accumulation of hydrogen peroxide together with the presence of superoxide radicals as a result of glucose-six-phosphate deficiency in the presence of an oxidant drug which initiates haemolysis. Wong (1) showed that environmental factors responsible for triggering of haemolysis are herbs taken traditionally by pregnant and nursing mothers and secondly, exposure to naphthalene balls in the newborn. An extract of dried roots called Coptis Chinensis is given to infants after boiling it with water and honey. Wong (1) showed that infants with glucose-six-phosphate dehydrogenase deficiency when given Coptis Chinensis had a significantly higher incidence of severe jaundice than those not given it. The newborn baby in Singapore is often exposed to naphthalene because of the local custom of putting mothballs in stored clothes.

Each mothball contains five grams of naphthalene which is absorbed via the skin or inhaled and taken to the liver for conjugation. α -naphthol produces haemolysis of normal erythrocytes and G6PD deficient infants develop severe jaundice than those not exposed to it as shown by Wong (1). Ninety percent of Singapore babies are delivered at major government maternity hospitals and all newborns have their blood screened for glucose-six-phosphate dehydrogenase deficiency (Figure 1 and 2). If deficient, all siblings, parents and grandparents are screened for this enzyme as seen in Figure 3 where a proband child with glucose-six-phosphate dehydrogenase deficiency producing kernicterus was traced to four generations. A quarter million of the population and a total of 4540 families are known to be enzyme deficient. The incidence of glucose-six-phosphate dehychogenase deficiency is 1.7% among the Chinese, 1.8% among the Malays and 0.34% among the Indians. The overall incidence among all races combined is 1.7%. The deficient babies are kept in the maternity unit for 3 weeks to prevent exposure to trigger factors. In the event of neonatal jaundice, a timely exchange transfusion is done to prevent mental subnormality and cerebral palsy.

With the help of the Ministry of Health, an intensive medical and lay public education campaign was organised to draw attention to the dangers of herbs and naphthalene. As a result of these measures, not only has there been a fall in the mortality (Figure 4) but also the morbidity, namely cerebral palsy and mental subnormality.

SCREENING FOR G-6-P-D ENZYME IN SINGAPORE NEWBORNS OVER A 15 YEAR PERIOD

CHINESE

YEAR	No TESTED	No G6PD Def	% G6PD Def.
1965	29,013	465	1·6%
1966	28,425	482	1·7%
1967	27,655	456	1·7%
1968	26,064	405	1·6%
1969	24,263	427	1·8%
1970	22,961	329	1·5%
1971	21,665	323	1·5%
1972	22,750	404	1·8%
1973	22,883	434	1·9%
1974	21,651	356	1·6%
1975	19,328	249	1·8%
1976	21,401	413	1·9%
1977	18,356	386	2·1%
1978	18,678	335	1·8%
1979	18,928	345	1·8%
TOTAL	344,021	5,809	1·7%

MALAY

YEAR	No TESTED	No G6PD Def	% G6PD Def
1965	4,126	76	1·8%
1966	4,307	93	2·1%
1967	4,624	94	2·0%
1968	4,107	62	1·5%
1969	3,460	56	1·6%
1970	3,250	56	1·7%
1971	2,933	46	1·6%
1972	2,958	61	2·1%
1973	2,790	48	1·7%
1974	2,924	40	1·4%
1975	2,804	31	1·6%
1976	2,766	63	2·3%
1977	2,780	60	2·2%
1978	3,209	60	1·9%
1979	3,443	51	1·8%
TOTAL	50,436	897	1·8%

INDIAN

YEAR	No TESTED	No G6PD Def.	% G6PD Def.
1965	2,289	5	0·1%
1966	1,798	6	0·3%
1967	1,719	4	0·2%
1968	1,420	8	0·5%
1969	1,733	5	0·3%
1970	1,003	2	0·2%
1971	831	4	0·5%
1972	917	3	0·3%
1973	753	7	0·9%
1974	455	3	0·7%
1975	554	4	0·7%
1976	595	3	0·5%
1977	609	7	1·2%
1978	779	2	0·3%
1979	866	4	0·5%
TOTAL	1,953	67	0·34%

Figure 1 to show screening of newborn among Chinese, Malay and Indian infants for glucose-six phosphate dehydrogenase.

SCREENING FOR G-6-P-D ENZYME IN SINGAPORE NEWBORNS OVER A 15 YEAR PEROD

OTHER RACES

YEAR	No TESTED	No G6PD Def.	% G6PD Def.
1965	509	3	0·6%
1966	865	7	0·8%
1967	1,042	7	0·7%
1968	1,017	6	0·6%
1969	404	1	0·2%
1970	771	1	0·1%
1971	715	2	0·3%
1972	492	3	0·6%
1973	582	5	0·9%
1974	862	6	0·7%
1975	559	2	0·7%
1976	629	1	0·16%
1977	509	4	0·6%
1978	364	4	1·1%
1979	320	1	0·6%
TOTAL	9,640	53	0·6%

ALL RACES

YEAR	No TESTED	No G6PD Def.	% G6PD Def.
1965	35,937	549	1·5%
1966	35,393	588	1·6%
1967	35,040	561	1·6%
1968	32,608	481	1·5%
1969	29,860	489	1·6%
1970	27,940	388	1·4%
1971	26,134	375	1·4%
1972	27,117	471	1·7%
1973	27,008	494	1·8%
1974	25,892	405	1·6%
1975	23,245	411	1·8%
1976	25,391	480	1·9%
1977	22,254	455	2·0%
1978	22,930	400	1·7%
1979	23,559	403	1·7%
TOTAL	420,818	6.950	1·7%

APPROXIMATELY 2 OUT OF 100 BABIES TESTED IN SINGAPORE WILL BE G-6-P-D DEFICIENT. ONCE DEFICIENT THEY ARE KEPT IN THE MATERNITY UNIT FOR 3 WEEKS TO PREVENT TRIGGER FACTORS WHICH WILL PRODUCE JAUNDICE. EVEN IF JAUNDICE DEVELOPS, WE PREVENT MENTAL DAMAGE BY A TIMELY EXCHANGE TRANSFUSION.

Figure 2 to show screening of newborn among all races combined in Singapore for glucose-six phosphate dehydrogenase.

GLUCOSE-SIX PHOSPHATE DEHYDROGENASE DEFICIENCY IN FOUR GENERATIONS

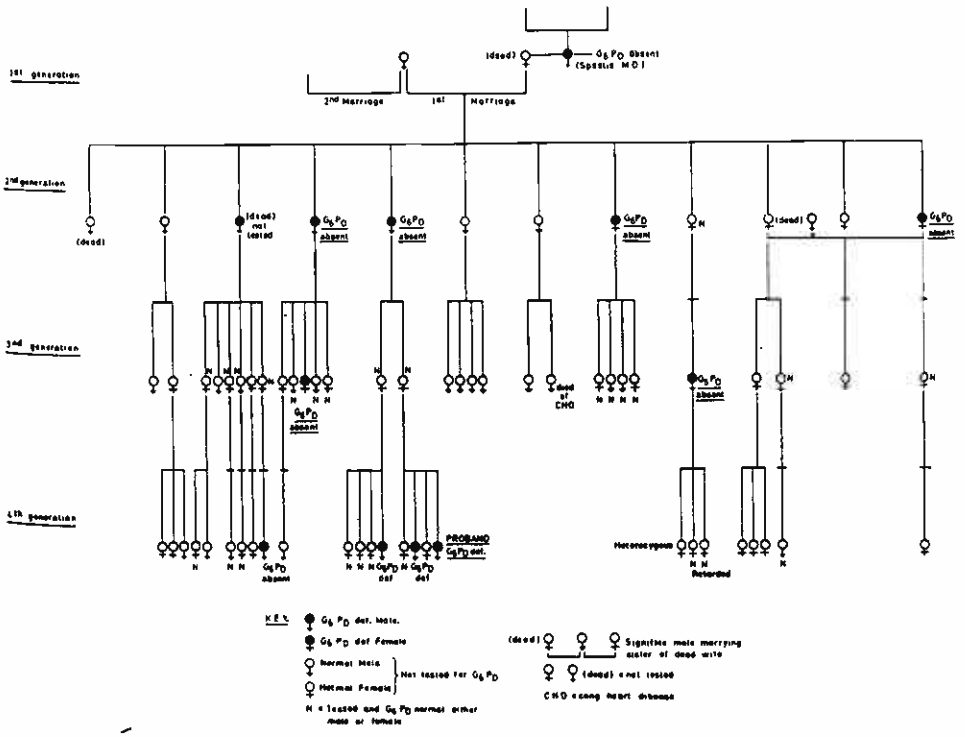


Figure 3

Note glucose-six-phosphate deficiency in four generations in a family.

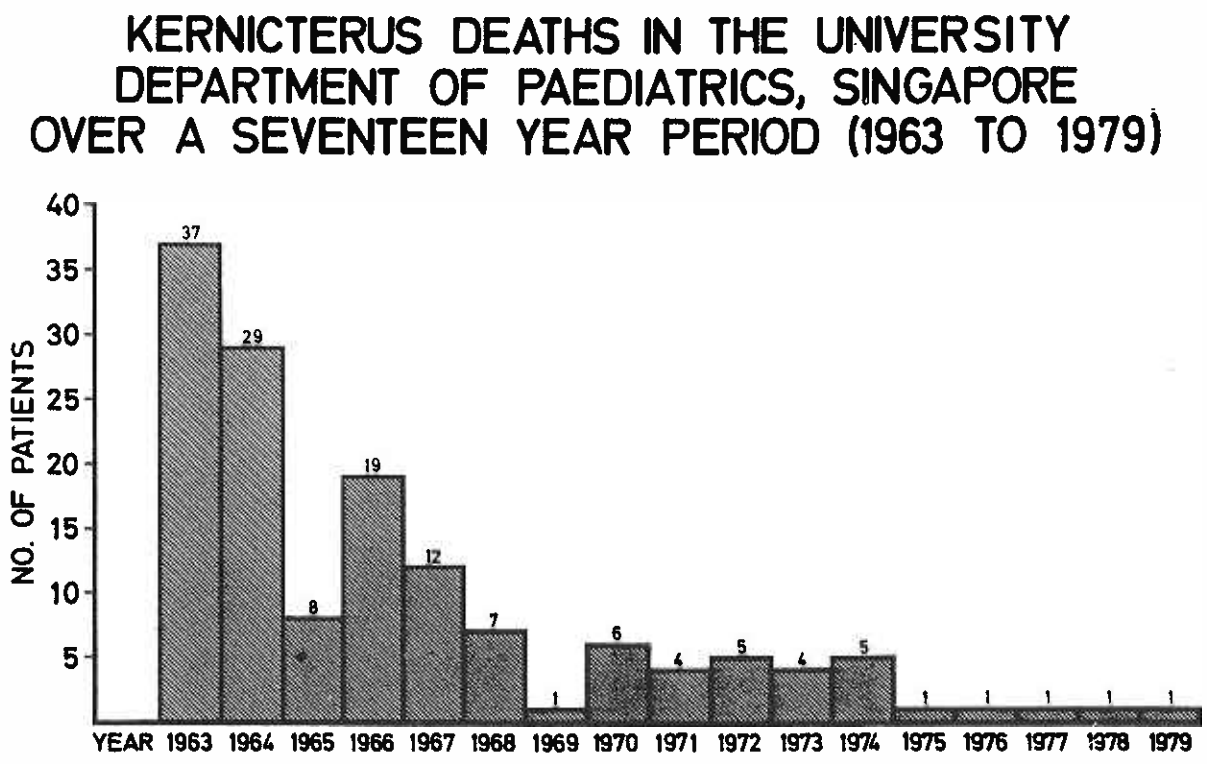


Figure 4 Note the reduction of deaths due to kernicterus over a seventeen year period.

CLINICAL MANIFESTATIONS OF KERNICTERUS

In 1968 Tan (2) classified 577 children with cerebral palsy and found 61% were classified as spastic, of whom 40% were diplegic, 16% were hemiplegic and 5% were paraplegic. The rest were 31.5% athetoid, 4% hypotonic, 1% ataxic and 0.5% rigid and 2% mixed. The high incidence of athetosis in the Singapore series was due to the large number of children who developed cerebral palsy as a sequel of kernicterus. Of the 183 children classified as athetoid, 56% had kernicterus in the neonatal period.

The diagnosis of kernicterus in the newborn period is based on the deep golden-yellow colour of the baby, the loss of primitive reflexes eg. the Moro's reflex, the suck and rooting reflex, evidence of dystonia involving the trunk and neck, internal muscles of the eye, or the extremities, and the feeble unsustained cry or the short, high-pitched shrill cry. The sex distribution of these 92 children showed a predominance of male children compared to female children. The racial incidence showed 90% were Chinese, 6.5% were Malays and 0.5% Indians with 3% of other races. The slight preponderance of Chinese compared to the population is due to the habit of the Chinese of taking herbs during pregnancy and in the immediate post-partum period.

These 92 children were graded as follows:—

(a) Group I included those patients who had severe dystonia and rigidity.
(b) Group II involved those where the dystonia was moderate but where involuntary movements were present.
(c) Group III included those where the dystonia was minimal or absent while the athetosis and choreiform movements were more obvious and dominated the clinical picture.
(d) Group IV included a group of children where there was no dystonia nor athetosisn nor choreiform movements. They had only delayed milestones and were all deaf.

The largest number of children were found in Group III i.e. athetosis with choreiform movements where the mean age of the children was 8.3 years. In contrast to western countries, this is the commonest type of cerebral palsy seen in Singapore, where the jaundice due to glucose-six-phosphate dehydrogenase mainly affected the basal ganglia producing kernicterus.

The age of which children would reach and grasp objects were analysed in four groups:
In Group I, where the child had severe dystonia and rigidity, the mean age of reaching objects was only 2½ years. In Group II, where there were 25 children where the dystonia was moderate, the mean age for reaching objects was 18 months. In Group III and IV where the main problem was athetosis and deafness respectively, all the children were able to reach and grasp objects and the mean age of doing this was at 10 months, the earliest being 8 months. Figure 6 illustrates the development of reach and grasp in the various groups.

Figure 7 illustrates the number of children and the age at which they were able to sit without support. There were only two patients in Group I who were able to sit alone, a six year old boy and a thirteen year old boy who sat up at the age of 4 years and 10 years respectively.

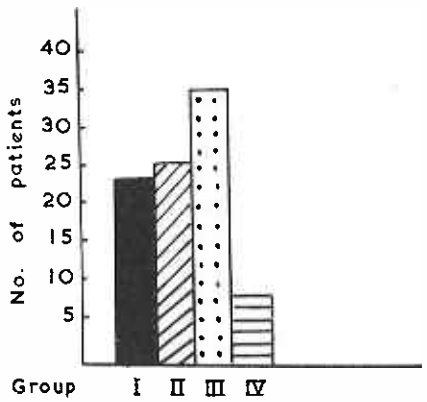
In Group II, 17 out of 25 children had learnt to sit without support. Six were able to sit between 2 to 4 years; all the children whose age exceeded 8 years were able to sit alone. The ages of the children who would not sit without support ranged from one to seven years.

In Group III which was the athetoid type, 32 of the 35 children were able to sit. All the children who were 5 years and above had learnt to sit without support. 28 of the children sat alone between the ages of 7 months and 3 years. The ages of the children who would sit were one year, 18 months and five years.

In Group IV, seven out of the eight patients were able to sit between the ages of 8 months to 2½ years. The majority sat between the ages of 8 months to 18 months. All the children who were 2½ years old and above were able to sit.

The developmental milestones for walking among the children in the four groups were shown in figure 8.

No. OF CASES AND AGES OF CHILDREN WITH CEREBRAL PALSY



Group I —severe dystonia and rigidity (mean-age 5·7 years)
Group II —dystonia was moderate (mean-age 8. 4 years)
Group III —athetosis with choreiform movement (mean-age 8. 3 years)
Group IV —deaf children with no dystonia nor athetosis (mean-age 1. 2 years)

Figure 6 DEVELOPMENT OF REACH & GRASP

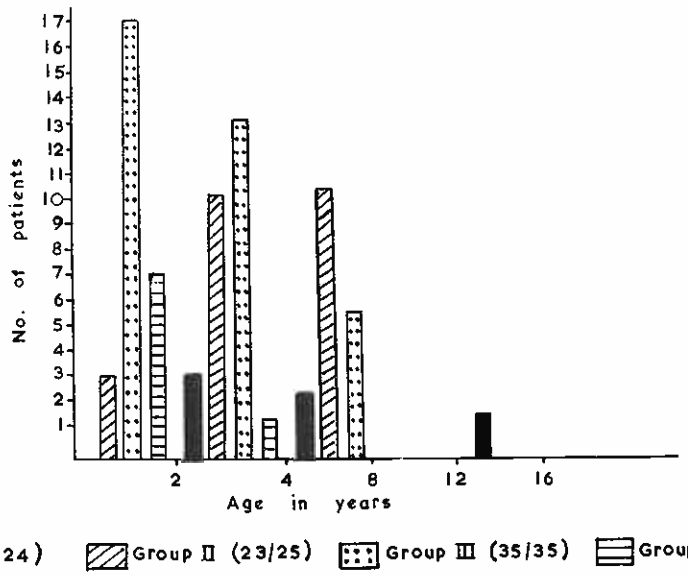
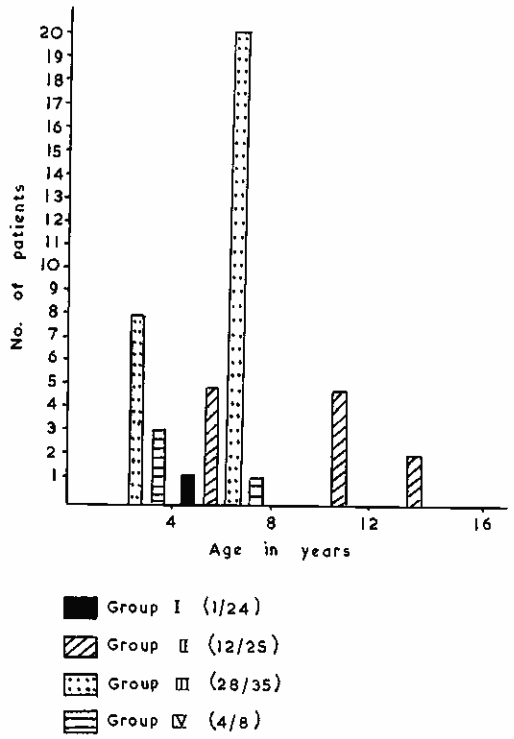


Figure 7 DEVELOPMENTAL MILESTONES FOR SITTING

Figure 8 DEVELOPMENTAL MILESTONES FOR WALKING



In Group I, only one child learnt to walk without support at the age of 6 years. The rest of the children were non-ambulant.

In Group II, 12 of the 25 children were ambulant. The earliest age when walking was acquired at 5 years. Thus none of the children in this group were able to walk below the age of 4 years. The ages of the children who were ambulant ranged from one year to thirteen years.

In Group III, twenty-eight children were ambulant, the earliest at 2 years and the latest at 6 years. The majority (20) learnt to walk between the ages to 4 and 6 years. All the children whose ages were above the ages of 5 years all learnt to walk without support.

In Group IV, 50% of the patients were able to walk, the earliest at 2 years and the latest at 4 years. The majority learnt to walk between 2 and 2½ years.

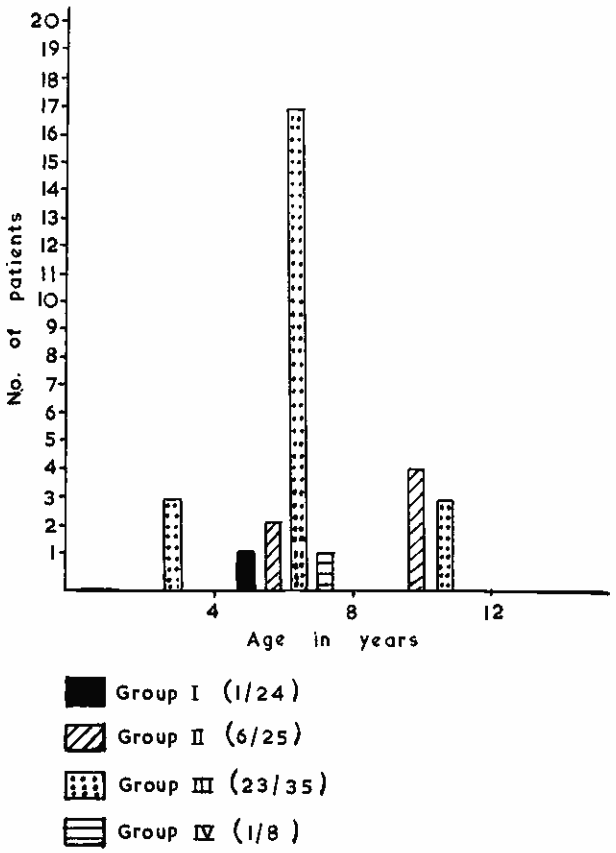
The handskills of the children in the four groups were tested. They were tested on their skills of daily living such as feeding, dressing and washing. The writing and drawing ability of the children in the normal school/school for spastic children were classified as good, fairly good, fair and poor.

The table below shows the handskills.

Handskills

	Good	Fairly Good	Fair	Poor
Group I	0%	0%	0%	100%
Group II	0%	8%	24%	68%
Group III	3%	31%	60%	6%
Group IV	37.5%	25%	37.5%	0%

Figure 9 SPEECH DEVELOPMENT



Group I was the worst with 100% poor skills. Their ability improved from Group II to Group III. In Group IV the majority of the children had good to fairly good performance with their hands. 36% of the children were left-handed.

Speech development was delayed in the majority and when it was developed the speech was slurred. Group I had only one child who began talking at 6 years and developed sentences at 13 years. Group II had 6 out of 25 children who had developed speech.

The majority in group III had developed speech. Their speech was slurred but intelligible.

Group IV had one out of eight cases who would talk in sentences, but all the children in this group were deaf.

Table

Incidence of Deafness

	No. of Cases	Percentage
Group I	16/19	84%
Group II	15/22	68%
Group III	22/33	67%
Group IV	6/7	86%
TOTAL	58/81	72%

Deafness was present in 58 out of 81 children. Audiograms showed partial perceptive deafness and high frequency deafness. It will be noted in the Table that deafness was present in 72% of the children tested.

Many of the children were emotionally labile. Temper tantrums were common among the younger children whereas hyperactivity and lack of concentration were noted among some of the school children.

Fits were noted in twenty out of 92 children giving an overall incidence of 22%.

Table

Incidence of Convulsions

	No. of Cases	Percentage
Group I	7/24	29%
Group II	6/25	24%
Group III	6/35	17%
Group IV	1/8	12.5%
TOTAL	20/92	22.0%

Group I had the highest incidence of convulsions and Group IV the lowest. The majority had grand mal epilepsy and were under control with antiepileptic drugs. Two patients in Group III had petit mal epilepsy.

It was not possible to do an intellectual assessment on all the children but those of the athetoid type had the highest I.Q.

OTHER CAUSE OF CEREBRAL PALSY

TUBERCULOUS MENINGITIS

The second environmental disease was childhood tuberculous meningitis causing either death or mental retardation and cerebral palsy in the form of spasticity of the limbs. A review of the departmental figures of tuberculous meningitis in the department show that twenty years ago tuberculous meningitis was a big killer of children and those who survived were spastic and mentally subnormal (3). Children under the age of two years were the most vulnerable group to tuberculosis. The average length of stay before a child sought medical treatment was two weeks or even longer (36%) while 60% were admitted in varying degrees of coma. The data from the University Paediatric Unit of the Singapore General Hospital show that there has been a progressive decrease in the incidence and mortality from tuberculous meningitis and over the last five years we have hardly had any children with tuberculous meningitis. Tuberculous meningitis as a cause of mental subnormality, spasticity and blindness has virtually disappeared from the island.

The factors responsible for the decline are:—

(1) The introduction of BCG Vaccination
(2) An improved nutritional status of the children
(3) A good tuberculous case finding system
(4) An improvement in the housing conditions
(5) A good outpatient medical service

JAPANESE B ENCEPHALITIS

The third environmental problem producing mental subnormality and spasticity was Japanese B encephalitis. In common with other parts of South-East Asia, Japanese B encephalitis exists in an endemic form in Singapore. Hale and Lee (4) have shown that 70% of the population over the age of 12 years in Malaysia, Borneo and Singapore have neutralising antibodies to the Japanese B encephalitis virus. The majority of the children admitted were more than 5 years of age (5). No child was seen less than 6 months of age because of the transplacental passage of the antibodies (4). The striking feature was the acuteness of the illness with the child admitted with high fever, malaise, drowsiness and convulsions. Headache was a significant symptom in the older child.

Seventy per cent of these children was admitted with varying degree of coma with neck rigidity, pupillary changes, abnormalities of conjugate eye movements, facial palsies and other motor deficits.

Abnormal purposeless movement due to basal ganglia involvement was also seen. The children remained unconscious for eight to nine days with the temperature falling by lysis on the 8th to 9th day. Forty percent of these children showed some form of sequalae, namely mental retardation and behaviour disorders in the form of temper tantrums, shrill cries, hallucination and emotional imbalance (6).

72% of the children with Japanese B encephalitis occurred in school children.

Both studies (5,6) show that most of the children with Japanese B encephalitis come from the rural area, probably because of the greater abundance of mosquitoes (Culex tritaenorrhyncus) and the animal reservoir, namely the pigs in those areas.

In countries where Japanese B encephalitis is endemic eg. in Japan epidemics are preceded by a peaking of viraemia in pigs.

Seasonal peaking is due to seasonal breeding of pigs. In Singapore, there is no seasonal viraemia because of the continuous breeding of pigs. A recent serological survey showed that the majority of the adult pigs had antibodies to Japanese B encephalitis virus. So obviously, mosquito control is the answer.

Due to the sustained efforts of the public health department in Singapore, the number of children and young adults affected with Japanese B encephalitis are reduced to a minimum.

PRENATAL CONDITIONS

Among the prenatal conditions that produce spasticity with mental subnormality are congenital syphilis, congenital toxoplasmosis, congenital rubella, cytomegalic inclusion body disease and herpes simplex infection.

CONGENITAL SYPHILIS

Congenital syphilis produces mental subnormality and spasticity of the limbs by causing a syphilitic meningitis which has been prevented by doing a Kahn test routinely on all mothers attending the antenantal clinic.

CONGENITAL TOXOPLASMOSIS

Toxoplasmosis is a common condition caused by Toxoplasma gonadii. Toxoplasmosis produces chorioretinitis, cerebral calcification and microcephaly. Studies in Malaysia and Singapore have revealed seropositively among a number of infants at the time of birth. However, there were only three reported children with congenital toxoplasmosis (7) and a further three causes were found in a survey of mental subnormality in Singapore (8).

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CONGENITAL RUBELLA SYNDROME

It is well known that rubella infection of the mother in the early months of pregnancy has resulted in mental deficiency, microcephaly and congenital heart disease.

While making a survey of mentally subnormal children the first few cases of rubella syndrome in Singapore were encountered (9). Doraisingham and Goh (10) studied the rubella immunity of women of child bearing age in Singapore. 2964 women in the reproductive age group (15 to 40 years) were tested for rubella haemagglutination inhibition antibody between January 1973 and December 1979. Most of them were pregnant women who were asymptomatic contacts of suspected rubella cases. Some were non-pregnant women who were asymptomatic contacts of susceptible rubella status. Some were non-pregnant women who were being screened for their immunity status and a relatively small number were women most of whom were pregnant, who were suspected of suffering from rubella.

The percentage of susceptible females varied from 51% in 1976 to 44% in 1979. There was a progressive increase in the percentage of immune female from the age of 15 years to 25 years after which these figures remained fairly constant at 52%. The high proportion of non-immune females in the reproductive age group in Singapore is similar to other island population e.g. Trinidad, Jamaica, Hawaii and Malaysia. It contrasts to the experience of western countries where a much higher percentage of females in the same age group have antibody to the virus. Rubella immunisation is offered to teenaged school children and the figure receiving rubella immunisation as a prophylactic measure against rubella are increasing.

OTHER VIRAL INFECTION CAUSING PRENATAL INFECTION

Cytomegalovirus infection is characterised by neonatal jaundice, chest infection, infection of the brain, spasticity and developmental delay. Unfortunately, there is no way of preventing cytomegalovirus body infection.

LOW BIRTH WEIGHT

Studies in Britain and other countries have shown that 28 to 42% of children with cerebral palsy were low birth weight or less than 2500 gms or less than 5½ lbs. Low birth weight babies have also been shown to carry an increased risk of epilepsy, autisum, blindness, deafness and mental retardation. The consequences of low birth weight babies have been reduced by the transfer of babies at risk, to a modern special care unit immediately after birth, and by cerebral monitoring after birth. We are fortunate in Singapore that all births are born in hospitals and premature units are present in government hospitals for the newborn.

Apart from infection which causes low birth weight, better nutrition of pregnant women is needed to reduce the number of low birth weight babies. Malnutrition is no longer a major problem in Singapore. Free nutritional counselling is carried out by the infant welfare clinics. However, nutritional counselling can be difficult because of the cultural habits of the people. Nutritional iron deficiency and megaloblastic anaemia are corrected at the antenatal period and later at postnatal follow-up.

NATAL CAUSES

Natal causes include abnormal pregnancy and labour, severe illness on the part of the mother e.g. toxaemia of pregnancy, prolonged labour, precipitate labour, cord round neck and cerebral anoxia. All these have to be dealt with by the obstetrician and the neonatal paediatrician at birth.

The extent to which cerebal anoxia may be responsible for cerebral damage and spasticity is always difficult to estimate as some children may be mildly affected by cerebral anoxia while others suffer a greater degree of physical and mental handicap.

However, conditions like a subdural haematoma and a hydrocephalus are treatable conditions. A subdural haematoma can be treated by daily needling and by evacuation of the membrane that causes haematoma. The prognosis is good if treated early.

Enlargement of the head due to a hydrocephalus can also be diagnosed by noting increase head size of the baby and by localising signs and symptoms which indicate increased intracranial pressure. Again if treated neurologically by insertion of valves to drain the cerebrospinal fluid from the ventricles into the jugular veins, the child's head is prevented from getting bigger and mental subnormality and spasticitiy are reduced.

FEBRILE CONVULSIONS AND EPILEPSY

In tropical countries, recurrent febrile convulsions produce brain damage. To prevent febrile convulsions, it is necessary to sponge the child with ice-cold water to induce hypothermia. A layer of water is left on the body to cool body by evaporation and this will further aid reduction of body temperature. Finally a fan is necessary to cool the body by evaporation. Repeated ice-water sponging becomes very necessary to prevent febrile convulsions. Epilepsy produces mental subnormality and cerebral palsy in 5% of our cases. Hemiplegic cerebral palsy due to an epilepsy is associated with a variable degree of

sensory impairment and the severity of the epilepsy depends on how controlled the condition is by antiepileptic drugs.

GENETIC CAUSES OF CEREBRAL PALSY

Cerebral palsy due to an autosomal recessive gene with multiple members in one family is rare.

CONCLUSION

In spite of preventive measures, one would still be left with children with cerebral palsy for which an assessment centre is needed so that one can assess them on an individual basis and plan out a programme of long-term care. Just as two normal children are never alike, so also spastic children must be assessed on an individual basis and a detailed comprehensive training programme outlined for each child. There is a great need in a country like Singapore to have an assessment centre for children with handicapping conditions.

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