Cerebral palsy in Nepal: a descriptive study of 136 disabled children presenting to a cerebral palsy centre in Kathmandu

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Abstract

There is little epidemiological data on the nature and causes of cerebral palsy (CP) in developing countries. This study reports 136 consecutive children presenting to a Cerebral Palsy centre in Kathmandu, Nepal. 108 had clinical evidence of nonprogressive disorders of movement or posture due to a defect of the developing brain (i.e. cerebral palsy) of which most (76%) were spastic in type. 90 of these children (82%) were additionally disabled by associated impairments of cognition, hearing or vision. There was a strong male bias in this series (male:female 74%:26%) reflecting in part the lower priority accorded to the girl child. Birth asphyxia of a severity sufficient to cause cerebral palsy always presents in the neonatal period with neonatal encephalopathy (NE). Of the 97 cases for which full historical details were available there was evidence of NE in 27 (28%). In only 19 cases (20%) was there good evidence of obstetric complications and birth asphyxia is the cause of the NE. The proportion of CP resulting from birth asphyxia in this series is similar to that found in industrialised countries. Even with improving maternity service provision in developing countries CP rates are unlikely to change dramatically. Provision of child disability services will remain a continuing priority for the foreseeable future.

Keywords: cerebral palsy; birth asphyxia; Nepal.

Introduction

The term Cerebral Palsy (CP) has proved clinically useful to define a sub population of disabled children with related problems predominantly affecting the motor system. CP results from a wide variety of pathological events or processes and maybe accomplished by seizures and impairments of hearing, vision, sensation and cognition. Epidemiological studies of cerebral palsy in industrialised countries have concluded that no more than 25% are attributable to birth asphyxia.1 There is little data relating to cerebral palsy in developing countries where perinatal morbidity and mortality is substantially higher. The most common cause of perinatal death in Nepal is thought to be birth asphyxia.2 This study examines what proportion of a series of over one hundred children with cerebral palsy in Nepal may have been caused by birth asphyxia.

When perinatal asphyxia significantly damages a term infant's brain it causes neonatal encephalopathy (NE). Studies elsewhere have shown that the encephalopathy must be of a moderate or severe character if cerebral palsy is to result.3 Encephalopathy of this degree is not subtle and causes delayed onset of sucking of greater than 24 hours duration, profound floppiness and may cause fits during the neonatal period.4 This study is based on the assumption that mothers can recall such serious disease in their newborn infants.

Methodology

Setting

The study was performed at the Self-help for Cerebral Palsy, Nepal in New Baneswor, Kathmandu. This local NGO has been offering services to children with cerebral palsy since 1987. At present it runs five programmes offering physiotherapy and counselling and a special education programme at the centre along with a home visit service, a district outreach programme and most recently a teacher training course in physical disability for primary school teachers. Over the years its services have expanded and to date it

has helped 642 CP cases and their families from 46 districts in Nepal. It has a staff of 9 therapists, 1 counsellor, 4 teachers and an administrator and is currently training a further 6 outreach workers for its expanding based programme. All new cases over a 14-month period between September 1995 and November 1996 were the subjects of this study. Historical details were collected by a combination of closed and open questions. Particular care was taken to critically assess the evidence for adverse perinatal events and significant neonatal disease. Neurological and developmental assessment followed conventional procedures.

Definitions and Classification

It is important to clarify terminology when discussing the complex field of disability.5 The World Health Organisation has clarified the following usages: *Impairment:* refers to abnormalities of structure; *Disability* to the consequences of that impairment in terms of functional disorder, and *Handicap* to the subjective assessment of the disadvantages of such functional disability. For example, a girl with cerebral palsy affecting the left arm and leg maybe described as having the impairment of hemiplegic spastic CP; her degree of disability depends on its impact on skills such as walking and the extent of her handicap will rest on many factors such as how far she lives from a road and how supportive her family is.

Cerebral palsy (CP) is an impairment of the motor system due to non-progressive brain damage sustained at any time during the period of brain growth. For epidemiological purposes CP resulting from recognised brain malformations (such as hydrocephalus) are usually excluded.

A consensus view on classification focuses on three predominant patterns of neurological involvement: spastic, dyskinetic and ataxic.6 *Spastic* CP results from involvement of the upper motor neurone of the pyramidal system which, depending on the location of the lesion, may affect only one side of the body in which case the CP is described as *hemiplegic,* both sides of the body with greater involvement of the legs than of the arms *diplegic*; or all four limbs equally, or with arm involvement predominating - *tetraplegic* (also called *quadriplegic*). *Dyskinetic* CP results from involvement of the extrapyramidal system, typically the basal ganglia, and includes those children with fluctuating dystonia, characterised by proximal mass reflex actions triggered by attempts at voluntary movement, and those with more discrete distal choreo-athetoid involuntary movements. *Ataxic* CP results from involvement of the cerebellum.

Hypotonic CP is a very non-specific category which is almost always associated with very profound degrees of cognitive impairment (mental retardation). Many epidemiological studies exclude such children since they are predominantly cognitively impaired. We therefore excluded those children whose mental handicap overwhelmed their physical impairment. Young infants with hypotonic CP which were considered likely to be in a transitory phase of an evolving CP syndrome have been retained.

Pathology often shows little regard for neuroanatomical subdivisions and many CP children have a mixed pattern of involvement. Following convention these children were classified according to the predominant pattern at the time of the examination. Retrospective assignation of the timing of the presumed insult causing CP is an inexact science. Dysmorphic features or severe intrauterine growth retardation were assumed to indicate antenatal pathology. Antenatal symptoms of illhealth in the mother were too vague to be useful and were ignored. Severe disease within the first week of life was taken as evidence of perinatal causation. Subgroups that could be distinguished by historical means were the very premature, good sized infants with neonatal encephalopathy (NE) following either adverse birth or severe jaundice and infants with neonatal sepsis. To meet the criteria for NE the mother had to confirm at least two of the three cardinal signs (lack of suck, floppiness or seizures) in the neonatal period. To meet the criteria for postnatal causation the developmental history of the child had to be normal prior to a clear episode of illness followed by a profound change in the child's abilities.

Results

A total of 136 consecutive new patients were assessed over the study period. Four of these were excluded from the study due to incomplete data. Of the remaining 132 referred to our centre, 108 met our case definition of cerebral palsy.

The clinical diagnoses of the 24 patients who did not meet our definition of CP are given below2

Referral Patterns

The background details of the 108 children with CP are presented in table I.

Table I: Background details of all newly referred Cerebral Palsy cases attending the Cerebral Palsy Group, Baneswor between September 1995 and November 1996.

Variable (n=number of subjects for which data available)	*Descriptive Statistics*
Sex Male:female (n=106)	81:25 (76:24%)
Age Mean (interquartile range) n=108	2.7 (1.2-6.0)
Age (by category) n=108 **1. Infant (<12 months)** **2. Preschool (1-5 years)** **3. Schoolage (>5 years)**	17 (16%) 57 (53%) 34 (31%)
Residence (n=106) **inside valley:outside valley**	72:34 (68%:32%)
Class (by paternal occupation) n=87 **1. Professional** **2. Wage earning/self employed** **3. unskilled/ subsistence farming**	3 (3%) 49 (46%) 35 (40%)
Referral (n=100) **HRDC (see text)** **Private Doctors** **Prasuti Griha Birth**	49 18 12 9

Asphyxia project:	7
Other hospitals	3
Self	2
CBR (see text)	
Gurkha Welfare Scheme	

There is a strong male bias in these series. Less than one fifth of patients reach the centre during the first year of life. Of the 72 valley residents 36 (50%) reached the centre by age 2. The majority of the children live in the Kathmandu valley reaching the centre through varied referral pathways. Most of the non valley residents were identified by Hospital for The Rehabilitation of Disabled Children (HRDC) fieldworkers based in the districts. 12 infant cases of perinatal brain damage identified in a prospective study of birth asphyxia were referred from Prasuti Griha Maternity Hospital.

Cerebral Palsy Types

Table IIa gives the proportions of this series of CP patients by clinical category. Spastic CP is by far the commonest general category. For each group information is also presented on the type and frequency of associated impairments. A majority of CP cases have an associated impairment of intellectual function with a minority in addition having impaired vision or hearing due to pathology of the central nervous system. Seizures were recorded in only three cases which is almost certainly the result of under-reporting. In Table IIb the spastic group is presented by clinical subcategory. The more severely affected quadriplegic patients with associated impairments clearly predominate. In contrast a significant proportion of diplegics and almost half of all hemiplegics had normal intellectual function.

Table IIa: Clinical categories of Cerebral Palsy and associated impairments.

	Spastic	*Dyskinetic*	*Ataxic*	*Hypotonic*	*Total*
CP only	14	4	1	0	19 (18%)
CP with cognitive impairment	49	8	6	7	70 (64%)
CP with cognitive impairment and impaired hearing/vision	19	0	0	0	20 (19%)
Totals	82 (76%)	12 (11%)	7 (6%)	7 (6%)	108 (100%)

Table IIb: Sub-categories of Spastic Cerebral Palsy and associated impairments.

	Diplegic	*Hemiplegc*	*Quadriplegic*	*Total*
CP only	4	9	1	14 (13%)
CP with cognitive impairment	9	9	31	49 (44%)

CP with cognitive impairment and impaired hearing/ vision	3	1	15	19 (19%)
Totals	16 (20%)	19 (23%)	47 (57%)	82 (100%)

Timing of insult

Table III analyses the CP cases by timing of the presumed insult.

Table III: Analysis of CP cases by presumed timing of insult.

Presumed timing/type of insult	*n*	*%*
Prenatal	33	31
Perinatal	41	38
Postnatal	16	15
Multiple Events	7	6
Insufficient information	11	7
Total	108	100

The clinical assignation of the timing of the insult followed the guidelines noted above. Where there was no evidence of insult in the perinatal or postnatal period (n=23) the pathology was presumed to have occurred antenatally. 41 (38%) of this series of CP cases had evidence of perinatal pathology of a severity considered likely to significantly contribute to their disability. 15% of this series were postnatally acquired. In seven cases there was evidence of multiple factors operating at different periods contributing to the cerebral palsy. In 11 cases there was insufficient information to retrospectively assign the timing of the insult.

Aetiology

Of the 41 cases with presumed perinatal insult 11 were significantly premature, six of whom developed a typical spastic diplegia. Three had strong evidence of hyperbilirubinaemia with marked jaundice and the characteristic dyskinetic choreoathetoid type of CP. The remaining 27 infants were apparently term infants whose mothers gave a history of neonatal encephalopathy. The majority (23/27) presented with severe spastic quadriparesis accompanied by cortical blindness in a substantial minority of cases (8/27). 19 of these were associated with an adverse birth history but in eight cases there was no evidence of obstetric complications. Some of these are likely to have resulted from perinatal infection.

Of the 16 cases with postnatal insults, nine occurred between one week and 12 months of age. The vast majority resulted from infection, with 12 cases giving a history of fever and fits considered likely to result from meningitis or encephalitis. Two cases were considered to result from severe dehydration due to diarrhoea and one case from anoxia caused by acute respiratory infection.

Discussion

Forty-one of the 97 patients for whom sufficient information was available had historical evidence of perinatally acquired CP. 27 (28%) of this series had evidence of neonatal encephalopathy which may have resulted from birth asphyxia. In only 19 (20%) was there good circumstantial evidence of obstetric complications resulting in neonatal encephalopathy and ultimately cerebral palsy.

There is no data describing the prevalence of CP in Nepal. Several disability surveys have been performed in recent years in Nepal.7 Most of these have been simple questionnaire surveys which importantly overestimate disability prevalence. Research has shown that the precision of a survey screening tool is significantly improved when backed up by a physical assessment of those screened positive.8 Studies employing this approach in Bangladesh9 and China10 have reported prevalence rates for physical disability amongst school age children of 3-4/1000 and 2/1000 respectively. In the only such study reported in Nepal a 25% fraction of the 12,552 reported disabled people in Kanchanpur, Nepal

(total surveyed population 246,938) were clinically examined.11 This study reported a physical disability rate of approximately 10/1000. Cohort studies and data from handicap registers estimate CP prevalence at school entry age in industrialised countries to be 2-4/1000.12 Longitudinal data suggests this has remained essentially unchanged since 1950 despite improving perinatal health services.

The current study is not population base and cannot inform us as to the prevalence of CP in Nepal. However, it does provide some insight into the types and possible causes of CP in this setting. It must be recognised that this group of CP patients, as with any institutionally recruited patient series, maybe seriously biased. Firstly, this group probably represents the severer end of the spectrum of childhood disability since these patients' families have shown persistence in finding their way to a specialised centre. Secondly, the demographic data shows that urban wage earners are over represented in this group. Thirdly, the strong link with a birth asphyxia project at Prasuti Griha Maternity Hospital during the period of this study has certainly caused an excess of early referrals following birth asphyxia. How may these biases have affected the proportion of perinatally acquired CP relative to other causes of CP in this group ? Studies looking at birth asphyxia have shown that it results in cerebral palsy at the severest end of the spectrum of disability. A recent study of birth asphyxia in a home delivering rural Indian setting showed that a higher proportion of damaged infants died in the neonatal period than is found in hospital delivering urban populations.13 It might therefore be argued that the proportion of CP thought to be perinatally acquired in this severely affected largely urban group is likely to be an overestimate of the actual proportion in the wider population.

Equivalent data from a series of 100 infants presenting to the Scottish Council for Spastics in 1990 shows 40% to be of antenatal causation, 37% to follow preterm birth, 16% to follow a asphyxia in term infants, and 7% to have been acquired postnatally.14 As with many industrialised world studies this demonstrates the increasing contribution of damaged premature survivors to the fairly constant overall level of CP.

What are the implications of these findings for the prevention of CP in Nepal ? Firstly, many cases of CP result from antenatal causes beyond our control. For example, a recent magnetic resonance study from Japan found 29% of CP cases to have structural lesions due to neuronal migration defects.15 Secondly, modern mass obstetric care has proved unable to eliminate a significant proportion of CP believed due to birth asphyxia in industrialised countries. This is likely to be the case in Nepal. Thirdly, postnatally acquired infective disease is probably the single most preventable cause of CP in Nepal.

So cerebral palsy is going to be with us for the foreseeable future. Clearly service provision for this disadvantaged group is a necessity. We know that early intervention gives the optimal chance of therapeutic impact. It also gives the parents the fullest opportunity to come to a realistic understanding of their child's problems. Most importantly it gives the child the best opportunity to learn to live with his impairment and thereby minimise his handicap. It is the physicians' duty to ensure referral at the earliest opportunity for therapeutic assessment. There are two current approaches to service provision for disability in Nepal. The centre-based approach exemplified by CPSHG provides specialised services from a central point. Community-Based Rehabilitation (CBR*) fosters a more participatory approach for a wide range of disabled people and their families. Both these systems are at an early stage of development as yet in Nepal. It is to be hoped that both types of organizations will realise each others strengths and work together. Medical practitioners should be aware of service providing NGO's in their vicinity.

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