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SPASTIC DIPLEGIA OF PREMATURE BIRTH

JOHN A. CHURCHILL, M.D.*

On the basis of differences in neurological findings, patients who have cerebral palsy can be grouped into several forms which are: (1) Athetosis, (2) Hemiplegia, (3) Ataxia, (4) Complex Diplegia, and (5) Simple Spastic Diplegia. The form of cerebral palsy termed "simple spastic diplegia", with which this presentation is particularly concerned, was described and differentiated from Little's disease in a recent publication¹. Patients who have simple spastic diplegia present the following findings: (1) Spasticity involving both lower extremities equally, the upper extremities being less spastic than the lowers, (2) Impairment of motility, especially of the legs, (3) Delayed onset of walking, (4) Contractures of the heel cords, thigh adductors and hip flexors, (5) Hyperactive deep tendon reflexes, and (6) Positive plantar responses. The findings can be interpreted in terms of bilateral damage to the so-called pyramidal tracts. Extraocular muscle imbalances are commonly found in these cases as in prematurely born infants generally². Patients who have simple spastic diplegia differ from those who have complex diplegia in a much lower incidence and lesser severity of mental subnormality and convulsive seizures. They lack positive tonic neck reflexes, athetoid and ataxic movements, and other manifestations which can only be explained on the basis of extensive cerebral dysfunction.

In our group of 70 patients who have simple spastic diplegia all except 11 were born prematurely. Six of the non-prematurely born individuals were delivered by breech extraction, and probably owe their disability to stretch injury of the spinal cord^{3,4}.

The distribution of birth weights of the patients with spastic diplegia is shown in figure one. These are compared to the birth weights of a group of patients who have no neurological disease, or have diseases acquired later in childhood; not of early origin. The predominance of cases with spastic diplegia in the lowest birth weight ranges is in sharp contrast to the lack of cases of low birth weight in the group of cases without neurologic diseases of early origin.

The correlation of simple spastic diplegia to low birth weight is even more strikingly demonstrated when the proportions of the cases in different birth weight ranges are corrected for neonatal mortality rates of infants in the different ranges⁵ (Figure 2).

The birth weight distribution curves of the other forms of cerebral palsy differ from those of patients with simple spastic diplegia (Figure 3). The birth weight distribution of the cases with complex diplegia reveals a fairly large proportion of the cases with moderately low birth weight, but even so, the curve is clearly distinct from that of simple spastic diplegia.

Groups of patients who have mental subnormality or idiopathic epilepsy differ from the group with simple spastic diplegia in the birth weight distribution curves (Figure 4).

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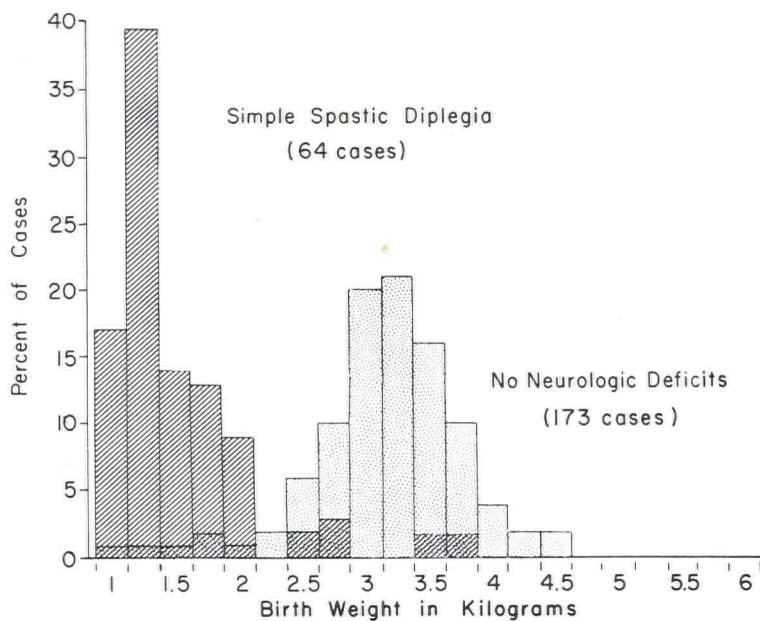


Figure 1
Birth weight and simple spastic diplegia compared to birth weight of normals.

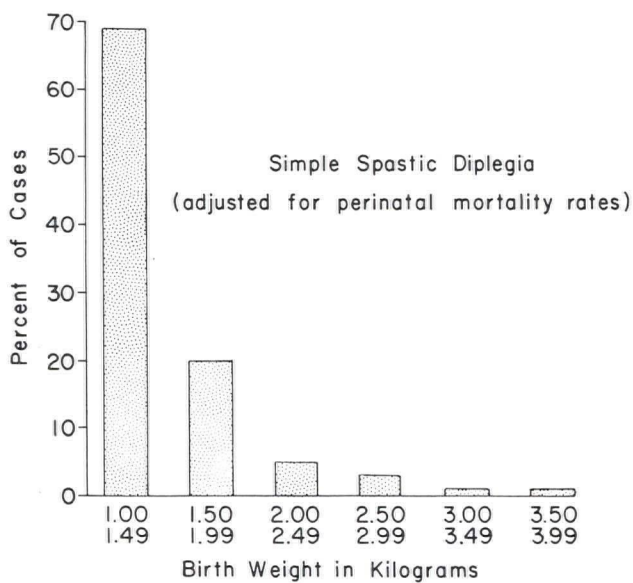


Figure 2
Simple spastic diplegia correlated with birth weight and corrected for neonatal mortality rate.

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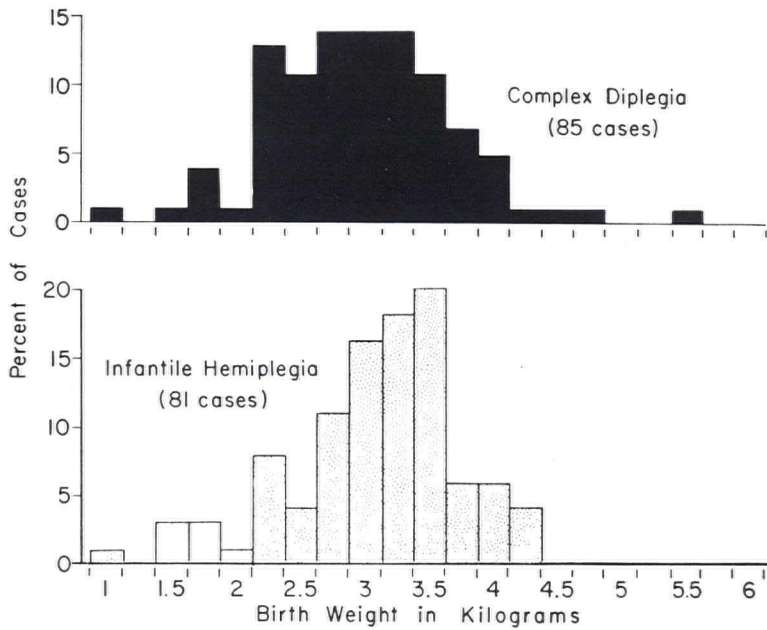


Figure 3
Birth weight by other forms of cerebral palsy.

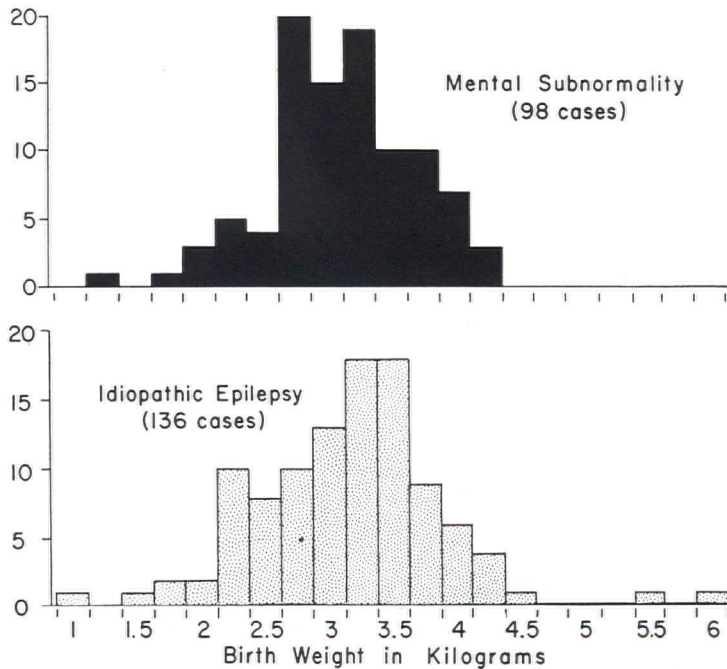


Figure 4
Birth weight by mental subnormality and by idiopathic epilepsy.

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Using gestation time as the criterion for prematurity it is again observed that the group of patients with simple spastic diplegia were born prematurely much more frequently than were patients who had no neurological disease of early origin (Fig. 5).

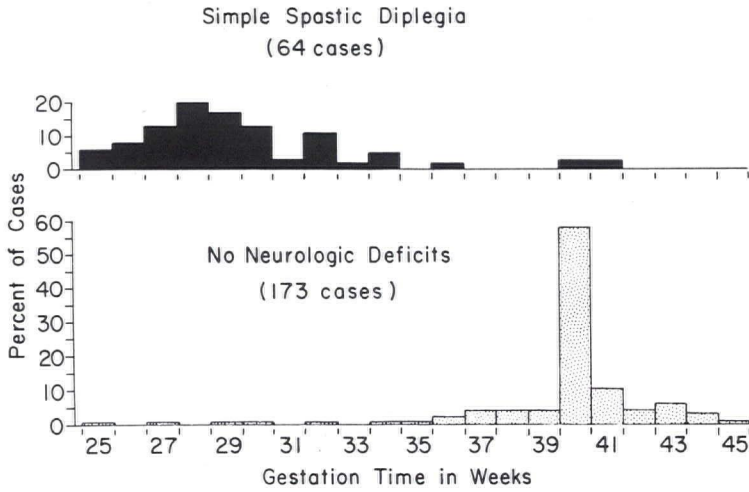


Figure 5
Simple spastic diplegia charted by gestation time.

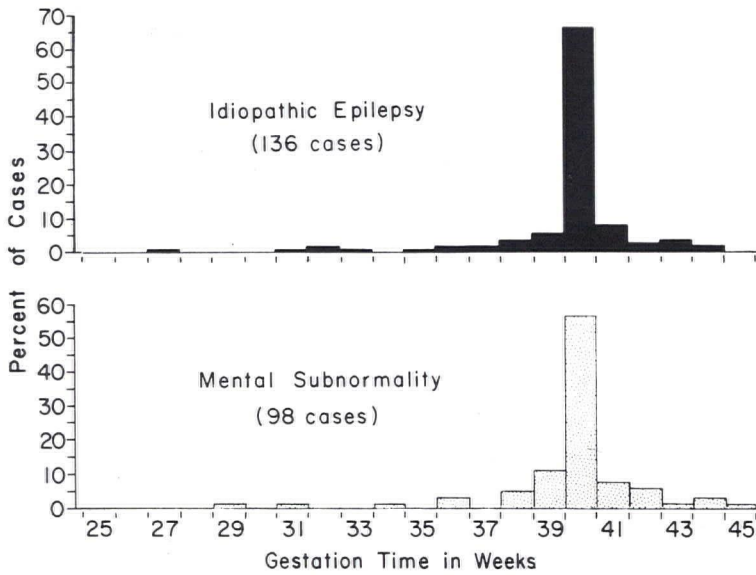


Figure 6
Complex diplegia and infantile hemiplegia charted by gestation time.

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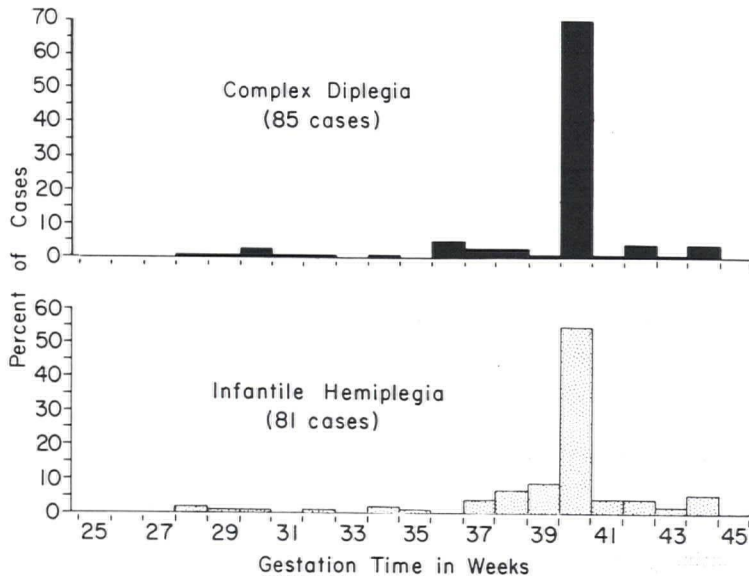


Figure 7

Idiopathic epilepsy and mental subnormality charted by gestation time.

Similarly, gestation times were much shorter in patients with simple spastic diplegia than in those with other forms of cerebral palsy, (Fig. 6) or in cases of mental subnormality or idiopathic epilepsy (Fig. 7).

The correlation of low birth weight, and also short gestation time, to simple spastic diplegia is very strong. No distinct correlation between birth weight or gestation time has been found in the complex diplegic (or quadriplegic), hemiplegic, athetoid, or ataxic forms of cerebral palsy. Neither has a correlation of this magnitude been found between prematurity and mental subnormality or epilepsy.

Spastic diplegia is now clearly established as a disease of the premature infant, and although the pathogenesis of this disease may not be directly derived from prematurity, this form of cerebral palsy can now be defined as spastic diplegia of premature birth.

The possibility that this disease may result from biochemical aberrations in the premature infant is now being investigated.

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