

Alphafetoprotein and neural tube defects

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The Neural Tube Defects

The neural tube defects (NTD) are a group of congenital malformations which are depressingly familiar to many obstetricians in the UK. Despite intensive investigation, their basic aetiology remains unknown. It seems probable that genetic factors are involved, interacting with environmental agents early in gestation to prevent closure of the neural tube. The number of the mutant genes is unknown, though it is probable that there is more than one locus (Carter, 1974). The environmental factors are also obscure in spite of recent attempts to implicate blighted potatoes (Renwick, 1972) and other common components of the mother's diet (Knox, 1972). Approaches to the prevention of spina bifida, anencephaly and hydrocephaly have thus been foiled by the inability to locate any of the causative agents needed in constructing a systematic epidemiology.

The two commonest forms of NTD are anencephaly and spina bifida cystica. Anencephaly is a lethal condition in which the brain is amorphous and the vault of the skull absent; it often occurs in association with spina bifida. Spina bifida cystica is a midline defect of the spine in which there is an external sacular protrusion. If the protrusion contains meninges and spinal fluid but no neural elements, it is referred to as a meningocele; if spinal cord and nerves are included in the sac, it is called a myelomeningocele or myelocele (Warkany, 1971). Only a small minority of cases of spina bifida cystica are meningoceles, a comparatively benign condition (Laurence, 1974). Thus the most common of these defects compatible with survival is the severe and often crippling myelocele spina bifida, more often than not associated with hydrocephalus. Less common to rare NTD are syringomyelocele, encephalocele, iniencephaly, exencephaly and uncomplicated hydrocephalus.

The incidence of neural tube defects has great geographical variation. High rates are found in Britain and Ireland, intermediate rates in other parts of Europe and North America and low rates in Africa, Asia and South America (Carter, 1974). In most surveys the number of cases of anencephaly

has been approximately the same as those of spina bifida. The highest incidence areas are Northern Ireland (7.2/1000), Wales (5.8/1000), Scotland (5.6/1000) and the Republic of Ireland (4.8/1000), while England has an overall rate of 4 per 1000 (Renwick, 1972). With the possible exception of the anatomical defects of the heart, neural tube defects are thus the commonest of the serious congenital malformations.

If a mother has already had a child with spina bifida the risk of recurrence for each subsequent child is about one in forty. There is, however, an equal risk that the next child will be an anencephalic, so that the overall recurrence risk of NTD is about one in twenty or five percent. The same situation applies if the index child is an anencephalic (Laurence, 1969). For the mother with two previous affected children, whether they both be spina bifidas, both anencephalics or one of each, the recurrence risk rises to over 10% (Carter and Roberts, 1967). These calculations are based on empirical data from retrospective surveys and are probably minimal; other categories of risk are shown in table I.

The severity of neural tube defects ranges in a continuum from the still-born anencephalic through to the minimally affected meningocele spina bifida. Though it is often argued that anencephalics present little burden to their families, it must be remembered that there is a chance that the next child may have spina bifida. In high incidence communities where knowledge of neural tube malformations is part of folklore such pregnancies are times of great anxiety. As any obstetrician knows, there is a great demand for a safe and reliable method of prenatal diagnosis which will prevent the recurrence of this type of family tragedy. Recent discoveries of the potential of amniotic fluid and serum alphafetoprotein measurements suggest that this demand can now largely be satisfied.

Alphafetoprotein

In 1956 Bergstrand and Czar discovered a new protein in the serum of early human fetuses. It had α_1 -electrophoretic mobility and was not detectable

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