

mending great caution in what is still an experimental treatment and for keeping careful records and measurements of what is being done. Nevertheless, when psoriasis is of such severity that potentially toxic drugs like methotrexate are under consideration, PUVA is probably the treatment of choice where it is available.

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Children who cannot read

Older children who cannot read are a sad group. They will have had many years of frustrating failure and will, all too often, be shorn of self-confidence and hope. Their prospects might have been better if they could have been identified and helped earlier—but identification is usually easier than help.

In finding poor readers it is useful to know which children are most at risk. Low intelligence (IQ) is an important association.¹ Children with brain damage and cerebral palsy are also at risk even when their IQ is normal.² Temperament is also important: quiet, persevering, reflective children find it easier to read, and impulsive, hyperactive, distractible children have difficulties.³

There are also family and social factors that provide pointers. Low socioeconomic status and large families are associated with low verbal IQ and reading retardation. This seems to be a result of both hereditary factors and persisting environmental factors. Rutter⁴ has emphasised the "social transmission" of poor reading by parents who cannot read and may bring up a child to despise books and conventional education. Such children are deprived of both opportunity and motivation.

Folklore suggests that left-handedness and mixed laterality are particularly associated with reading difficulty. Numerous studies show that folklore is wrong. Certainly left-handed children may need to be taught a slightly different method for writing⁵ which depends on a left-to-right movement across the page, but they should not end up with reading problems. Nor should those with mixed laterality. Jimmy Connors, whose tennis racket is as effective in his left as in his right hand, need not be expected to have difficulty reading or signing lucrative contracts. Left-right confusion, however, is associated with reading difficulty.⁶

Specific reading retardation is defined as a degree of retardation that cannot be explained by low intelligence and generally retarded skills. While uncommon, it has generated a disproportionate amount of publicity in recent years. To those with no personal interest the controversy over the existence of "dyslexia" seems at times to be a semantic argument; and, indeed, dyslexia is an unsatisfactory term for specific reading retardation. Early reports suggested a unitary definition for

dyslexia—one cause, one disorder, and even at times one treatment. Specific reading retardation is a more general term, admitting the possibility of many different causes, including hereditary and environmental adversity. It does run in families, and there seems to be a genetic basis; but those who inherit the trend are particularly likely to express the behaviour—poor reading—in an adverse environment (for example, a large family or a poor school).⁷

There are many theories about the best education for children with reading problems. Some of the objections to the crusade for better provisions for "dyslexic" children come from people who recognise the needs of all children with reading backwardness to receive help. (Some would go on to emphasise that children with general reading retardation often respond better to special teaching than those with specific reading retardation.) There are enthusiastic advocates of specific teaching methods, many of them dramatically different, but all methods have enthusiastic advocates, who are likely to achieve their results by their particular skills as remedial teachers. Regardless of the exact method, they have the skill to motivate the child and to organise his or her education in small stages which allow progress to be appreciated and achievement to bring reward. Unfortunately there is a shortage of remedial teaching for poor readers. It is this shortage which makes early identification of children with reading problems less useful than it should be.

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Fetal haemoglobin, sickling, and thalassaemia: a therapeutic lead?

Sickle-cell anaemia is responsible for considerable mortality and chronic ill health in Africa and is a major public health problem wherever there is a large immigrant population of African origin.¹ It results from a single amino-acid substitution in the beta-chain of adult haemoglobin. In a way that is still not completely understood this structural alteration causes cross-linkage of the haemoglobin S (Hb S) molecules in the deoxygenated state. This leads to the formation of filamentous structures. The distortion of the red cell membrane by these filaments causes sickling, reduced survival of the cells, and recurrent blockage of the microcirculation; the result is infarction of tissue.²

The clinical course of sickle-cell anaemia is remarkably variable. In part this results from environmental and sociological factors, but some of the heterogeneity is genetic. For example, the unusual mildness of the disease in Saudi Arabia may be due to the unusually high concentrations of fetal haemoglobin (Hb F) in those affected,³ for erythrocytes containing relatively large amounts of Hb F as well as Hb S survive longer in the circulation and do not sickle as readily as cells containing mainly Hb S.⁴⁻⁶ Further information about the natural history of sickle-cell anaemia in Saudi Arabs has come from Perrine and his colleagues,⁷ who studied 270 people with the disease from the oasis villages in Quatif and Al Hasa. The condition was remarkably mild. Serious complications occurred much less often than in affected American or Jamaican blacks; leg ulcers were not seen; death rate under the age of 15 was greatly