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, distractable 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. Rutter[4] 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 writing[5] 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

reduced; there was less anaemia; and the reticulocyte count was considerably lower. Indeed, most of the 270 affected were completely asymptomatic and were found only in the course of population screening. Again the mildness of the disorder was ascribed mainly to the unusually high levels of Hb F.

Relatively large amounts of Hb F are also an advantage in the other common inherited disorder of haemoglobin production, beta-thalassaemia. Individuals who inherit a gene for persistent Hb F synthesis in addition to that for beta-thalassaemia have much less severe anaemia than those who inherit only the thalassaemia.[8 9] Furthermore, those forms of beta-thalassaemia in which the molecular defect in haemoglobin synthesis itself results in increased Hb F are generally milder than those in patients with limited Hb F production.[10]

If, then, relatively large amounts of Hb F in adult life are so helpful to sufferers from sickle-cell anaemia and homozygous beta-thalassaemia, clearly we should be looking for some way to manipulate the normal switch from fetal to adult haemoglobin production that occurs about the time of birth. Unfortunately, we know virtually nothing about the mechanisms that control the switch. Indeed, it is not even clear why fetal haemoglobin persists at all in patients with sickle-cell anaemia or homozygous beta-thalassaemia. All normal adults continue to produce a few red cells that contain Hb F (F cells), and in sickle-cell anaemia and beta-thalassaemia the increased concentrations of Hb F possibly reflect no more than simple selection of the normally occurring F-cell population during erythroid maturation or in the peripheral blood.[11] Clearly, however, such cellular selection does not account for the very high concentrations of Hb F found in Saudi Arabs with sickle-cell anaemia: some other genetic mechanism must be acting to cause an absolute increase in Hb F production.

Research into the control mechanisms concerned in the change from fetal to adult haemoglobin is progressing, albeit slowly.[10] Several approaches are being explored, including detailed investigation of adults with genetically determined persistence of Hb F, study of the change from fetal to adult haemoglobin production in animals such as sheep and goats,[12] and analysis of the interesting phenomenon whereby Hb F synthesis can be reactivated in adult red cells maintained in tissue culture.[13 14] At the moment none of these approaches has given any real insight into the precise mechanism of the control of haemoglobin switching, though evidently it relies on certain critical sites in the cluster of genes that determine the structure of the gamma- and beta-chains of fetal and adult haemoglobin respectively.[10] Shortly the new techniques of restriction mapping of DNA[15] should be able to clarify the detailed structure of this area of the genome. Meanwhile we shall look forward to the results of further detailed genetic analysis of the remarkable Saudi Arab population.

The idea of being able to make therapeutic use of genes that are normally switched off during development is novel. Fetal haemoglobin is a perfectly adequate oxygen carrier in adult life; if its synthesis could be maintained after birth, even at a low level, we could control the distressing and frequently fatal clinical manifestations of both sickle-cell anaemia and beta-thalassaemia.

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"Proceed with the pricing"

The CCHMS has achieved a satisfying result to the ballot on the new contract (p 67). It confirms the favourable reception given to the proposals at the countrywide meetings of consultants addressed by the chairman of the committee and his senior colleagues. The 12 308 valid votes cast, representing over 68% of the electorate, is a respectable response in a poll of this nature. With decisive majorities in the returns from each of the groups balloted—NHS contract holders and senior registrars voted 71% in favour, while honorary contract holders voted 3:2 in favour—and with 50% of *all* NHS contract holders supporting the new contract the CCHMS has an unarguable mandate to take the next step: to ask the Review Body to price the contract. Certainly a well-attended special meeting thought so when last week (p 67) it voted overwhelmingly—nem con with one abstention—to instruct the Negotiating Subcommittee "to proceed with the pricing exercise, taking into account all the relevant factors."

The BMA has been criticised for its conduct of the ballot (p 58). The background to the poll was given to the CCHMS at its meeting. To maintain an accurate roll of its own members' addresses and type of employment is not easy, for the BMA can depend only on the individual member to report changes of address or employment. To maintain a similar roll for the whole profession is even more problematical because non-members have no obligation to tell the BMA of such changes. Yet the Association is the only medical organisation which maintains a list of doctors classified by their employment and grade. To offset the list's known defects the BMA did two things: it obtained lists of staff in post from employing authorities to check against the Tavistock Square lists and it publicised widely invitations to eligible voters to apply directly for ballot papers if they had not received one. Given the imperfections of the electoral roll it is hardly surprising that some voting papers failed to reach their destination. But it is reasonable to assume that most arrived and that most of the 5700 non-voters either did not wish or perhaps could not be bothered to vote. That is an acknowledged hazard of elections and polls in a democratic society.

The CCHMS chairman, Mr A H Grabham, and the chairman of the Negotiating Subcommittee, Mr D E Bolt, have ably led the CCHMS over some formidable obstacles in the quest for improved pay and conditions of service for their constituents.[1] It has also been an exemplary exercise in democracy. Yet the most difficult part of the course may well be the next few months. The Negotiating Subcommittee will have to prepare evidence to convince the Review Body—and the Government—that for the new work-sensitive contract to be effective will require more than a redistribution of consultants' present pay. Extra money is essential. Effecting a radical change of this complexity will not be easy (though criticising this or that part of the package will be all too easy) and CCHMS members understand consultants' wariness about the committee judging the final outcome without a further ballot. A good or bad result would present little problem, but last Thursday's discussion in the committee should reassure everyone that a ballot will be held if there are doubts. Meanwhile, consultants must trust their negotiators. So far, the CCHMS has done a difficult task democratically and commendably well: it deserves the profession's unwavering support during the coming months.

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