insist on jogging while taking beta-adrenoceptor blockers will have to choose their drug on the basis of trial and error.

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[1] Pearson SB, Banks DC, Patrick JM. The effect of β-adrenoceptor blockade on factors affecting exercise tolerance in normal man. *Br J Clin Pharmacol* 1979;**8**:143-8.
[2] Anderson SD, Bye PTP, Perry CP, Hamor GP, Theobald G, Nyberg G. Limitation of work performance in normal adult males in the presence of beta-adrenergic blockade. *Aust NZ J Med* 1979;**9**:515-20.
[3] Kaijser L, Kaiser P, Karlsson J, Rössner S. Beta-blockers and running. *Am Heart J* 1980;**100**:943-4.
[4] Williams AJ. Propranolol and marathon running. *Am Heart J* 1979;**98**:542.
[5] Bowman WC. Effect of adrenergic activators and inhibitors on the skeletal muscles. In: Szekeres L, ed. *Adrenergic activators and inhibitors*. Berlin: Springer-Verlag, 1980.

The hyperkinetic child: two views

For many years British child psychiatrists and paediatricians have been puzzled by the apparently considerably higher incidence of the hyperkinetic syndrome in children found by North American clinicians. Behaviourally defined, the syndrome includes motor restlessness (overactive, fidgety behaviour), distractibility, impulsiveness, and excitability. It is at least four times commoner in boys, starts early in life, and is often accompanied by difficulties in learning and antisocial behaviour. In North America between 5% and 20% of primary school children seem to be affected, and the syndrome is diagnosed in up to half of children referred to child psychiatrists.[1] In Britain, in contrast, the diagnosis is made in only 1-2% of children within the normal range of intelligence seen by child psychiatrists.[2] Are these considerable differences a result of different diagnostic criteria or categories, differing rates of referral to specialist clinics, or different beliefs about the efficacy of stimulant drugs, or do they reflect a real difference in incidence, perhaps due to environmental influences?

The scientific approach to these questions would require comparisons of American and British diagnosticians rating case histories and observing videotapes of the same children, but no such study has been carried out—though such a project has considerably clarified diagnostic differences in schizophrenia.[3] Recently, however, Rutter and his colleagues have reported two studies, one clinical and one epidemiological, showing that the broader concept of the hyperkinetic syndrome put forward by American paediatricians and psychiatrists cannot be sharply differentiated from what would in Britain be called conduct disorder.[4] Nevertheless, there appears to be a narrower concept of "pervasive" hyperactivity, which may be distinguished from other clinical syndromes.[5] This view is based on a further analysis of data from the survey of 10-11-year-old children living in the Isle of Wight in 1964 and followed up in 1968. A "hyperactivity" factor was extracted from questionnaires administered to parents and teachers. Children rated as hyperactive in one setting only (situational hyperactives; 14% of the total population) were distinguished from pervasive hyperactives (hyperactive both at school and at home; 2%) on several factors.

Pervasively hyperactive children were of significantly lower IQ on the non-verbal scale of the Wechsler Intelligence Scale for Children than both situationally hyperactive and non-hyperactive children. They were also of lower social class, but the difference held good even when social class was controlled for. In all social-class groups the pervasively hyperactive children had lower cognitive scores. Especially when pervasive, hyperkinesis was associated with conduct disorders but even so only half the pervasively hyperactive group were rated as having any other disturbance, so that the narrower concept of the syndrome certainly could not be equated with disorders of conduct. Furthermore, follow-up showed that persistent behavioural disturbance was associated with pervasive hyperactivity even in children of normal intelligence. For children who had emotional and conduct disorders at 10 years who were not hyperactive the prognosis was good: the disorder persisted into adolescence in only one child in 10. Of the pervasively hyperactive children with conduct or emotional disturbances, in contrast, these problems persisted into adolescence in 80% of cases and in 47% of the situationally hyperactive children.

Hence there seems to be a small but important group of children who are hyperactive at home and at school, who have a high rate of general behavioural disturbance, appreciable cognitive impairment, and a poor prognosis in adolescence. The poor prognosis is, moreover, a function of their hyperactivity rather than their conduct or emotional disorder. Despite the limitations of this study—the children were not examined clinically, cognitive tests were administered to groups and not individuals, and the children were past the peak age for hyperactive behaviour—it helps us to understand the differences between British and American findings.

Treatment cannot be based on a sound understanding of aetiology.[1] Some evidence exists for a genetic (possibly temperamental) contribution, for structural or physiological brain abnormalities (for example, defects of arousal), for toxic or allergic reactions,[6 7] and for combinations or interactions with family factors including maternal depression, early deprivation, and poor family organisation.

Pervasively hyperactive children of school age respond well to treatment with stimulants, but in younger children the effects are less certain. Reviewing 110 studies, Barkley concluded that three-quarters of hyperkinetic children given amphetamines or methylphenidate improved, while a quarter did not change or became worse.[8] The rationale for this treatment is far from clear, but cortical inhibitory systems are thought to be stimulated by these drugs, and certainly severely affected children improve in concentration and their control of impulsive behaviour.

Stimulants seem generally safe—the side effects of insomnia and suppression of appetite and growth can be controlled by restricting administration to the daytime and by holidays from the drug. Some children, however, do become depressed and apathetic during treatment and a few are quite severely adversely affected. Virtually no reports have appeared of amphetamine addiction or dependency or of other long-term adverse effects,[9] and in some children the therapeutic effect is instant and dramatic. The drug of choice is methylphenidate 5-40 mg in two daily doses. Imipramine has also been reported to be effective but is more toxic. Drugs are not panaceas, however, and need to be combined with other treatments if their short-term impact is to be maintained, for they appear not to affect outcome otherwise. The results of trials of various behavioural techniques with and without drug treatment have been promising,[10 11] and these methods are probably more effective in controlling hyperactivity, whereas drugs have a greater impact on attention and impulsive behaviour.

Diets low in allergens or food additives are on trial both in the United States and in Britain. Keeping to such diets entails great maternal determination and some social hardship, but some evidence has been produced that food colourings may be a toxic factor affecting some hyperactive children.[7]

While it is unlikely that the pervasively hyperactive child's behaviour is caused by parental behaviour,[12] the child and his family need to be helped to understand the disorder and to avoid the scapegoating and rejection which sometimes occur. Family therapy techniques appear to offer the most promising psychotherapeutic approach to helping the handicapped child.[13]

The prognosis for hyperkinetic children is poor in adolescence. The hyperactivity diminishes, but antisocial and cognitive difficulties persist and increase. One controlled follow-up study which extended beyond adolescence, however, gives a more hopeful picture. Weiss and her colleagues[9] followed up for 10 years 75 children who fitted the criteria of pervasive hyperkinetic syndrome and 44 matched controls and found that, while hyperactive subjects continued to be more impulsive and restless and had more car accidents, there was no excess of antisocial behaviour, severe psychopathology, job instability, or drug abuse compared with the control group. Virtually none of these patients had had any form of effective drug treatment.

Werry,[13] reviewing Cantwell's book[14] on the hyperactive child, ends by saying "true or false, the notion of the hyperactive child has been one of the most heuristic in child psychiatry since Bowlby's concept of maternal deprivation. . . . That alone makes it a good scientific concept . . . the fact that many American doctors overdiagnose hyperactivity and overprescribe stimulants is no more an argument against either the diagnosis or its treatment than is the gross misuse of penicillin . . . an argument against careful bacteriological diagnosis and antibiotic treatment."

Good child psychiatric practice now requires the identification of children with pervasive hyperactivity severe enough to interfere with functioning and a pragmatic approach to treatment which embraces the child and his family.

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1 Cantwell DP. Hyperkinetic syndrome. In: Rutter M, Hersov L, eds. *Child psychiatry: modern approaches*. London: Blackwell Scientific Publications, 1977.
2 Rutter M, Shaffer D, Shepherd M. *A multi-axial classification of child psychiatric disorders*. Geneva: WHO, 1975.
3 Edwards G. Diagnosis of schizophrenia: an Anglo-American comparison. *Br J Psychiatry* 1972;**120**:385-90.
4 Sandberg ST, Rutter M, Taylor E. Hyperkinetic disorder in psychiatric clinic attenders. *Dev Med Child Neurol* 1978;**20**:279-99.
5 Schachar R, Rutter M, Smith A. The characteristics of situationally and pervasively hyperactive children: implications for syndrome definition. *J Child Psychol Psychiatry* 1981;**22**:375-92.
6 Taylor E. Food additives, allergy and hyperkinesis. *J Child Psychol Psychiatry* 1979;**20**:357-64.
7 Swanson JM, Kinsbourne M. Food dyes impair performance of hyperactive children on a laboratory learning test. *Science* 1980;**207**:1485-6.
8 Barkley RA. A review of stimulant drug research with hyperactive children. *J Child Psychol Psychiatry* 1977;**18**:137-65.
9 Weiss G, Hechtman L, Perlman T, Hopkins J, Wener A. Hyperactives as young adults. A controlled prospective ten-year follow-up of 75 children. *Arch Gen Psychiatry* 1979;**36**:675-81.
10 Christensen D-E, Sprague RL. Reduction of hyperactive behaviour by conditioning procedures alone and combined with methylphenidate. *Behav Res Ther* 1973;**11**:331-4.
11 Douglas VI, Parry P, Marton P, Garson C. Assessment of a cognitive training program for hyperactive children. *J Abnorm Child Psychol* 1976;**4**:389-410.
12 Humphries T, Kinsbourne M, Swanson J. Stimulant effects on co-operation and social interaction between hyperactive children and their mothers. *J Child Psychol Psychiatry* 1978;**19**:13-22.
13 Werry JS. Critical notice. *J Child Psychol Psychiatry* 1977;**18**:195-6.
14 Cantwell DP. *The hyperactive child: diagnosis, management, current research*. New York: Spectrum Publications, 1975.

Personal Review

Schools of public health

ROY M ACHESON

Throughout the world there are probably some 50 schools of public health, 21 of them in the Americas.[1] All have contributed notably to the health of the country in which they are located and usually far more broadly too. W H Welch[2]—the creator and first dean of Johns Hopkins School of Medicine and later the founder of Johns Hopkins School of Hygiene in 1918—had enough trust in their potential to encourage their dissemination with generous support from the Rockefeller Foundation. "Merely from a mercenary and commercial point of view it is for the interest of the community to take care of the health of the poor," he declared. "Philanthropy assumes a totally different aspect in the eyes of the world when it is able to demonstrate that it pays to keep the people healthy. . . . It is estimated, and of course such an estimate can be only a rough one, that nearly 100 000 deaths occur annually in this country from preventable causes. For each death there are of course several cases of illness not fatal, due to preventable causes. One can form from such a statement some idea of the enormous loss of money and productive labour which we suffer from preventable causes of illness and death."

The purpose of schools of public health was achieving the ends spelt out by Welch. He looked on two of the most important as being his own school in Baltimore and from 1929 the London School of Hygiene and Tropical Medicine. He told his old Yale Medical School classmates in 1920 that he had "retired from my chair in pathology in Johns Hopkins University to assume a new kind of work . . . to me extremely interesting and somewhat novel. . . . Laboratories and institutes of hygiene have existed as parts of medical schools, but the conception here is an independent faculty, and independent school, as part of a university existing side by side with the other faculties of the university, particularly the medical faculty