Three Pitfalls in the Early Diagnosis of Mental Retardation

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Editors' Note: This brief clinical editorial, written from the point of view of a consultant in the field of developmental disabilities, is aimed at raising the clinician's index of suspicion for the diagnosis of mental retardation at an early age. One reviewer of the article felt that the counterpoint of each of the author's three pitfalls needed to be considered: one, dull appearance does not rule out normality; two, delayed motor milestones do not equate with retardation; three, there may not be a lot to gain by intensive early evaluation for possible borderline retardation. The practicing clinician must strike a balance between the opposing concerns of neither delaying inordinately the diagnosis of mental retardation nor creating an iatrogenic "vulnerable child" in an otherwise normal situation.

Early diagnosis of mental retardation is valuable because it allows identification of entities with a recurrence risk, institution of supportive counseling for parents, and institution of an infant stimulation program for the child. Pediatricians, however, generally overestimate the abilities of young mentally retarded children, systematically misclassifying most such children as normal. The author, as a consultant in the field of developmental disabilities, has been dismayed by the regularity with which referring physicians commit one or another of three significant errors, leading to unreasonable delay in the diagnosis of mental retardation. These errors may be summarized as follows: first, cute children are missed; second, ambulatory children are missed; third, it is erroneously assumed that below a certain age children are "too young to test." Although each of these errors may seem trivially obvious, they remain distressingly frequent.

Cute Children

Three per cent of the pediatric population have IQs below 70 and are mentally retarded. One-sixth of the retarded population have IQs of 54 or less (moderate, severe, and profound mental retardation). Mildly retarded children (IQ 69–55) comprise the bulk of the retarded population; most of these children do not have syndromes, are not dysmorphic, and hence do not "look retarded." Even among children with IQs below 54, many do not "look retarded." (Fig. 1) Signs such as delayed speech, when occurring in a cute child, are often erroneously ascribed to laziness or lack of interest: "He doesn't have to talk because his older brothers and sisters do all the talking for him." The most common cause of delayed speech, however, is not laziness but mental retardation; there are few, if any, "lazy" 2-year-olds. The younger the child with abnormal developmental status, the more likely the cause is to be an organically determined developmental disability rather than an emotional or motivational problem.

Ambulatory Children

Gross motor milestones receive undue emphasis in the diagnosis of mental retardation. The author recently reviewed data on gross motor milestones from his own referral population (Table 1). Twenty-seven children with isolated mental retardation (i.e., no seizures, cerebral palsy, hydrocephalus, orthopedic, or sensory deficit) had been seen by the author for an initial evaluation during the two years preceding this report. The mean IQ of these children was 54.1 (SD = 9.9); the mean age was 19.7 months (SD = 4.4 months). The age of independent walking was selected as a key gross motor milestone. The mildly mentally retarded children (IQ 69–55) had walked at a mean age of 18.0 months, the upper limit of normal. Even the moderate, severe, and profoundly retarded chil-
FIG. 1. Severely retarded 2-year-old child with an IQ of 30. Child gives good eye contact, gazing attentively at parents or physician. Attractive facies and studious countenance are frequently mistaken for normal intelligence ("child seems bright").

Children had walked at a mean age of 21.2 months, only slightly above the upper limit of normal. Therefore, although mentally retarded children often have slightly delayed gross motor milestones, normal gross motor milestones are never sufficient to exclude the diagnosis of mental retardation.

Predictability of IQ Testing

The fallacy persists that certain children are "too young to test" for the presence of mental retardation. This misconception arises from a failure to appreciate the different predictive power of developmental testing in retarded, as opposed to normal, children. It is true that developmental testing of normal children during infancy and early childhood cannot discriminate among those who will go on to perform as dull, average, or bright students in elementary school. In this sense, therefore, there is an age below which normal children may be too young to test. It is also true, however, that developmental testing can distinguish between retarded and nonretarded children, even during infancy (Fig. 2). Once developmental delay is suspected, no child is too young to test.

Opinion

The psychological barrier to even considering the diagnosis of mental retardation in an attractive, attentive, ambulatory young child is very great, but physicians must guard against hastily relying upon the outward appearance of brightness as a substitute for developmental testing. Developmental screening should be a part of routine pediatric care for all preschool children, with referral to a developmental pediatrician or clinical psychologist for formal assessment, once developmental delay is suspected.

References

Recurrent Paroxysmal Supraventricular Tachycardia Complicating Poliomyelitis

Acute anterior poliomyelitis (AAP) is a rare disease in Western countries but still occurs in less developed areas. This unusual case of paroxysmal supraventricular tachycardia (PSVT) complicating AAP is reported as a reminder that the disease, with its cardiac complications remains prevalent.

A 3-year-old unimmunized Bedouin boy, living in the Negev desert, was admitted to the Chaim Sheba Medical Center with ascending paralysis of the lower extremities and low grade fever of two days' duration. On physical examination the patient had flaccid paralysis of both lower extremities. Electrocardiogram (ECG) on admission showed a few atrial premature beats. Poliomyelitis virus type I was cultivated from saliva samples. Progressive respiratory failure, following paralysis of the diaphragm and of the intercostal muscles, prompted artificial ventilation with continuous positive airways pressure (Bonnet ventilator-1'vfA-I) on the 12th day after onset of symptoms. On day 15 development of severe pallor was noted. Body temperature was normal, and systolic blood pressure was 95 mm Hg; ECG showed PSVT at a rate of 210 beats per minute. The PSVT bout was terminated 33 seconds following the intravenous administration of 1 mg of verapamil (0.075 mg/kg body weight). The ECG and the myocardial enzymes were found to be normal following the termination of the PSVT attack. Treatment was started with verapamil orally, 5 mg four times a day. Six days later he experienced an additional attack of PSVT at a rate of 220 beats per minute. Sinus rhythm was resumed 30 seconds following the intravenous administration of verapamil orally and was seen on ECG. Oral verapamil was increased to 15 mg four times daily. No further PSVT attacks were recorded and the ECG remained normal.

A variety of ECG abnormalities have been recorded during the course of AAP, the most frequent being ST-T changes reported in 50.8 per cent of patients in one study and in 44.9 per cent in another. Prolongation of PR, QTC, QRS intervals and T wave changes were also common. However, rhythm disorders of atrial or nodal origin have only been rarely encountered. Only a single episode of PSVT has been previously reported. 


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