Oral-motor dysfunction in children who fail to thrive: organic or non-organic?

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Forty-seven children with non-organic failure to thrive (NOFT) were identified from a whole-population survey of children's growth and development. A significant proportion (N=17) of these 47 children were found to have oral-motor dysfunction (OMD) identified using a previously validated assessment tool. NOFT children with OMD and those with normal oral-motor function (N=30) were compared in order to ascertain whether there were any neurodevelopmental differences which might explain this finding. We hypothesized that children with OMD might have a subtle neurodevelopmental disorder. Few psychosocial variables discriminated the two groups. However, cognitive stimulation within the home and cognitive-growth fostering during mealtimes was much poorer for children with OMD. Some evidence has suggested that NOFT children with OMD may be 'biologically' more vulnerable from birth. We suggest that the continued use of the term 'non-organic' to describe failure to thrive in such children is questionable and requires redefining.

Failure to thrive (FTT) is defined as an abnormally low weight and/or height for age (Skuse 1985). While some children have an identifiable organic aetiology (endocrine deficiencies, congenital, or genetic anomalies), others have no obvious cause underlying their growth failure. Therefore, the term 'non-organic failure to thrive' (NOFT) has been used to describe this latter group.

The origins of NOFT appear more complex than originally proposed and probably involve an interaction between both child and family variables. Undernutrition has been proposed as a common causal factor (Skuse 1985, Ramsey et al. 1993), although it has become increasingly clear that the causal mechanisms vary. While undernutrition may result from failure to offer adequate calories, it may also occur because of inadequate ingestion of food by the infant.

The presence of oral-motor dysfunction (OMD) may prevent some children from achieving a satisfactory nutritional intake. Lewis (1972) proposed that OMD (sucking, chewing, and swallowing difficulties) could contribute to FTT in infancy by leading to prolonged mealtimes and inappropriate environmental features. Selley and Boxall (1986) described 'incoordination of the feeding mechanism' as a cause of FTT. Approximately half the 4 year olds with chronic growth retardation studied by Heptinstall et al. (1987) had some disorder of OMD and had begun to fail to thrive in the first year of life.

Ramsey et al. (1993) saw 38 infants with NOFT and 22 with organic FTT. The histories of the children with so-called NOFT were suggestive of an oral-sensorimotor impairment reported to be present from birth or early infancy. Although an objective assessment of oral-motor functioning was not included in the study, the researchers found many characteristics suggestive of OMD. High proportions of the children studied were reported to have a history of sucking difficulties, abnormal duration of feeding times, poor appetite, delayed texture tolerance, and difficult feeding behaviour. The study concluded that in spite of the fact that no diagnostic label had been applied to the NOFT children, many had histories and developmental profiles suggestive of minimal neurological impairment.

Mathisen et al. (1989) administered a structured oral-motor assessment to nine, 1-year-old infants with NOFT and compared them with a group of healthy children. The infants with NOFT were found to have OMD associated with developmental delay; the OMD was considered to be similar to that seen in neurologically impaired infants. However, only tentative conclusions could be drawn because of the small sample and the fact that the children had been identified for the study by health visitors. In a follow-up study, Reilly et al. (1995) and Skuse et al. (1995) replicated these findings on a whole-population sample of children who were failing to thrive. Significantly more children with FTT were found to have abnormal oral-motor scores than the comparison group of healthy children. Furthermore, when the children with FTT were compared with children of the same developmental age with cerebral palsy, the oral-motor profiles were not remarkably different. Reilly (1995) suggested that these findings added weight to the notion that a subgroup of children previously classified as having NOFT may indeed have a subtle, unidentified, neurodevelopmental deficit and that the term 'non-organic' may be inappropriate.

Further study of the aetiology of OMD in NOFT is significant for several reasons: the identification of an as yet unappreciated organic component to the disorder would alter the

classification and description of the children; and the management of so-called NOFT would be different. Historically, studies of NOFT have focused on the mother's role in providing adequate nutrition and treatment approaches have primarily focused on family dysfunction. As a result, few attempts have been made to ascertain whether the child is able to ingest the nutrients provided.

The aims of this study were: first, to describe the characteristics of the OMD seen in children with NOFT using a standardized schedule to assess oral-motor function; second, to determine whether children with NOFT and OMD were 'neurodevelopmentally different' compared with children with NOFT and normal oral-motor function; and third, to ascertain whether the OMD observed in children with NOFT was part of a global developmental delay and to explore possible causative factors for the NOFT seen in children with normal oral-motor function. We hypothesized that children with NOFT and normal oral-motor skills may come from more disadvantaged homes and experience poorer parenting styles than those who had NOFT with OMD.

Method

The study was part of a prospective longitudinal survey of all infants (2510) registered with participating child-health clinics or family-doctor practices born in one calendar year (1986) in an inner-city health district (population circa 140 000) of London, UK. The district has an ethnically diverse population, which in socioeconomic terms is relatively homogeneous and severely disadvantaged. Detailed information about the design of the study, sample selection, and characteristics of the children can be found in two previous publications (Skuse et al. 1992, 1994).

SUBJECTS

The growth of the 2510 infants who attended participating health clinics and group practices for weighing and developmental checks and who remained living in the district through their first year of life was monitored (Skuse et al. 1992). During this time 14.4% of the population moved out of the area, data were missing on a small number of subjects (1.2%), and a further 3.3% of subjects were untraceable. Further details regarding missing data are available in Skuse et al. (1994).

A set of exclusion criteria was developed to identify potential cases of NOFT. Cases were limited to term (≥ 38 weeks), singleton deliveries, with no known intrauterine growth retardation (birthweights ≤ 3rd centile on charts standardized for length of gestation, sex, ordinal position, maternal height, and mid-pregnancy weight [Tanner and Thompson 1970]). Preterm and low-birthweight babies were excluded because of the known association with below average postnatal growth (Brothwood et al. 1987). Cases of faltering growth were defined as having a weight for age ≤ 3rd centile, with this growth trajectory having been sustained for at least 3 months or more (Tanner and Whitehouse 1984).

There were 1554 potential subjects after these exclusion criteria had been applied. Of these, 52 (3.3%) cases of FTT were identified at 12 months of age. Three were excluded because an obvious identified organic cause was found for the growth failure. The remaining 49 cases of FTT (3.1%) were diagnosed after full paediatric and neurological examination. Two families failed to complete the study. Full details are given in the studies by Skuse et al. (1992, 1994). The final data set comprised 47 children who failed to thrive during the first 12 months of life.

Data were obtained by two methods. First, information was collected retrospectively from hospital or health-clinic records, for example, the anthropometry. Second, each child was assessed by one of the three main researchers (SMR, DHS, or DW). The primary caregiver, usually the child's mother, was interviewed about various aspects of caregiving, the child's history to date, and the child's current behaviour.

The manner in which the sample was recruited is fully described in a variety of publications, including Skuse et al. (1992, 1994, 1995). Briefly, each family was visited at home by SMR and data were usually collected during one home visit, although occasionally two visits were necessary. The home visit pertaining to feeding comprised three parts: first, a semistructured interview with the child's primary caregiver – in most cases the child's mother; second, a video recording was made of the child's main meal of the day; and third, an assessment of oral-motor functioning, using the SOMA, was administered (Reilly and Skuse 1992, Reilly et al. 1995, Skuse et al. 1995).

Table I: SOMA screening version for the OMC category 'purée' (Reilly et al. 1995, Skuse et al. 1995)

Purée
Fromage frais mousse puréed fruit other (Circle choice)

Non-rateable			*Rateable*	
Refused	*Omitted*	*Not observed*	*Yes*	*No*
React 1	Head orientation to food		y	n
Sequence 1	Smooth rhythmic sequence		y	n
Lip 1	Lower lip draws inwards around spoon		y	n
Lip 2	Upper lip removes food from spoon		y	n
Lip 3	Lower/upper lip assist in cleaning		y	n
Lip 11	Lower lip active during suck/munch/chew		y	n
Tongue 11	Consistent/considerable protrusion		y	n
Tongue 12	Protrusion beyond incisors		y	n
Jaw 1	Graded jaw opening		y	n
Sum of shaded boxes				
Cut-off points: >3 indicates oral motor dysfunction			Oral-motor	
<3 normal oral-motor function			dysfunction	

The shaded boxes indicate abnormal responses and are summed to give a total abnormality score.
Range of scores: 0 – 2, normal; 3 – 4 mild dysfunction; 5 – 6, moderate dysfunction; 7 – 9, severe oral-motor dysfunction.

MEASURES

Assessment of oral-motor function

Information on the oral-motor functioning of the subject was obtained from interviews with their mothers about early and current feeding difficulties, and from direct observation by SMR of the subject's behaviour. The screening version of the Schedule for Oral Motor Assessment (SOMA), found to be highly effective in identifying children with OMD, was used (Reilly 1995, Skuse et al. 1995). It entails the standardized presentation of a variety of tastes and textures including purée, semisolids, and solids – these are the oral-motor challenge categories (OMC). Two types of solid categories are administered by SMR, including spooned solids and solids requiring biting and chewing such

Table II: Examples of scoring guidelines for each oral-motor behaviour included in the screening version of the OMC 'purée'. Reilly et al. (unpublished scoring manual)

Variable	*Description of behaviour*	*Scoring*	*Score*
React 1 – head orientation to food	The infant moves his/her head, body or trunk towards the spoon or cup. This movement may involve trunk or head extension or a variety of other movements. The movement should be carefully observed as it may be very subtle in some children	Definite movement of the head, neck or trunk towards the spoon	Yes
		No discernible movement towards the spoon	No
Sequence 1 – smooth sequence	A smooth sequence is one containing a rhythmic suck and swallow. The sequence may consist of one or more sucks and swallows or of a single suck and swallow. There are no obvious coordination difficulties with integrating the suck/swallow sequence	Sequence is smooth and rhythmic	Yes
		Disruption of sequence, uneven pauses, arrhythmic suck/swallow	No
Lip 1 – lower lip draws inward around spoon	Evans-Morris (1982) describes this as part of the process of separation of movement. The lips no longer move in unison with the jaw/tongue as in the younger child. Instead the lower lip moulds around the spoon independently and draws inward to help keep food in the mouth once the spoon is withdrawn	Lower lip moulds or seals around the spoon independently	Yes
		No moulding around spoon. Lips appear immobile and do not move separately from the jaw	No
Lip 2 – upper lip actively removes food from the spoon	The upper lip moves forwards and downward to clean the spoon of food or remove food from the spoon. The lips may fully or partially mould around the spoon and the midpoint of the upper lip makes definite contact. As described above, this is part of the process of separation of movement	Upper lip actively assists in removal of food from the spoon	Yes
		Upper lip does not move independently. No downward or forward movement	No
Lip 3 – lower/upper lip assist in cleaning	The lower and upper lip assist to clean food from the spoon or are actively involved in removing food from the upper or lower lip. For example, the lower lip is moved against the upper teeth or gums or upper lip in order to clean and retrieve small remnants	Upper and lower lip actively remove or clean food	Yes
		No active attempts to clean food or residue from either the upper or lower lip	No
Lip 11 – lower lip active during sucking/chewing/ munching	The lower lip is active during the suck/swallow sequence. During early infant development this movement is not separated from the total movement pattern of the jaw and tongue. However, with the separation of movement that occurs, the upper and lower lip begin to function independently and are capable of a variety and range of movements. This movement may assist in the cleaning process, for example, the lower lip moves against the teeth or the tongue to remove remnants or the lower lip moves independently to stop food spilling from the mouth	Active, independent lower-lip movement during the suck/ swallow sequence	Yes
		No separation or independent movement seen	No
Tongue 11 – consistent/ considerable protrusion	Consistent and considerable tongue protrusion is abnormal, although occasional tongue protrusion throughout a feed is within normal limits. When the tongue protrudes consistently throughout the sucking/munching sequence (that is, more than 50% of the time) this represents an infantile pattern of extension/retraction or may be indicative of the rarer pathological tongue thrust. The tongue may protrude to different degrees, either beyond the lower teeth or beyond the lower lip	Tongue protrudes more than 50% of the time	Yes
		Tongue protrudes less than 50% of the time	No
Jaw 1 – graded jaw opening to accept spoon	The jaw must open sufficiently to accept a loaded spoon. The opening is neither too wide or too narrow an excursion. In young babies this opening is often poorly controlled or exaggerated. Alternatively, in some children with cerebral palsy the jaw excursion may be too narrow and not allow placement of spoon	Well controlled jaw opening to accept spoon	Yes
		Poorly controlled jaw opening/ closure for the spoon/unsteady movements	No

as crackers or biscuits. The OMC categories involving liquids were not used in the screening version of the SOMA because previous research had indicated that they were less sensitive in predicting abnormality and could not reliably be assessed solely by observational means (see Reilly et al. 1995 for details). While most children cooperate with the procedure, some refuse and occasionally it is necessary to instruct parents in the administrative procedure. The assessment is easy to administer and score, taking between 20 and 30 minutes in all. For the purposes of the study, the assessment process was videotaped and behaviour rated later. Full details can be found in the work by Reilly et al. (1995).

Table I gives detailed information about one of the SOMA OMC challenge categories (purée) administered in this study. Nine oral-motor behaviours found to be highly effective in discriminating normal from abnormal oral-motor function (Reilly et al. 1995 and Skuse et al. 1995) made up the screening test for purée. Each behaviour was simply scored either as 'yes' or 'no' in order to avoid confusion: for example, the presence of consistent and considerable tongue protrusion was considered 'abnormal' and its absence, 'normal', whereas the absence of graded jaw opening to accept the spoon was regarded as 'abnormal' and its presence, 'normal'. In Table II, the scoring decisions for the nine behaviours are given. More detailed information regarding scoring decisions can be found in the SOMA scoring manual (unpublished manual available from the authors) and in Reilly (1995). Abnormality scores for each OMC category were obtained by simply summing the shaded boxes. The highest possible abnormality score for purée was 9.

Table III: The Waldrop Scale – examples taken from each group of anomalies and the scoring weights (Waldrop et al. 1968)

Anomaly	*Weight*
Head	
Electric hair	
Very fine hair that won't comb down	2
Fine hair that is soon awry after combing	1
Eyes	
Epicanthus	
Where upper and lower lids join the nose, point of union is	
Deeply covered	2
Partly covered	1
Ears	
Low seated	
Bottom of ears in line with	
Mouth (or lower)	2
Area between nose and mouth	1
Mouth	
High palate	
Roof of mouth	
Definitely steepled	2
Flat and narrow at the top	1
Hands	
Fifth finger	
Markedly curved inward toward other fingers	2
Slightly curved inwards toward other fingers	1
Feet	
Third toe	
Definitely longer than second toe	2
Appears equal in length to second toe	1

After rigorous and detailed analysis (see Reilly 1995 and Skuse et al. 1995) cut-off points, found to be effective in discriminating normal from abnormal, were developed. For example, subjects scoring below the cut-off point of 3 (for the OMC category 'purée' see Table I) were classified as having 'normal' oral-motor function and those scoring 3 or above as having OMD. The cut-off points vary for each OMC category (see Skuse et al. 1995 and Reilly 1995). Abnormal scores (for example, 3 or above for 'purée', see Table I) were then divided into mild (3 to 4), moderate (5 to 6) or severe (7 to 9) OMD for each category (Reilly 1995, Reilly et al. 1996). Finally, a total abnormality score, summed across each of the four OMC categories, was computed.

Anthropometry

Anthropometry at birth (birthweight, length, and head circumference) was obtained from hospital records. Birthweights were standardized for maternal stature, mid-pregnancy weight, length of gestation, ordinal position, and sex according to the method of Tanner and Thompson (1970). Corrected birthweights were then converted to standard deviation scores. Similarly, birth-length standards (Kitchen et al. 1981) and head circumference (Yudkin et al. 1987) were also corrected for sex and gestational age and converted to standard deviation scores.

Growth trajectories from birth were computed from weight data recorded during clinic visits and expressed as standard deviation scores corrected for age and sex (Hamill et al. 1979, Jordan 1986). Scores were interpolated to target ages at 4 weeks, 6 weeks, 3 months, 6 months, 9 months, 12 months, and 15 months of age. Full details are given in Skuse et al. (1994).

Neurodevelopmental factors

Evidence about neurodevelopmental attainments was obtained from a number of sources. Physical anomalies that could be entered into the computation of a congenital anomalies score were recorded during the physical examination undertaken by one of the authors (DHS) (Waldrop et al. 1968). A total anomaly score of 24 was obtained from a list of six groups of possible anomalies (these included head, eyes, ears, mouth, hands, and feet). Within each group, scores were weighted as is illustrated in the examples shown in Table III. An assessment of neurological functioning and maturity was undertaken. Items included in the scale were based largely on the work of Touwen (1976) and Amiel-Tison and Grenier (1986). A composite score of gross and fine motor skills was derived. Skills such as the ability to walk or sit unsupported, visual following while sitting, optical placing, hand function, were scored according to the weighted system recommended by Touwen (1976).

Individual antenatal and perinatal risk scores were computed for each child and included key variables, such as vaginal bleeding during pregnancy, infection during pregnancy, albuminuria with high blood pressure, Apgar scores, fetal distress, and meconium staining of amniotic fluid.

Cognitive and motor development

The Bayley Scales of Mental development (Bayley 1969) were administered to assess cognitive ability. The mental scale contains language items and problem-solving tasks whereas the psychomotor scale mainly addresses gross motor develop-

ment. The cognitive growth-fostering subscale of the Nursing Child Assessment Feeding Scale (NCAFS) was used to assess the quality of parent-infant interaction for stimulating mental development (Barnard et al. 1989). The mother's cognitive stimulation of the infants was rated, double blind, by an independent researcher not involved in data collection. Ratings were made from video recordings of a typical mealtime. The quality of the home environment for fostering child development was obtained using the Home Observations for Measurement of the Environment (HOME) scales (Caldwell and Bradley 1984). The scale is designed to assess the quality of stimulation and support available to the child in the home environment. The infant version was used which comprises 45 binary-choice items clustered into six subscales, including parental responsiveness, acceptance of the child, organization of the environment, play materials, parental environment, and variety of stimulation.

Psychosocial and demographic factors

A range of psychosocial and demographic measures, including socioeconomic status (Osborn Social Index) (Osborn and Morris 1979), family size, social support, birth order, number of siblings, sex, and race were obtained. Full reports of these measures can be found in Skuse et al. (1992, 1994). Mothers also completed the 28-item General Health Questionnaire (GHQ) (Goldberg and Hillier 1979) and the Rosenberg Self-esteem Scale (Rosenberg 1965).

ANALYSIS

The statistical analysis, using SPSS/PC version 6.1 (SPSS Inc., Chicago, IL, USA), included student's *t* tests and correlational analysis for continuous variables, and χ^2 for categorical variables.

Results

The mean age of the 47 children with NOFT (23 males, 24 females) was 14.6 months (SD 1.4, range 12 to 17 months).

ORAL-MOTOR FUNCTION

Table IV shows the proportion of children with NOFT who scored above the threshold for each OMC category, as well as the proportion scoring in the mild, moderate, and severe range. Purée proved to be the most discriminative OMC category with 22 of the children with NOFT scoring above the threshold.

Table IV: Number of NOFT children (*N*=47) with normal oral-motor function and OMD for each of the four OMC categories

Range of scores	*Purée*	*Semisolids*	*Solids*	*Cracker*
Normal	25	37	32	41
Mild	9	0	5	0
Moderate	10	7	0	1
Severe	3	3	10	5

Range of scores: Purée: normal, 0–2; mild, 3–4; moderate, 5–6; severe, 7–9
Semisolids: normal, 0–3; mild, 4–5; moderate, 6–7; severe, 8
Solids: normal, 0–3; mild, 4–5; moderate, 5–6; severe, 7–9
Cracker: normal, 0–8; mild, 9–13; moderate, 14–18; severe, 19–22.

For the purposes of further analysis children were defined as having normal oral-motor function (*N*=30) if they scored within the normal range or had mild OMD. Children with significant (*N*=17) OMD were defined as those scoring in the moderate or severe OMD range. Group comparisons were made on this basis. Abnormality scores for each of the four OMC categories (purée, semisolids, solids, and cracker) and the total SOMA abnormality score (the scores from all four categories summed) differed significantly between NOFT children with OMD and those with normal/mild OMD (see Table V).

ANTHROPOMETRY

There were no significant differences in the children's anthropometry at birth. Children with OMD tended to be shorter, have slightly smaller head circumferences, and slightly lower birthweights (see Table VI). Neither was there a difference in gestational age. Children with OMD tended to weigh less during the first 6 months of life but the differences were not statistically significant. The total SOMA abnormality score did correlate with weight in standardized scores at 3 months but not at any other time.

The postnatal growth of children with moderate to severe OMD (*N*=17) differed slightly from children with normal oral-motor skills in that the children tended to weigh less (particularly during the first 6 months); these differences were non-significant.

NEURODEVELOPMENTAL FACTORS

The gross and fine motor function mean scores for the two groups differed slightly. The children with OMD have slightly higher abnormality scores although the differences were not statistically significant and no individual item discriminated the groups. The mean score for both the fine and gross motor scale are shown in Table VI. The Waldrop scale did correlate significantly with the total SOMA abnormality score (r=0.29, P=0.04) and those children with OMD had significantly higher mean Waldrop scores than those without OMD (see Table VI). Children with OMD tended to have higher prenatal and perinatal abnormality scores (mean 27.71, SD 36.6) than the children with normal oral-motor function (mean 17.35, SD 25.2); this just failed to reach statistical significance.

COGNITIVE AND MOTOR DEVELOPMENT

Correlations between the total SOMA abnormality scores

Table V: Scores for each of the four OMC categories for FTT children with normal oral-motor function and those with OMD

OMC category	*Normal oral-motor function*[a] *(N=30) Mean (SD)*	*OMD*[b] *(N=17) Mean (SD)*	*P*[c]
Purée	1.9 (2.1)	4.1 (2.4)	0.002
Semisolids	0.4 (0.77)	4.4 (3.5)	0.000
Solids	1.4 (2.4)	4.8 (3.5)	0.000
Cracker	1.6 (4.2)	7.4 (9.0)	0.004
Total abnormality score	10.5 (10.5)	25.6 (9.2)	0.000

[a] children with either normal oral-motor function or mild OMD.
[b] children with moderate to severe oral-motor dysfunction.
[c] *t* test for independent samples.

(the total scores summed across all four OMC categories) and the Psycohomotor Developmental Index (PDI) (–0.14, P=0.33) and the Mental developmental index (MDI) (–0.19, P=0.19) of the Bayley Scales of Mental Development (Bayley 1969) were non-significant. The mean scores on both the Psychomotor (mean 92.9, SD 18.2) and Mental Scales (mean 94.7, SD 16.2) were slightly lower in children with OMD than among those with normal oral-motor function (PDI mean 98.8, SD 16.6; MDI mean 100.2, SD 20.3) but these differences were not statistically significant. There were no significant correlations between MDI and PDI Bayley Scale scores and the scores for each of the individual OMC categories (purée, semisolids, solids, and cracker).

PSYCHOSOCIAL AND DEMOGRAPHIC FACTORS

A range of measures such as socioeconomic status (Osborn Social Index) (Osborn and Morris 1979), family size, and total adversity score obtained by summing individual items revealed no significant differences between children with OMD and those with normal oral-motor function. There were no significant differences in mother's health as measured by the 28-item General Health Questionnaire (Goldberg and Hillier 1979) and the Rosenberg Self-esteem Scale (Rosenberg 1965).

There was little variation in the mean scores for the majority of the HOME subscales. However, for the Acceptance subscale (avoidance of restriction and punishment) there was a statistically significant difference; children with OMD had lower scores (mean 3.2, SD 2.4) and were less accepted by their mothers than children with normal oral-motor function (mean 5.3, SD 2.4) (P<0.001).

Observational ratings of the cognitive-growth fostering during mealtimes are reported in Table VII. Although both groups had a striking lack of verbal interaction, there was a tendency for mothers of children with OMD to be less responsive, i.e. they did not respond with the same frequency to their child's verbalizations or gross motor movements.

Table VI: Comparisons between children with and without OMD on a number of perinatal and postnatal variables

Variable	*Normal oral-motor function (N=30) Mean (SD)*	*OMD (N=17) Mean (SD)*	*P*[a]
Perinatal and antenatal factors			
Birth length (SD scores)[b]	0.24 (1.0)	–0.41 (1.3)	ns, 0.09
Head circumference at birth (mm)[b]	–0.81 (0.93)	–0.98 (1.1)	ns
Ponderal index[b]	0.23 (0.03)	0.25 (0.04)	ns
Birthweight (SD scores)	–0.63 (0.65)	–0.97 (0.77)	ns
Prenatal and perinatal index[c]	17.3 (25.2)	27.7 (36.5)	ns, 0.09
Gestation (wk)	39.23 (1.0)	39.6 (1.0)	ns
Developmental assessment			
Gross motor scale[d]	20.1 (4.4)	21.3 (3.9)	ns
Fine motor scale[d]	7.0 (2.2)	7.8 (1.6)	ns
Waldrop scale[e]	1.2 (1.1)	2.6 (2.9)	0.04

[a] *t* test for independent samples.
[b] Numbers vary because some items had some missing data.
[c] Mean scores on the risk index of perinatal and antenatal factors.
[d] Gross and fine motor scale compiled from Touwen (1976) and Amiel Tison and Grenier (1986).
[e] Waldrop scale, a method of assessing minor physical anomalies in young children – see Table III (Waldrop et al. 1968).

Discussion

A subgroup of children with NOFT were shown to have clinically significant oral-motor dysfunction, using an assessment schedule previously shown to be both reliable and valid. This study is the first whole-population survey, using direct assessment and observation, to show that a substantial number of children previously described as having NOFT, in fact, have significant OMD. The children did not necessarily have problems eating an isolated food texture but tended to have abnormal scores on more than one OMC category. The results raise some important issues.

GLOBAL DEVELOPMENTAL DELAY

One possible explanation proposed for the OMD seen in the FTT children with OMD could be that they are developmentally delayed. Skuse et al. (1992, 1994) and Wilensky et al. (1996) have shown that children with FTT had significantly lower scores on the Bayley Scales of Mental Development than a matched comparison group of children with normal growth. However, the FTT children with OMD had only marginally lower scores on both the MDI and PDI of the Bayley

Table VII: Ratings of cognitive-growth fostering during meal times as measured by the NCAFS (Barnard 1978)

Variable	*Normal oral-motor function (N=30)*[a,b]	*OMD (N=17)*[a,b]	*P*[c]
Maternal behaviour			
Provides child with objects, finger-foods, toys, and/or utensils	19	13	ns
Encourages and/or allows child to explore food/utensils or parent during feeding	21	14	ns
Talks to child using two words at least three times during session	27	13	ns
Verbally describes some aspect of food or feeding situation	12	2	0.04
Talks to child of things other than food	10	2	0.02
Uses statements that describe, ask questions, or explain consequences of behaviour more than commands	15	8	ns
Verbalizes to child within 5 s of child vocalization	13	4	ns[d]
Parent verbalize to child within 5 s after child's movement of arms, legs, hands, head, trunk	10	1	0.03
Parent does not talk baby talk	27	15	ns
Other mealtime variables			
Mean mealtime duration (s) (SD)	1119.2 (670.8)	1582.0 (1008.8)	ns[d,e]

[a] Number of 'yes' answers for each item are shown for both groups.
[b] Numbers vary slightly as there was a small amount of missing data for some items.
[c] χ^2 test.
[d] P=0.06.
[e] *t* test for independent samples.

Scales and these differences were not statistically significant. Even those children with moderate to severe OMD were not found to have a global developmental delay. These results do not support the theory that the OMD was part of a global developmental delay.

For many years clinicians and researchers believed that the ability to manage different food textures followed a strict developmental sequence with liquids and purées being the 'easiest' textures to ingest, followed by thick and lumpy foodstuffs until the child was able to chew and manage a full adult diet. However, more recent evidence has suggested that the oral-motor skills necessary to ingest puréed foodstuffs were the last to mature and in fact might not fully mature until well beyond 2 years of age (Gisel 1992). We analysed each OMC separately in this study to see whether there were any correlations with developmental age. However, our results did not reveal any differences in developmental factors as measured by the Bayley Scales (Bayley 1969). Some clinicians and researchers (for example, Ramsey et al. 1993) have suggested that OMD might occur as an isolated neurological sign that only becomes apparent in some children when solids are introduced, requiring a more organized and mature feeding pattern. Such difficulties could affect the child's ability to achieve an adequate intake and presumably result in FTT.

We believe these findings add further support to the theory that the aetiology of the OMD seen in a subgroup of infants with FTT cannot be attributed solely to developmental delay.

DIFFERENCES BETWEEN CHILDREN WITH AND WITHOUT OMD

There are no significant results in this study to support the theory that children with OMD differ subtly from those with normal oral-motor function, although there are some trends among the data. Children with OMD tend to be of lower birthweight, have reduced length at birth, and smaller head circumferences, and the more severe the OMD the greater the difference in perinatal and postnatal growth. Prenatal and perinatal risk scores were higher for those children with OMD, although the differences were non-significant. However, given the power to detect differences of our small subsample size, this could suggest a 'biological vulnerability', a theory proposed by Altemeier et al. (1985) and discussed more recently by Wilensky et al. (1996). Children with OMD also had significantly higher scores on the Waldrop scales. Waldrop et al. (1968) and Waldrop and Halverson (1971) suggested that an inverse relation exists between intellectual functioning and the number of anomalies in young children. Therefore, it seems reasonable to ask if the minor congenital anomalies identified could be consistent with a subtle neurodevelopmental problem.

Ramsey et al. (1993) suggested that almost half the children they assessed with NOFT had a history of questionable 'neurological involvement' in the absence of any diagnosable condition. Recently Wilensky et al. (1996) found that significantly more of the children they studied with FTT had a history of hypotonia reported in the first year of life compared with normal infants. The gross and fine motor function measures used in our study did not significantly differentiate FTT children with OMD from those with normal oral-motor function, although children with OMD did have slightly higher abnormality scores. It is possible that there were subtle differences between the two groups and had we undertaken a more rigorous and detailed neurological examination, these may have been identified. Increased 'biologic vulnerability', demonstrated in FTT children with OMD, may be dependent on more detailed and earlier examination of the child to identify possible neurodevelopmental risk factors. Follow-up studies currently in progress may also contribute valuable information about the children's achievements and developmental outcome.

CHILDREN WITH NORMAL ORAL-MOTOR FUNCTION WITH FTT

A range of psychosocial and demographic factors were used in this study. We hypothesized that one causal mechanism for the FTT in children with normal oral-motor function could be psychosocial deprivation. However, there were no major differences between FTT children with normal oral-motor function and those with OMD in the demographic characteristics or in many of the psychosocial variables we used. We hypothesized that children with FTT with normal oral-motor function might come from more disadvantaged homes, with less stimulating and responsive environments and that this may in part explain why they failed to thrive. However, there were no differences in demographic factors such as socioeconomic status, adversity scores, and social support mechanisms. One subscale of the HOME scale did discriminate between the two groups, but not in the expected direction: the children with OMD who were given fewer opportunities for exploring and playing and may have been exposed to more restrictive behaviour and punishment. Clearly this finding requires more detailed investigation. Given the multiple comparisons made within this study, this result should be interpreted with caution as it may be spurious.

Differences were found in cognitive-growth fostering during mealtimes showing that mothers of children with OMD were less likely to talk to their children during meals and did not respond to their child's physical cues. We had predicted that the children with normal oral-motor function would have less interactive environments and it is interesting to speculate as to the reasons for these unexpected findings. Feeding is the first 'joint task' of the mother–infant relationship and any interruption of this delicate relationship, such as a 'difficult feeder' may place extra demands and stresses on both the child and mother and so affect interactional style. Ramsey et al. (1993) found that almost half the mothers of children with FTT exhibited negative effect and interactions during feeding time and suggested that the early feeding impairments may trigger the development of maladaptive interactional patterns. Lindberg et al. (1996) studied mother–child interaction when children refuse food and found that the more difficult the infant, the less responsive the mother was to the child's cues. Our revised hypothesis is that the presence of OMD provokes a 'maladaptive' maternal style of interaction in mothers who may be otherwise marginally able to cope.

The concept of FTT has undergone some radical changes since the 1940s when it was first suggested to be caused by emotional deprivation. Current thinking suggests that the term 'non-organic failure to thrive' should be revised. Some children may in fact have an underlying but not readily identifiable organic cause. It has been proposed that different mechanisms cause FTT. Therefore, different aetiologies may underlie the condition.

In this study we identified a subgroup of children with clinically significant OMD which may contribute to their

FTT. Our results did not support the coexistence of a subtle neurodevelopmental disorder which might explain the OMD, although trends in the data suggest that a subgroup may be more vulnerable from birth, and determining this would only be possible by a prospective study which investigated oral-motor function in the context of the child's overall development. Valuable data might also be gained from studying high-risk infants such as the very preterm infant at risk of growth failure. However, there is no doubt that other aetiological factors are of great interest; while for a subgroup 'biologic vulnerability' might be a causative factor, in others it has been proposed that environmental factors play an important role. In this study we found no evidence to suggest that the children with normal oral-motor skills were raised in an environment in which they were neglected and with less stimulation.

This study highlights the importance of carrying out detailed multidisciplinary paediatric examination of the infant who is failing to thrive. Less than 20% of the children with FTT in this sample had been referred for further investigation and as a result the OMD had failed to be identified as a contributing factor. To identify the subtle but significant OMD observed in children with NOFT it is vital to include both a thorough neurological and developmental assessment as well as an evaluation of oral-motor function. Such assessments are crucial in order to apply the appropriate management strategies.

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