The Robin anomalad (Pierre Robin syndrome) —a follow up study

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SUMMARY During a 10-year period 55 patients with the Robin anomalad were admitted to the Liverpool Regional Cleft Palate Units. Fourteen (26%) children died. All deaths were within 3 months of birth. Congenital abnormalities other than mandibular retrognathia and cleft palate were present in 14 (26%) children. Peripheral limb defects were particularly common. Thirty children were recalled and reviewed to assess speech, hearing, growth, and educational achievement. There was a clear association between severe nasal escape of air in speech and atypical articulatory patterns. Almost half the children tested had abnormal articulation. Only 4 (13%) of 30 children showed delayed language development. Half the children tested audiometrically showed a binaural handicap but in only one patient was this sufficiently severe to warrant amplification. There was no trend towards abnormalities of growth and only 2 children could be firmly classified as educationally subnormal.

Although the problems of retrognathia in infancy were recognised by Virchow as early as 1864¹ the credit for the full description of the anomalad embracing mandibular retrognathia and glossop-tosis is given to Pierre Robin.² Palatal abnormalities, particularly clefts, are recognised generally as forming part of the complete anomalad. There are good accounts of the disorder although the emphasis tends towards aetiology and treatment.^{1 3-7} We know of no follow-up study of a large series. We describe here the clinical features and subsequent developmental progress of 55 British children with the Robin anomalad, including cleft palate, followed over a period of between 1 and 10 years after birth.

Patients and methods

During the 10-year period July 1966 to June 1976, 55 patients with mandibular retrognathia, glossop-tosis, and cleft palate (Robin anomalad) were admitted to the Liverpool Regional Cleft Palate Units at Alder Hey Children's Hospital and the Royal Liverpool Children's Hospital, Heswall. Thirty children were recalled and assessed by a paediatrician (AJW), a speech therapist (MAW), an educational psychologist (CAW), and an ortho-dontist (PGB) with informed parental consent.

Fourteen children had died and 11 could not be traced or declined to attend. The case records of this group of 25 were reviewed and we sought educational information by questionnaire on the 11 surviving children not formally assessed.

Results

Fourteen (26%) of the 55 children had died. All deaths had been within 14 weeks of birth. Of the 14 who died, 10 (71%) had additional congenital abnormalities (Table 1). Two deaths were considered a result of concomitant congenital heart disease. Four children were shown to have bronchopneu-monia at necropsy although in only one was there a clear history of aspiration of feeds. Two children died suddenly and unexpectedly in hospital at age 2 months and no clear cause of death could be found

Table 1 *Additional abnormalities in 10 children dying with the Robin anomalad*

Abnormality	No
Limb deformities	5
Other skeletal abnormalities	1
Congenital heart disease	3
Renal abnormalities	1
Ocular abnormalities	2

at necropsy. The other causes of death were septicaemia (1), pulmonary haemorrhage (1), transverse sinus thrombosis (1), and atelectasis (1). Necropsy examination was not performed in 2 children.

Only 4 (10%) of the 41 survivors had other abnormalities (Table 2).

The cleft palates of the surviving group were repaired between 12 and 24 months (mean 15.8). The mean length of hospital stay for the same group was 113 days (range 28–206). One child was excluded because his hospital stay was prolonged for over a year for social and legal reasons.

Assessment of 30 children recalled. The age range of this group of children was 10 months to 11 years.

Speech

Each child was assessed in terms of language, articulation, and resonance.

Language. Seventeen children aged 10 months to 6 years were tested using the Reynell developmental language scales (RDLS) experimental edition.⁸ One showed delayed development of comprehension, defined as being 2 SD below mean for age, together with delay in expressive language. One other child showed delay in expressive language only. Thirteen children over age 6 years were tested by subjective assessment with composite pictures to stimulate conversation. Two of these 13 showed unequivocal severe delay in language skills both in fields of comprehension and expressive language. One child showed an immature pattern and the remaining 10 had normal language skills.

Articulation. The Edinburgh articulation test (EAT)⁹ was used on 25 children (Table 3). This test is standardised for children of 3–6 years only but is useful in defining articulatory defects in a wider age range. Four children were seen before palatal surgery and could not be assessed and one child had insufficient language for testing.

Nasal resonance. Nasal escape of air in consonants in speech was assessed subjectively on the scale nil-mild-severe. It was not feasible to test the 4 children seen before surgery and one child had

Table 3 *Articulatory patterns of 25 children tested with the Edinburgh articulation test*

	Adult form of articulation	Immature articulation	Atypical substitution
Standardised EAT group	1	4	7
EAT used in children over 6 years	3	6	4

EAT = Edinburgh articulation test.

insufficient language for assessment. Of 25 children tested, 5 showed mild and 7 severe nasal escape. All patients with severe nasal escape had atypical articulatory patterns.

Hearing

Audiometry was undertaken on 26 children and a further 4 children had free field screening tests. This includes results from some children retested recently. In the group tested audiometrically hearing loss was calculated for each ear in each child by taking the average of the summated loss in decibels (dB) at 500 Hz, 1 kHz, and 2 kHz. The monaural loss is defined as 1.5% for every decibel by which the average exceeds 26 dB. The overall (binaural) loss is obtained by combining the two ears using the formula:¹⁰ % binaural handicap = (% monaural loss better ear × 5 + % monaural loss worse ear × 1)/6. Using this formula, 12 (46%) of 26 patients tested showed binaural handicap. Only one of these patients recorded a binaural handicap of greater than 20%.

Educational assessment

In the preschool age group (0–60 months) the developmental level was matched against standardised norms. In the school age group (60+ months) basic numeracy, literary skills, and general intellectual ability were examined. All assessments were carried out in the presence of a parent.

Preschool group (n = 11). The Griffiths's scale was used for assessment and applied rigorously where possible.^{11 12} If a child was unable to co-operate (for example one had a plaster cast encasing both legs) the scale was completed by careful observation and details of performance obtained by questioning the parents. In this small sample the data suggested that the children's development profiles were within the range expected of normal children in the same age group (Table 4). There was an indication of poorer scoring in hearing and speech subscales although the sample is too small for statistical analysis.

School age group (n = 19). These children were

Table 2 *Additional abnormalities in 4 surviving children with the Robin anomalad*

Abnormality	No
Limb deformities	3
Other skeletal abnormalities	1
Congenital heart disease	1
Ocular abnormality	1

Table 4 12 children tested on the Griffiths's scales

Case	Age at examination (months)	Developmental age (months)				
		Locomotor	Personal/social	Hearing and speech	Eye/hand Co-ordination	Performance
1	13	9	13	14	13	12
2	10	NC	10	12	10	9
3	15	19	18	14	17	13
4	16	19	17	14	18	15
5	15	18	17	14	17	NC
6	20	24	20	16	24	22
7	27	30	28	24	24	28
8	30	34	34	34	34	34
9	30	30	30	22	NC	NC
10	40	48	48	48	48	NC
11	49	54	56	46	37	42
20*	83	52	56	48	48	50

*Tested on Griffiths's scale as unable to complete WISC tests.
NC = not completed.

given a range of tests: the Bender test with Koppitz scoring,^{13 14} Wechsler intelligence scale for children (WISC) coding subtest, WISC digit span, WISC arithmetic subtest,¹⁵ English picture vocabulary test,¹⁶ Daniels and Diack reading age test 1,¹⁷ and the Columbia mental maturity scale¹⁸ (Table 5).

The length of the interview was 40–50 minutes. One child of school age was excluded as he was unable to understand the instructions and instead the Griffiths's scale was used. Of the 18 children

remaining, one produced depressed scores on all tests and was receiving special education at the ESN (M) level, one child with poor attainments was having remedial support within an ordinary school, and a third child did not co-operate with testing and was thought likely to be of limited ability.

Vision

Five (17%) of the 30 children were noted to have a strabismus on examination. Visual acuity was

Table 5 18 children tested on various attainment scales

Case	Age at test (years)	Bender test	EPVT	Daniels and Diack reading age test 1	CMMS	Wechsler intelligence scale for children		
						Arithmetic Subtest	Coding Subtest	Digit span
12*	5.4	Perceptual difficulty	73	Not scored	Not completed	Not scored	Not scored	Not completed
13	5.7	No problems	115	100	99	6.10	16	12
14	5.9	No problems	84	Not scored	84	Not scored	11	8
15	5.9	Tension	111	89	131	7.10	12	12
16	6.0	Tension	92	90	82	Not scored	9	8
17	6.0	Tension	98	Not scored	91	5.10	12	5
18	6.6	No problems	140	160	132	8.10	9	9
19	6.9	No problems	93	85	90	7.10	16	9
21	7.3	No problems	76	100	Not completed	9.6	8	13
22	7.7	No problems	108	115	123	8.6	16	16
23	7.8	No problems	92	99	102	6.2	12	14
24	8.3	No problems	78	105	Not completed	11.2	8	11
25	8.3	No problems	90	104	102	10.6	13	15
26	8.11	No problems	93	127	Not completed	12.2	17	14
27	10.1	No problems	92	138	Not completed	12.2	11	11
28	10.1	No problems	104	139	Not completed	12.2	7	15
29†	10.5	No problems	60	68	Not completed	8.10	11	12
30‡	11.2	No problems	<60	Not scored	Not completed	5.10	4	4

*Did not co-operate during test, †receiving education at ESN (M) school, ‡receiving remedial help in normal school.
EPVT = English picture vocabulary test, CMMS = Columbia mental maturity scale.

examined using Snellen's charts in all children over age 5 years. No loss of acuity was noted in children without strabismus.

Growth

Height, weight, and head circumference were measured in all children. Two children were more than 3 SD below the mean height for age.¹⁹ One patient had no other obvious skeletal problems but had been substantially small for gestational age. The second short child had bilateral short femora and was also more than 3 SD below mean weight for age. One other patient was more than 3 SD below the mean weight for age for which no cause could be found. All children had appropriate head circumferences for age.

Family history

Seven (23%) children of the 30 reviewed had a close relative with cleft palate, cleft palate with cleft lip, or isolated cleft lip (Table 6). One of these children had a brother with the Robin anomalad. (He is not included in this series as he was born outside the 10-year study period.)

Maternal history

Two mothers had had a significant medical illness during pregnancy. One had had pre-eclamptic toxæmia treated with an unspecified hypotensive agent, and the other had had asthma treated with disodium cromoglycate. Five mothers admitted taking drugs other than iron and folic acid during their pregnancies. These drugs were Distalgesic (1), Stelazine (1), and drugs of the benzodiazepine group (3). One mother took diazepam for 2 months in early pregnancy. All the other benzodiazepine prescriptions were for short periods in the 3rd trimester. No other anticonvulsant preparations were taken.

Treatment

Twenty-two (73%) out of the 30 children reviewed were nursed prone on a specially designed cradle.²⁰

Table 6 Cleft palate or cleft lip in close relatives of patients with the Robin anomalad

Case	Cleft palate or cleft lip	Relative
2	CP	Father
10	CP/CL	Uncle
12	CP	Mother
13	CL	Cousin
16	CP/CL	Father
23	CP	Aunt
23	Robin anomalad	Sibling
24	CL	Cousin

CP = cleft palate, CL = cleft lip.

Table 7 Incidence of associated anomalies and mortality with Robin anomalad

	Total cases	Mortality		Cardiovascular		Limb	
		No	%	No	%	No	%
Routledge (1960)	18	6	(33)	2	(11)	3	(17)
Smith & Stowe (1961)	39	—	—	6	(15)	8	(21)
Forrest & Graham (1963)	25	5	(20)	3	(12)	—	—
Monroe & Ogo (1972)	65	14	(22)	14	(22)	7	(11)
Hanson & Smith (1975)	28	—	—	3	(11)	6	(21)
Williams et al. (1980)	55	14	(26)	4	(7)	8	(15)
Current Study							

Only 1 child of the 30 required a tongue suture. No other surgical procedures were undertaken. Details of the non-operative treatment will be the subject of a separate publication.

Discussion

Robin anomalad is the preferred term for the Pierre Robin syndrome²¹ and has been used throughout this paper.

The mortality rate in this series (26%) is similar to rates previously reported (Table 5). Pneumonia, with or without aspiration of feeds, was not unexpected and sudden death in hospital without an identified cause has been described by others.^{5 6}

Limb abnormalities are commonly associated with the anomalad.^{4 22 23} Our study confirmed this. The abnormalities included talipes equinovarus, syndactyly, short digits, absent digits, and hypoplastic ulna. Two children had short femora. A syndrome of femoral hypoplasia and unusual facies has been reported.²⁴ Micrognathia and cleft palate are said to be commonly associated with this syndrome but severe glossoptosis is rare. Neither of our 2 patients could be described as having a facies other than typical of the Robin anomalad. It has been suggested that about a quarter of patients might have the Robin anomalad as part of a specific syndrome.²³ Apart from the possibility of femoral hypoplasia—unusual facies syndrome, we could not identify other syndromes in the group of children reviewed. Cardiovascular system abnormalities have often been described in association with the anomalad and with facial clefts generally.²⁵ The reported incidence has been as high as 20%.²⁶ Although seen less frequently than other system abnormalities in our series, cardiovascular lesions should be sought in all children with the anomalad.

Ocular involvement was first recognised in 1961 by Smith and Stowe.⁴ Subsequent estimations of frequency of ocular problems have been about

10%.^{26 27} Abnormalities described include microphthalmia, strabismus, cataract, ptosis, and congenital glaucoma. Retinal detachment may occur alone or as part of the syndrome of hereditary progressive arthro-ophthalmopathy (Stickler syndrome).²⁸ Congenital ptosis, microphthalmia, and a severe congenital strabismus were the only major ocular abnormalities present in the 30 children reviewed and in the 14 children who died. Ophthalmological review is essential in children with the Robin anomalad.

An increased incidence of short stature in children with cleft palate has been reported.²⁹ Growth hormone deficiency has also been described.³⁰ We did not confirm an increased incidence of short stature in our patients.

Subsequent normal mandibular growth is well known³¹ and this was a feature of all older patients in our survey.

A high proportion of children with cleft palate have articulatory difficulties. Fricative (for example 'f' 's' 'sh') and plosive (for example 'p' 't') sounds are particularly affected, nasal sounds (for example 'm' 'n' 'ng') are usually unimpaired.³² In our study 11 (44%) of 25 children tested with a standard test of articulation showed atypical substitutions of speech sounds. The articulatory patterns were identical with those observed in children with isolated cleft palate. Fricative sounds were invariably incorrectly produced and plosives and affricates (for example 'ch' 'j') frequently impaired. Language was the least affected communication skill. Only 4 of 30 children tested showed language delay, 2 of whom were receiving special educational help. We conclude that the presence of mandibular retrognathia does not appear to prejudice the speech skills of children with Robin anomalad compared with children who have an isolated cleft palate. A wide variety of methods has been used in the assessment of velopharyngeal competency in cleft palate. These include lateral cephalometry, video-fluoroscopy, nasendoscopy, and anemometry. Apart from the first-named which is probably of limited value, these techniques are not routinely available in the UK. For this reason a simple clinical assessment of nasal resonance was used in this study. Almost half of the children tested had mild or severe nasal escape of air in speech. This correlates well with previous reports.³³ Our study showed the expected clear association between atypical articulatory patterns and velopharyngeal incompetence as judged by nasal escape of air in speech. Adequacy of velopharyngeal closure is paramount in the development of normal articulatory patterns. Moreover it has been suggested that speech therapy is ineffective in improving articulatory skills in velopharyngeal incompetence.³⁴

Given the frequency of this problem in children with cleft palate, efforts towards routine accurate ascertainment of the degree of incompetence in young children should be vigorously pursued. At present expertise in this field is limited to a few centres in the UK, but expansion of these facilities would allow earlier identification of patients requiring further palatal surgery to overcome articulatory difficulties.

Over half of the adult cleft palate population has a conductive hearing loss.³⁵ The criteria for diagnosing hearing loss vary greatly. We have used the widely accepted AAO classification which uses frequencies found on all audiometers. However, it does not use high frequency tones which might impair interpretation of speech in certain circumstances. Though binaural handicap was identified in half of the patients reviewed, only one child had a binaural handicap greater than 20% which is about the level at which amplification is needed in everyday speech. Middle-ear function improves after palatal closure³⁶ and those children with hearing loss tested soon after operation may subsequently improve. It has been suggested that an increased incidence of hearing loss is associated with later repair of palatal defect.³⁷ However, different surgical procedures in palatal closure were used within each age group in that study. All patients in our series had a procedure of closure with lengthening and we did not confirm the findings of Masters *et al.*³⁷ The incidence of hearing impairment in Robin anomalad is similar to that in isolated cleft palate. Regular audiometric evaluation is mandatory from an early age as it has been shown that middle-ear disease in association with cleft palate develops soon after birth.³⁸ Adenoidectomy should be avoided because of the possible adverse effect on velopharyngeal closure. We found no congenital ear anomalies.^{4 26}

Some authors have reported a high incidence of mental retardation in children with the Robin anomalad.^{4 39} We did not confirm this. Only 2 (7%) of 30 assessed could be firmly classified as educationally subnormal. Of the 11 surviving children not formally assessed, 6 responded to a questionnaire and were receiving normal education. In the unlikely event that all 5 non-responders were receiving special education the overall incidence of mental retardation in this series would still be well below the 25% incidence noted by Smith and Stowe.⁴

Although the exact pattern of inheritance in the Robin anomalad is poorly defined there seems little doubt that hereditary factors are important. Eight (27%) of 30 children in this series had near relatives with cleft lip, cleft palate, both of these, or the Robin anomalad. Estimates of frequency of affected relatives have varied from 8%⁴ to 32%.⁴⁰ All these

reports describe the anomalad in siblings of an index case. Appreciation that the anomalad may be part of a wider recognised syndrome may allow more accurate prediction of a risk of recurrence.

One should avoid using the term Robin anomalad to describe every infant with a cleft palate and a small chin. We believe that respiratory difficulty is an essential feature whether it occurs soon after birth as in all our patients or later.⁴¹ When appropriately defined the anomalad implies a significant risk to neonatal life in the absence of experienced nursing care. In our experience, in the absence of a specific syndrome, a satisfactory intellectual and physical outcome can be expected if the infant survives the neonatal period.

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