The Association of Fetal Wastage with Facial Cleft Conditions

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The variation seen in the severity of some congenital malformations suggests that the genetic determinants of these malformations may have a range of effects. It is not unreasonable to expect that this range may extend to lethality (*13, 23*). Indeed, there is good evidence that mothers of anencephalics have an increased frequency of spontaneous abortions among their other pregnancies (*2, 6, 23*). The substantially elevated incidences of malformations found in spontaneous abortions (*22*), therapeutic abortions (*18*), and stillbirths (*4, 16*) provide further evidence of the reduced prenatal viability associated with such defects.

That fetal wastage in the sibships of propositi with a particular type of congenital malformation should be low seems unlikely. The present paper presents evidence suggesting a reduction of fetal wastage in the sibships of certain classes of facial cleft propositi, and examines the implications of this possible reduction.

Data Collection

Family pedigrees and maternal reproductive data were collected for 346 propositi with cleft palate (CP), cleft lip (CL) and cleft lip and palate (CL+P). Propositi were children attending for review at plastic surgery out-patient clinics held at the Fleming Memorial Hospital for Sick Children, Newcastle upon Tyne. Data collection was by interview of the relative, usually the mother, accompanying the child to the clinic. As most of the propositi returned for semi-annual or annual review, information obtained at the initial interview could usually be amplified at subsequent interview. The occurrence in propositi of defects other than facial clefts was determined from examination of the hospital notes on each case. These cover diagnosis of facial cleft, plastic and reconstructive surgery, diagnosis and treatment of additional clinical conditions, and continuing semi-annual and annual review. Their completeness with respect to the recording of additional defects in propositi is therefore regarded as quite adequate.

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In a small proportion of cases a facial cleft occurs as part of a recognized syndrome of congenital malformation (*11*). Some of these syndromes (primarily Downs' syndrome in living cleft patients) are caused by chromosomal abnormalities; others are of monogenic etiology. So that analyses may relate to conditions as homogeneous as possible with respect to etiology, the 16 such syndrome-associated facial clefts in the present series (8 cases of Pierre Robin syndrome, 3 of Klippel-Feil, 2 of Down's, 2 of lip pit syndromes and 1 of Crouzon's syndrome) are excluded from further consideration. Also excluded is a case of median cleft lip, since the embryonic growth failure resulting in this condition is distinct from that of the more common lateral lip clefts. The findings of the present study thus relate to 329 propositi whose facial clefts are typical and not part of a syndrome of malformation.

Results

Table 1 presents the numbers and frequencies of spontaneous abortions, stillbirths, and neonatal deaths observed in the sibships of propositi. Excluded from the tabulation of abortions are 2 ectopic pregnancies, 2 suppressed twins of liveborn sibs, 2 'suspected' miscarriages specified as such by the informants, 1 hydatidiform mole, and therapeutic terminations. Neonatal deaths do not include 5 infant deaths from infectious disease (pneumonia and meningitis). Twins have been counted as 2 liveborn sibs and in one instance as two abortions. Only full sibs have been tabulated.

The overall incidence of abortion found in the sibships of propositi is low—10.5 $\pm$ 1.2%. Using retrospective interview methods comparable to those of the present study, Boon et al. (*3*) found 15.4% of the pregnancies of mothers of 100 control children from Newcastle upon Tyne resulted in abortions. Similarly, Warburton and Fraser (*23*) found the incidence of abortion to be 14.7 $\pm$ 0.4% in a Canadian series of 2134 sibships.

The overall incidences of stillbirth and neonatal death in the sibships of propositi are 1.2% and 2.3% respectively. These values are comparable with 2.3% stillbirths and 1.6% neonatal deaths found by the 1958 British Perinatal Mortality Survey (*4*) in a series of 17,204 births.

The occurrence of abortions and fetal wastage in sibships is not uniform with regard to sex and cleft type of the propositi. Fetal wastage of all types is less common in the sibships of male as opposed to female propositi ($\chi^2 = 4.62$, $P < 0.05$), although the deficiency of abortions considered alone falls short of formal significance ($\chi^2 = 3.38$, $P < 0.10$). (Numbers of abortions have in every instance been compared with numbers of surviving sibs in computing χ^2 statistics.) This overall disparity results from the very low incidences of abortion and fetal wastage observed in the sibships of males with CL and CP. The deficiency of fetal wastage is highly significant in the sibships of CP propositi: comparing abortion incidences in the sibships of male v. female CP propositi, $\chi^2 = 5.96$, $P < 0.025$; comparing fetal wastage incidence $\chi^2 = 6.96$, $P < 0.01$).

TABLE 1. Fetal wastage in the sibships of propositi.

propositi	*CL*			*CL+P*			*CP*			*all clefts*		
	male	*female*	*total*	*male*	*female*	*total*	*male*	*female*	*total*	*male*	*female*	*total*
	52	*33*	*85*	*91*	*56*	*147*	*30*	*67*	*97*	*173*	*156*	*329*
brothers	56	27	83	85	51	136	29	56	85	170	134	304
sisters	31	33	64	74	47	121	21	56	77	126	136	262
stillbirths	0	2	2	3	3	6	0	0	0	3	5	8
frequency	—	0.029	0.012	0.016	0.025	0.020	—	—	—	0.009	0.015	0.012
neonatal deaths	3	3	6	3	3	6	0	3	3	6	9	15
frequency	0.033	0.043	0.037	0.016	0.025	0.020	—	0.022	0.016	0.018	0.028	0.023
spontaneous abortions	2	5	7	25	14	39	1	22	23	28	41	69
frequency	0.022	0.071	0.043	0.132	0.119	0.127	0.020	0.161	0.122	0.084	0.126	0.105
total wastage	5	10	15	31	20	51	1	25	26	37	55	92
frequency	0.054	0.143	0.093	0.163	0.170	0.166	0.020	0.183	0.138	0.111	0.169	0.140
total pregnancies exclud-propositi	92	70	162	190	118	308	51	137	188	333	325	658

Observed incidences of abortion and fetal wastage in the sibships of male propositi vary markedly with respect to cleft type in the propositus. Fetal wastage as a whole and spontaneous abortions alone are more common in the sibships of male CL+P propositi. For comparisons of abortion incidence, $\chi^2 = 7.43$, $P < 0.01$ for male CI v. CL+P propositi; $\chi^2 = 4.37$, $P < 0.05$ for male CP v. CL+P propositi. For comparisons of fetal wastage incidence, $\chi^2 = 5.65$, $P < 0.025$ for male CL v. CL+P propositi; $\chi^2 = 6.00$, $P < 0.025$ for male CP v. CL+P propositi. Observed incidences of abortion and fetal wastage do not vary significantly by cleft type among the sibships of female propositi.

It is well established that among facial cleft propositi additional defects are markedly more common than in the general population of livebirths and children (*7, 8, 9, 12*). Table 2 presents the numbers and proportions of propositi in the present series who are affected with major or minor additional defects, with the total series subdivided by cleft type and sex of propositus. Defects forming a recognized syndrome of malformation have not been included, a practice at variance with some other studies (*8, 10*). Additional defects were taken to include all defects of presumably intrinsic origin, without being limited to the congenital malformations *in sensu strictu*. The additional defects noted in propositi of the present series are listed in the appendix.

Relatively fewer CL than other propositi have additional defects. Comparing incidences of affected, $\chi^2 = 7.5$, $P < 0.01$ for CL v. CL+P propositi; $\chi^2 = 7.9$, $P < 0.005$ for CL v. CP propositi. Overall, 22% of male and 16% of female propositi of the present series exhibit one or more additional defects, whereas perhaps 4 or 5% of all children might be expected to show additional defects as presently defined. The overall excess of males showing additional defects is not evenly distributed among the cleft types, being formally significant among CP propositi ($\chi^2 = 4.89$, $P < 0.05$ for male v. female propositi), and lacking in CL propositi. Among female propositi, additional defects affect approximately the same proportion of propositi of each cleft type. Among male propositi, however, the proportion of CP propositi with additional defects is relatively large and that of

TABLE 2. Additional defects in propositi.

	CL			*CL+P*			*CP*			*all clefts*		
	male	*female*	*total*	*male*	*female*	*total*	*male*	*female*	*total*	*male*	*female*	*total*
number of propositi	52	33	85	91	56	147	30	67	97	173	156	329
number with additional defects	3	3	6	22	10	32	13	12	25	38	25	63
frequency of affected	0.058	0.091	0.071	0.242	0.179	0.218	0.433	0.179	0.258	0.220	0.160	0.191

CL propositi with additional defects relatively small. Comparing numbers of male propositi affected with additional defects, $\chi^2 = 4.03$, $P < 0.05$ for CP v. CL+P propositi; $\chi^2 = 4.02$, $P < 0.05$ for CL v. CL+P propositi.

Table 3 presents the incidences of spontaneous abortion and fetal wastage in the sibships of propositi with and without additional defects. It can be seen that fetal wastage in the sibships of those propositi with additional defects tends to be greater than in the sibships of those without. This excess is confined to the sibships of female propositi with additional defects. Comparing the sibships of female propositi with and without additional defects, $\chi^2 = 11.81$, $P < 0.005$ for the difference in frequency of abortions; $\chi^2 = 11.64$, $P < 0.005$ for the difference in total fetal wastage. The excess of fetal wastage among the sibships of female propositi with additional defects is greatest among the sibships of female CL+P propositi with additional defects. Comparing abortion and fetal wastage frequencies with those in sibships of female CL+P propositi without additional defects, $\chi^2 = 9.57$, $P < 0.005$ and $\chi^2 = 6.73$, $P < 0.01$ respectively. In the sibships of female CP propositi with additional defects fetal wastage is significantly elevated ($\chi^2 = 4.73$, $P < 0.05$) but the incidence of abortion alone is not. In the sibships of female CL propositi with additional defects, neither fetal wastage nor abortion frequencies appear significantly elevated.

It is noteworthy that among the sibships of female propositi a substantial proportion of total abortions and fetal wastage occurs in the sibships of propositi with additional defects, and that the frequencies of abortion and fetal wastage in the sibships of female propositi without additional defects appear rather low.

Discussion

Present findings result from an investigation undertaken for other purposes, and a control series of family histories was not collected. Thus, the apparent overall deficiency of fetal wastage found in the sibships of facial cleft propositi cannot be considered conclusively demonstrated. There are, however, several indications that the observed deficiency of fetal wastage does not reflect inadequacies of the interviewing procedure:

1. For 27 mothers of propositi (8.2% of the study series) who had been delivered one or more times in the Princess Mary Maternity Hospital, Newcastle upon Tyne, it was possible to check reproductive histories obtained by examining obstetricians during confinements against the data obtained in the study interviews. Outcomes of 61 pregnancies (9.3% of all pregnancies reported) were thus verified. No abortions or other losses were reported to the examining obstetricians which were not reported to present interviewers. Nor were any losses reported to present interviewers but not to hospital obstetricians. One illegitimate livebirth noted in hospital rec-

TABLE 3. Effect of additional defects in propositi on fetal wastage in their sibships

Sibships of propositi with additional defects:

	CL			*CL+P*			*CP*			*all clefts*		
	male	*female*	*total*	*male*	*female*	*total*	*male*	*female*	*total*	*male*	*female*	*total*
total pregnancies excluding propositi	6	7	13	36	25	61	25	32	57	67	64	131
spontaneous abortions	0	1	1	4	7	11	0	8	8	4	16	20
frequency	—	0.143	0.177	0.111	0.280	0.180	—	0.250	0.140	0.060	0.250	0.153
total wastage	0	1	1	4	9	13	0	10	10	4	20	24
frequency	—	0.143	0.077	0.111	0.360	0.213	—	0.313	0.175	0.060	0.313	0.183

Sibships of propositi without additional defects:

	CL			*CL+P*			*CP*			*all clefts*		
	male	*female*	*total*	*male*	*female*	*total*	*male*	*female*	*total*	*male*	*female*	*total*
total pregnancies excluding propositi	86	63	149	154	93	247	26	105	131	266	261	527
spontaneous abortions	2	4	6	21	7	28	1	14	15	24	25	49
frequency	0.023	0.063	0.040	0.136	0.075	0.113	0.038	0.133	0.115	0.090	0.096	0.093
total wastage	5	9	14	27	11	38	1	15	16	33	35	68
frequency	0.058	0.143	0.094	0.175	0.118	0.154	0.038	0.143	0.122	0.124	0.134	0.129

ords had not been reported to present interviewers. These admittedly limited findings suggest that the present interview procedure elicited information regarding fetal wastage as complete as mothers were able to provide.

2. 64 of the family histories of the present series were collected by interviewers other than the writer; the incidences of spontaneous abortion, stillbirth, and neonatal death in this subgroup do not differ from those in the series of families as a whole. This internal consistency is a further indication that ascertainment of fetal wastage is not seriously inadequate.

3. Warburton and Fraser (*23*) in a study of spontaneous abortion based on 2,134 sibships, found for a subgroup of 248 sibships of propositi with CL and/or CP an incidence of spontaneous abortion of 9.7%. This value is significantly different ($P < 0.02$) from their estimate of $14.7 \pm 0.04\%$ spontaneous abortions in the series as a whole, and similar to the $10.5 \pm 1.2\%$ spontaneous abortions found in the sibships of the present series. Similarly, Henriksson (*12*) observed a frequency of spontaneous abortion in the sibships of 233 CL and/or CP propositi not significantly different from, but lower than, the accepted frequency of spontaneous abortion in Sweden.

Variation in the occurrence of spontaneous abortion and fetal wastage among the sibships of propositi with different types of facial cleft seems not to have been previously reported. Nor are there reports of variation in occurrence of fetal wastage in sibships according to whether or not a facial cleft propositus has an additional defect. Because present findings of such variation occur in a single series of sibship data collected by a uniform interview method, they are not readily explained as resulting from any variation in method of collection. Demonstration of such variation is independent of the indications of an overall reduction of fetal wastage in the sibships of facial cleft propositi.

Studies of the occurrence of other defects among facial cleft propositi give a very wide range of reported incidences, resulting for the most part from methodological factors (*8*). The incidences of additional defects ascertained for propositi of the various cleft types in the present series are comparable with those reported by other investigations of similar thoroughness (*8, 9, 12, 15*). Within a particular study, variation in occurrence of additional defects among propositi of different cleft types is not readily attributable to methodological factors. Most studies report a greater association of additional defects with CL+P than CL, paralleling present findings. On the other hand, as emphasized by Drillien et al. (*8*) most studies find a somewhat higher incidence of additional defects among CP than CL+P propositi. That this is not so in the present study probably reflects tabulation by other workers of subjects whose palate clefts occur as part of a syndrome of malformation, and the number of minor defects associated with CL+P in the present material.

Reports of the sex distribution of additional defects among cleft propos-

iti give no consistent picture. Parallel to the preponderance of males seen in the present material is the high proportion of males with multiple malformations including CL+P found by Stevenson et al. (*20*) in a world survey of congenital malformations. Farkas et al. (*9*) report associated defects to be proportionately more common among males than females for each cleft type (CL, CP, CL+P) in a series of 1,000 patients. Meskin and Pruzansky (*17*), however, report that females are more likely than males to have an additional malformation regardless of cleft type, while the data of other workers (*7, 8, 12*), show no particular pattern of difference in incidence of additional defects in the two sexes.

In Henriksson's (*12*) material, male CP propositi have significantly more additional defects than male CL+P propositi, while female propositi do not show a significant difference in incidence of affection with additional defects by cleft type. This parallels the findings in the present series. In the material of Czeizel and Tusnadi (*7*) additional defects affect significantly more CP than CL±P propositi among both males and females, while in the material of Drillien et al. (*8*) for neither sex are additional defects significantly more commonly associated with one genetic group or the other. Differing definitions of additional defect render comparison of these studies uncertain, but it seems likely that among males additional defects are more often associated with CP than CL±P.

Interpretation

The present findings suggest that fetal wastage in the sibships of facial cleft propositi, as ascertained by maternal interview, may be only ⅔ of that usually found. While numerous intermediary steps preclude the equation of reduced fetal wastage with increased fertility, it is tempting to speculate that such an association may in part account for the retention in human populations of genes disposing to these congenital malformations. This is particularly the case since the facial cleft conditions, while disabling, do not preclude survival, and particularly in primitive populations do not preclude reproduction.

There is good evidence that the etiology of cleft lip with or without cleft palate (CL±P) is in the majority of cases multifactorial and largely polygenic (*5, 11, 19, 24*) and this mode of causation also seems plausible for those CP cases not associated with syndromes of malformation (*11*). At present, however, ignorance of the actual processes controlled by the numberous genes presumed to be operating is almost total. It is conceivable that variation in fetal wastage between the sibships of cleft propositi, as seen in the present study, may be a main or pleiotropic effect of any of the genes postulated to be operating.

One may consider for the sake of argument a collection of sibships as originating from a pool of conceptions, some of which will develop with facial clefts, some of which will have other defects, and some of which will have both clefts and additional defects. Defective and particularly

multiply defective conceptuses stand a reduced chance of surviving to become infants. Kraus et al. (*14*), for a series of 60 spontaneously aborted fetuses with facial clefts, found about 85% of CL±P fetuses had additional external malformations, whereas 50% of CP fetuses had such defects. This is the reverse of the situation in living cleft propositi. These findings must be taken with some reservation since the karyotypes of the fetuses are not known; spontaneous abortions are often chromosomally abnormal and the syndromes of malformation related to several commonly occurring chromosomal abnormalities include facial clefts. For present purposes, however, this finding can be taken to indicate that in a pool of conceptions the fetuses whose defects do not result in death *in utero* are born as infants with congenital malformations.

On such premises, present findings regarding the CP condition may be explained by postulating that males with CP have "escaped" fetal wastage as a result of genetic factors associated with the etiology of the cleft condition. Fetal wastage is lower in the sibships of male propositi and additional defects are more common in male than female propositi, suggesting that factors affecting male prenatal survival may be sufficient to counteract the reduction of viability implied by possession of additional defects. A higher—that is, more usual—frequency of fetal wastage in the sibships of female CP propositi, and a relatively low frequency of additional defects in the female CP propositi themselves, suggest that no factors for prenatal survival counteract the operation of fetal wastage processes in their sibships. Indeed, the high frequency of fetal wastage in the sibships of female CP propositi with additional defects suggests a familial occurrence of such additional defects, and of fetal wastage eliminating defective conceptions from the postnatal population.

The CL±P picture is more complex. Although there is good evidence that CL and CL+P comprise a single genetic entity, the two conditions differ with regard to observed frequency of fetal wastage in the sibships of propositi, and also in observed incidence of additional defects in propositi. Several lines of evidence combine to prove an at least plausible explanation of this complexity. Adams and Niswander (*1*) have found evidence of an increased tendency to morphological assymetry in CL±P propositi by comparison with controls, which they suggest is "an indication of the inability of the genetic information to control development effectively in the presence of disturbing factors." It seems likely (*10*) that CP associated with CL may result simply from the disturbance of embryonic palate formation by the presence of a lip cleft. Such a mechanism has been demonstrated in mice (*21*). There is also some suggestion that genetic liability to lip cleft conditions increases from unilateral CL to bilateral CL+P (*5*). If the inability of genetic control to stabilise development played a part in the occurrence of a facial cleft, its occurrence in a lesser degree could account for CL, while its occurrence in a greater degree

could account not only for CL+P, but also for the greater association of non-cleft defects with CL+P.

For the CL+P condition, the reduction of fetal wastage in the sibships of propositi, if any, is less than that for the CL condition. This could reflect the association of additional defects with CL+P. If additional defects reflected a familially distributed inability of the genetic information to adequately control development, they would act to increase fetal wastage in the sibships of propositi. In the sibships of CL propositi, fetal wastage is low, particularly in the sibships of male propositi, suggesting that as in the CP condition there may be associated in the genetic etiology of CL±P factors acting to reduce male fetal wastage. There are other indications that factors favourable to the prenatal survival of males are operating in the families of CL±P propositi. One is the substantial excess of CL+P propositi who are males (*10, 11*). The other is that here occurs a notable excess of uncles over aunts, and of male over female first cousins, in the families of CL±P propositi. Tables 4 and 5 present values for the present series combined with that of Knox (*13*) which was also collected in Newcastle upon Tyne. Data presented by Woolf (*24*) for a series of 496 CL±P propositi ascertained in the southwestern U.S. show similar formally significant excesses of uncles over aunts and of male over female first cousins.

Summary

From the study of the occurrence of abortions, stillbirths, and neonatal deaths in the sibships of 329 facial cleft propositi, it is suggested that fetal

TABLE 4. Numbers of aunts and uncles of CL±P propositi, present series combined with that of Knox (1963).

propositus	*paternal*		*maternal*	
	uncles	*aunts*	*uncles*	*aunts*
CL				
male	153	137	196	146
female	98	79	122	102
CL+P				
male	301	272	275	260
female	176	154	187	176
CL±P				
male	454	409	471	406
female	274	233	309	278
Total	728	642	780	684

Total uncles 1,508
Total aunts 1,326 $\chi^2 = 11.69$, $P < 0.005$

TABLE 5. Numbers of cousins of CL±P propositi, present series combined with that of Knox (1963).

propositus	*paternal*				*maternal*			
	uncles		*aunts*		*uncles*		*aunts*	
	male	*female*	*male*	*female*	*male*	*female*	*male*	*female*
CL								
male	102	104	98	101	137	110	100	98
female	72	81	83	58	83	70	107	89
CL+P								
male	254	217	269	250	170	175	205	206
female	114	114	163	123	117	120	127	155
CL±P								
male	356	321	367	351	307	285	305	304
female	186	195	246	181	200	190	234	244
Total	542	516	613	532	507	475	539	548

Total males 2,201
Total females 2,017 $\chi^2 = 3.96$, $P < 0.05$

wastage may be reduced in the sibships of facial cleft propositi. In particular the overall frequency of spontaneous abortion—10.5%—is lower than expected. Degree of reduction appears to vary with the sex, cleft type, and additional defects of the propositus. The frequency of abortions found in the sibships of male CL and CP propositi—0.02—is particularly low, while that found in the sibships of female CL+P and CP propositi with additional defects—0.36 and 0.31 respectively—seems high. It is postulated that there are associated with the genetic component in the etiology of the CL, CL+P and CP conditions factors which enhance prenatal viability of male embryos. The pattern of variation seen in the occurrence of fetal wastage in the sibships of cleft propositi may be explained as resulting from the interaction of these postulated factors with the reduction of prenatal viability occasioned by the occurrence of multiple defects in embryos.

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APPENDIX

Additional defects of propositi with CL

Defects	Male	Female
None	49	30
Cephalohematoma over R occiput	1	
Bilateral inguinal herniae.	1	
Bilateral "bat-like" ear deformity	1	
Mental retardation		1
Stigmatism		1
Renal glycosuria		1
	52	33

Additional defects of propositi with CL+P

Defects	Male	Female
None	69	46
R. inguinal hernia, low intelligence	1	
L. bat ear, congenital deafness, very poor vision	1	
Micrognathia, short femurs, bilateral talipes	1	
Hypospadias, imperforate anus, R. inguinal hernia with hydrocele	1	
L. hallux valgus, segmental megacolon	1	
Multiple cysts attached to L. tunica vaginalis, L. inguinal hernia, L I-IS hydrocele	1	
Agenesis of fingers and syndactyly, obstruction of nasalochrymal duct		1
Idiopathic hypocalcemia	1	
Tetralogy of Fallot	1	
Dextracardia	1	
Hypospadias	1	
Epilepsy		1
L. external angular dermoid cyst	1	
Tongue tie, L inguinal hernia	1	
Degree of mental retardation	1	2
Degree of deafness	2	2
Bat ear(s)	2	
Micrognathia	1	
Cheek nevus with junctional activity	1	
Strabismus	1	1
L. congenital inguinal hernia	1	
L. accessory auricle	1	
Deficient maxilla		1
L. ptosis		1
Large rhinolith		1
	91	56

Additional defects of propositi with CP

Defects	Male	Female
None	17	55
Foot deformity, deafness.	1	
ESN, unusual habitus and facies	1	
Unusual nevus affecting face, head, sclera and cornea, sebaceous glands	1	
Micrognathia, misshapen lower lip, recurring teratoma at base of skull, fibrous thyroglossal cysts	1	
Bat ears, hearing loss, slanting eyes, hypertelorism	1	
Sacral meningocele, L. plagiocephaly and torticollis, wasting of R. leg impaired perineal sensation	1	
Absent R. pinna, L. pto-		

Additional defects of propositi with CP

Defects	Male	Female
sis, L. finger contractures		1
Talipes, umbilical hernia, hearing loss		1
Large hemangioma of inner canthus, deafness.		1
Finger contracture		1
L. obstructed nasolachrymal duct		1
Abnormally large pharynx	1	
Club foot	1	
Unstable L. hip		1
Atrial septal defect		1
Micrognathia	4	1
Degree of deafness	1	2
Severe intermittent eczema		1
Congenital hiatus hernia.		1
	30	67