Height-Weight Growth of Cleft Children, Birth to Six Years

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In 1960 the Toronto Research Institute of the Hospital for Sick Children (*7*) issured a five-year Report (1955–1959) on children aged five to fifteen years who had cleft lip and palate and isolated cleft palate. Their non-cleft control was derived from the height and weight data on 90,000 Toronto children. It was concluded that "the study leaves us with an impression that the physical development of the cleft palate children is impaired" (p.107). It was further stated that "a similar trend appears to be present in the birth weights of cleft and non-cleft children" (p.107).

Sex	*CL(P) + CL*		*CP Only*		*Control* (1)	
	N	Av.	N	Av.	N	Av.
M	100	7.22 lbs.	64	7.47 lbs.	175	7.59
F	100	6.76 lbs.	81	6.80 lbs.	159	7.09

(1) 1959 data from "a large Ontario Hospital"

The Report ended: ". . . impressions regarding lower birth weight and subsequent physical retardation among cleft lip and cleft palate children have not been proven to date." (p. 108).

In 1966 Drillien *et al.* (*1*) stated that in a group of children with congenital malformations the incidence of prematurity (birth weight below 5½ lbs.) is "rather higher than that found in the general population". As non-cleft controls for height and weight "Edinburgh standards" were used.

For *height* no significant difference was found in CL(P) or CP only (both FH + ve or FH − ve)†. However, in both FH − ve groups more patients than their sibs were below average height: 38% of FH − ve patients were below minus 1 S.D., but only 14% of sibs (Table 1).

For *weight,* corrected for clothing weight,‡ no significant difference was found in CL(P) or CP only (both FH + ve or FH − ve). Again, however, in both

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† FH + ve = family history positive, or "those patients having one or more known relative with a cleft of whatever type." (p. 15). FH − ve = family history negative, or "those patients who have no known relative with any type of cleft defect" (p. 16).

‡ The Institute data for weight includes ordinary indoor garb. In other words our data are gross weight, the Edinburgh net.

TABLE 1

age	height: male (cms.)									
	group a		group b		group c		group d		control	
	N	$\bar{X}$ (S.D.)	N	$\bar{X}$ (S.D.)	N	$\bar{X}$ (S.D.)	N	$\bar{X}$ (S.D.)	N	$\bar{X}$ (S.D.)
Birth	50	50.7 (3.5)	44	51.2 (2.8)	24	50.7 (2.3)	39	51.0 (3.4)	49	49.6 (2.3)
0:6	17	67.7 (4.0)	22	66.4 (4.9)	8	65.8 (5.0)	22	66.8 (4.3)	70	67.5 (2.3)
1:0	17	75.2 (4.4)	26	75.3 (4.8)	12	73.6 (2.8)	25	76.1 (3.3)	72	75.8 (2.8)
1:6	23	83.0 (4.3)	19	80.8 (3.8)	9	79.4 (4.0)	24	82.6 (3.6)	75	82.2 (2.8)
2:0	26	87.1 (4.1)	23	87.7 (7.8)	12	87.7 (3.0)	21	86.4 (3.6)	69	87.9 (2.8)
3:0	25	95.6 (5.5)	16	94.7 (4.4)	9	95.1 (1.7)	15	95.7 (3.5)	59	95.2 (3.2)
4:0	18	102.7 (5.6)	17	104.2 (7.7)	11	104.0 (3.7)	12	101.3 (4.4)	74	102.3 (2.9)
5:0	20	110.0 (5.5)	16	110.2 (6.7)	8	111.2 (3.7)	11	110.2 (6.1)	87	109.5 (3.3)
6:0	17	118.2 (6.6)	16	116.8 (4.9)	8	119.2 (4.8)	7	123.8 (7.0)	91	116.2 (3.5)
age	height: female (cms.)									
	group a		group b		group c		group d		control	
	N	$\bar{X}$ (S.D.)	N	$\bar{X}$ (S.D.)	N	$\bar{X}$ (S.D.)	N	$\bar{X}$ (S.D.)	N	$\bar{X}$ (S.D.)
Birth	62	49.5 (3.2)	27	50.2 (2.5)	13	50.8 (2.6)	21	49.9 (2.2)	56	49.0 (1.7)
0:6	25	64.7 (3.0)	12	63.8 (3.9)	8	69.0 (5.9)	12	63.9 (2.8)	63	65.4 (2.2)
1:0	32	73.5 (4.7)	17	73.0 (3.8)	7	75.5 (4.4)	15	72.2 (4.0)	75	74.0 (2.3)
1:6	33	80.3 (3.9)	17	80.5 (4.3)	9	80.8 (3.7)	10	80.5 (5.4)	74	80.4 (2.8)
2:0	31	86.0 (3.4)	18	87.7 (5.3)	7	86.9 (3.9)	13	86.3 (4.2)	68	86.8 (2.9)
3:0	27	93.6 (5.0)	14	93.0 (4.3)	6	92.0 (4.9)	11	93.9 (2.8)	71	93.8 (3.4)
4:0	22	101.0 (6.3)	14	102.2 (5.7)	6	102.0 (5.8)	8	103.0 (5.0)	78	101.8 (3.4)
5:0	25	106.9 (6.5)	10	109.9 (3.0)	3	108.2 (7.9)	6	108.3 (4.7)	90	108.9 (4.2)
6:0	12	114.5 (6.2)	7	115.8 (5.3)	1	—	3	114.5 (3.1)	95	115.6 (4.5)

FH – ve groups more patients than sibs were below average weights: 32% of FH – ve patients were below minus 1 S.D., but only 9% of sibs (Table 2).

The Edinburgh data were handled on the basis of intrauterine growth (expected birth weight for gestation age, per Lubchenko *et al., 4*). This type of sorting-out was not available for our Institute birth weight data. How much this effects comparability we do not know.

The Edinburgh study provides a causative lead in the matter of postnatal weight growth, viz., patients with a severe feeding problem were significantly more often below average weight (but not below avearge height) compared to the same cleft-type and family groups with no feeding problems. While no exact figures are available we feel that in the Institute cleft population sample "severe" feeding problems were minmal.

In 1972 Ross and Johnston (*8*) stated that, "Most studies indicate that CL(P) children tend to be shorter and lighter than control children. Data from studies of discordant MZ twins support this conclusion, but show, in addition, that the affected twin gradually *catches up* (italics ours) and may eventually pass the unaffected co-twin" (p.95). The authors feel that the H-W growth retardation is due to feeding problems + a heightened frequency of infections.*

In 1973 Spriestersbach, *et al.*, (*9*) in an overall "state of the art" assessment, observed that "in several (earlier) studies the height and weight of children with cleft palate have been found to be lower than those of siblings or other controls."

* Ross and Johnston observe that S.A. (maturation age) is retarded, while D.A. (eruption age) is not. We concur in the latter, have no data on the former.

TABLE 2

age	*weight: male (kgs.)*									
	group a		*group b*		*group c*		*group d*		*control*	
	N	$\bar{X}$ (*S.D.*)	*N*	$\bar{X}$ (*S.D.*)	*N*	$\bar{X}$ (*S.D.*)	*N*	$\bar{X}$ (*S.D.*)	*N*	$\bar{X}$ (*S.D.*)
Birth	50	3.4 (0.5)	44	3.4 (0.5)	24	3.7 (0.6)	39	3.3 (0.5)	50	3.2 (0.5)
0:6	17	7.3 (0.8)	22	7.4 (1.2)	8	7.3 (1.3)	22	7.8 (1.0)	70	7.8 (0.9)
1:0	17	9.7 (1.2)	26	9.8 (1.4)	12	9.3 (1.0)	25	10.1 (1.2)	73	10.0 (1.1)
1:6	23	11.2 (1.5)	19	11.1 (1.6)	9	10.0 (1.3)	24	10.9 (2.5)	82	11.4 (1.2)
2:0	26	12.0 (1.9)	23	12.4 (2.1)	12	12.5 (1.5)	21	11.6 (2.9)	88	12.5 (1.3)
3:0	25	14.3 (2.2)	16	14.2 (1.8)	9	14.3 (1.0)	15	13.1 (3.8)	92	14.6 (1.4)
4:0	18	16.0 (2.2)	17	17.0 (2.9)	11	17.1 (1.9)	12	15.8 (2.4)	91	16.4 (1.5)
5:0	20	18.4 (2.8)	16	19.0 (3.4)	8	18.9 (1.3)	11	18.9 (2.6)	87	18.7 (1.7)
6:0	17	20.8 (3.4)	16	20.2 (2.2)	8	22.6 (3.0)	7	20.7 (3.6)	91	21.0 (2.2)
age	*weight: female (kgs.)*									
	group a		*group b*		*group c*		*group d*		*control*	
	N	$\bar{X}$ (*S.D.*)	*N*	$\bar{X}$ (*S.D.*)	*N*	$\bar{X}$ (*S.D.*)	*N*	$\bar{X}$ (*S.D.*)	*N*	$\bar{X}$ (*S.D.*)
Birth	62	3.0 (0.5)	27	3.1 (0.5)	13	3.9 (0.5)	21	3.3 (0.4)	54	3.2 (0.4)
0:6	25	6.4 (1.0)	12	6.8 (1.1)	8	7.1 (1.0)	12	6.8 (0.8)	63	7.2 (0.8)
1:0	32	8.8 (1.4)	17	8.9 (0.8)	7	9.2 (1.1)	15	9.1 (1.4)	78	9.4 (1.0)
1:6	33	10.1 (1.4)	17	10.5 (1.2)	9	10.5 (1.2)	10	10.2 (1.6)	88	10.6 (1.2)
2:0	31	11.1 (2.6)	18	11.7 (1.1)	7	11.8 (1.4)	13	11.4 (1.3)	90	11.9 (1.3)
3:0	27	13.7 (1.9)	14	13.1 (1.4)	6	13.3 (2.7)	11	12.1 (4.2)	93	13.9 (1.4)
4:0	22	15.5 (3.1)	14	16.1 (1.1)	6	16.2 (3.5)	8	15.9 (2.0)	92	16.0 (1.6)
5:0	25	16.5 (5.0)	10	17.9 (3.0)	3	18.5 (5.2)	8	15.5 (6.6)	90	18.2 (1.9)
6:0	12	19.8 (4.8)	7	19.7 (2.5)	1	—	3	20.5 (1.4)	95	20.5 (2.5)

A correlation between "severe feeding difficulties in infancy" and reduced weight in later years was pointed out. The possibility of prenatal factors rather than post-natal nutritive problems must be considered.

The present study is a contribution to the H-W growth of cleft children, based upon the serial data-files of the Institute, with the exception of birth length and weight which were derived from hospital records. Height and weight were secured at the Institute at six-months intervals up to two years, thereafter annually (within two to four weeks of the birthday). Children below two years were weighed and measured in a supine position, after that standing.

Our longitudinal sample consists of 279 patients (155 M, 124 F). We decided to use as our non-cleft control the serial data from the Denver Child Research Council (*5*). As of 1969 the actively-followed enrollment was 179. Wherever possible first-born children were enrolled, of the middle and upper middle class, with family of each child having the medical care and advice of a private physician. Ethnically the familial backgrounds were of mixed European origin, predominantly North European. Our Institute children are comparable in both socio-economic milieu* and in ethnic composition, though not quite in specifics of medical care.

In the handling of our 279 propositi we decided on the following breakdown:

Group A—cleft of soft palate, cleft of soft and hard palate

H sample range M 17–50, F 12–62; W sample range M 17–50, F 12–62

Group B—unilateral cleft lip and palate

* With respect to the socio-economic factor Peter (*6*) has established that our Institute population represents a sample typical of the N.E. United States.

H sample range M 16–44, F 7–27; W sample range M 16–44, F 7–27

Group C—bilateral cleft lip and palate

H sample range M 8–24, F 3–13; W sample range M 8–24, F 3–13

Group D—cleft of lip, cleft of lip + alveolar process, both unilateral and bilateral

H sample range M 7–39, F 3–21; W sample range 7–39, F 3–21

Patients with syndromes involving palatal clefting were excluded from all sampling.

We established our objectives as follows:

1) to determine if the severity of the cleft-type affected the H-W growth of the patient on an age basis;
2) to determine if sex in relation to severity of cleft-type was significant;
3) to determine if the H and W of the cleft patients differed significantly from that of the non-cleft control.

As a basis for our discussion all data are presented in Table I for height and Table II for weight. The data are presented for each Group and for the Control.

Results

No marked or significant differences exist between the several cleft-types. Hence, severity of cleft-type is not a factor, since its effect is much the same for H and W, no matter what the cleft-type. This is not to say that clefting, per se, has no possible effect on H and W, only that severity of clefting is not a factor.

Sex appeared to be a factor for height and weight, birth to 36 months, with more direct male/female comparability thereafter. This B-3:0 and 3:0–6:0 apparent dichotomy may reflect improved nutrition and health post-operatively, i.e., after about 2:0, with a slight 2:0–3:0 lag.

In general the cleft Groups do not age-for-age and sex-for-sex show any real departure from the non-cleft Control averages. In W the cleft Groups seem to be a bit less, but not significantly so.

If we re-consider the before 3:0 and after 3:0 situation we may interpret this as a manifestation of H-W *catch-up* growth in the cleft samples. This restorative or recuperative phenomenon has been demonstrated in craniofacial growth, studied roentgenographically in lateral view (*3*) and in p-a view (*2*). Evidently general physical growth, as measured by H and W, and craniofacial growth march along together.

In more specific detail, by age 36 months, males had caught up to the norm for both H and W and thereafter began to be slightly above the norm. At no time did the females markedly exceed the norm, but by 36 months, females of Groups A and C had caught up and by 60 months, Group D had also caught up to the norm.

The overall trend appeared to indicate that the cleft children of this study are born a bit heavier and longer than the norm, but following birth they begin to show a lag. This lag may be attributed to early feeding difficulties, a tendency to frequent upper respiratory infections, and repeated hospitalization for lip and/or palate surgery.

By three years, the cleft individuals in our study tend to catch up to the norm.

Having the inherent potential for normal growth in H and W and given the impetus of increased feeding ability via surgical and prosthetic intervention, these individuals tried to rebound to growth equality and in some instances even slightly exceed the norm.

Summary

A study was conducted on 279 patients from the longitudinal patient series at the Institute to determine the general physical growth of cleft children using height and weight measurements.

The objectives were to determine if severity of cleft type and sex in relation to severity of cleft type significantly affected height and weight and to determine if the height and weight of cleft children vary significantly from the non-cleft norm.

The results showed that severity of cleft type is not significant while sex in relation to cleft type may be significant in several instances. It was also found that cleft children are neither consistently shorter nor consistently lighter than the norm. An early lag period occurred, but by three years cleft children catch-up to the norm and rebound to growth equality, thus appearing to conform to the concept of catch-up growth.

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References

1. Drillien, C. M., T. T. S. Ingram, and E. W. Wilkinson, The Cause and Natural History of Cleft Lip and Palate, Williams and Wilkins, Baltimore, 1966.
2. Ishiguro, K., W. M. Krogman, M., Mazaheri, R. L. Harding, J. Meier, H. Canter, and P. Ross, A longitudinal study of the morphological craniofacial pattern via P-A X-ray films, birth to six years. (unpublished Ms., 1974).
3. Krogman, W. M., M. Mazaheri, R. L. Harding, K. Ishiguro, P. Bariana, J. Meier, H. Canter, P. Ross, A longitudinal study of the craniofacial growth pattern in children with clefts as compared to the normal, birth to six years. *Cleft Pal. J. 12,* 59–84, 1975.
4. Lubchenko, L. O., C. Hansman, M. Dressler and E. Boyd, Intrauterine growth as estimated from liveborn birth-weight data at 24 to 42 weeks of gestation. *Pediatrics 32,* 793–800, 1963.
5. McCammon, R. W. (ed.), Human Growth and Development (p.101 ff., by C. F. Hansman, M.D., "Anthropometry and related data and skinfold thickness measurements") Thomas, Springfield (IL). 1970.
6. Peter, J. P. and R. R. Chinsky, Sociological aspects of cleft palate adults. I. Marriage *Cleft Pal. J. 11,* 295–309, 1974.
7. Research Institute, The Cleft Lip and Cleft Palate Research and Treatment Centre: a Five Year Report, 1955–1959 (M.A. Cox, Ed.), Hospital for Sick Children, Toronto, Canada, 1960.
8. Ross, R. B. and M. C. Johnston, Cleft Lip and Palate, Williams and Wilkins, Baltimore, 1972.
9. Spriestersbach, D. C., D. R. Dickson, F. C. Fraser, S. L. Horowitz, B. J. McWilliams, J. L. Paradise, and P. Randall, Clinical research in cleft lip and cleft palate: the state of the art *Cl. Pal. J. 10,* 113–164. April, 1973.