SURVEY OF CLINICAL RICKETS IN THE INFANT POPULATION IN CAPE TOWN 1967 - 1968*

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As infant rickets is present in children's wards in the Cape hospitals to a considerable extent, a survey of representative sections of the local infant population was undertaken to ascertain the incidence of clinical rickets in infancy.

Unselected groups of White, Coloured and Bantu infants were examined at immunization sessions, which are attended by over 90% of infants under 1 year.

ANALYSIS OF RESULTS

Certain selected groups were examined for particular parts of this study. The total number examined was made up as set out in Table I.

TABLE I. ANALYSIS OF PATIENTS EXAMINED

	White	*Coloured*	*Bantu*
Unselected infants attending for immunization			
Infants 3 - 8 months born May-June 1967 (winter babies)	98	453	104
Infants 3 - 8 months born Dec. 67 - Jan. 68 (summer babies)	—	71	—
Toddlers 15 - 22 months	—	52	—
Selected groups of infants			
Additional twins including 1 set of triplets	—	21	10
Infants given intramuscular injection vit.-D concentrate at 2 weeks of age	—	51	—
Infants fed for 3 months on Lactogen (winter babies)	—	20	—
Infants fed for 3 months on Pelargon + vit. D (summer babies)	—	15	—
Children of 1 - 3 years attending 'problem clinic' for malnourished children	—	112	—

Rickets was diagnosed if 2 or more of the following signs were present: Rosary, chest recession, Harrison's sulcus, funnel chest, craniotabes away from sutures, marked bossing of skull and wrist thickening.

The frequency of these findings in cases diagnosed as rickets is shown in Table II.

TABLE II. INCIDENCE OF CLINICAL SIGNS IN INFANTS EXAMINED

Sign	*Occurrence (%)*
Rosary	97
Chest deformities	50
Craniotabes	80
Frontal bossing	14
Wrist thickening	56

Rickets was reported as 'mild clinical rickets' or 'moderately severe' according to clinical findings.

All observations were made 'blind' by the same investigator. When examining a baby, the investigator had no knowledge as to the feeding, vit.-D intake or birthweight.

The over-all incidence of clinically recognizable rickets among unselected winter babies was found to be 17% for the Coloured and Bantu infants and 8% for the White infants (Table III).

TABLE III. INCIDENCE OF RICKETS IN INFANTS IN DIFFERENT RACIAL GROUPS EXAMINED (WINTER BABIES)

Racial group	*Number examined*	*Mild rickets*	*Moderately severe rickets*	*Total rickets*	*%*
White	98	8	—	8	8
Coloured	453	60	18	78	17·2
Bantu	104	14	4	18	17·3

The unselected Coloured summer babies showed an incidence of 15·6% (11 infants, all with mild rickets).

The more detailed investigations which follow were confined to the group of Coloured infants, as these form the largest section of the infant population. Over 500 Coloured infants in all were examined.

Full-term, Premature and Twin Infants

The incidence of clinical rickets among unselected winter babies was found to be 16% in single, full-term infants, 24% in single premature infants and 32% in twins (the additional twins examined being included in this figure) (Table IV).

TABLE IV. INCIDENCE OF RICKETS IN COLOURED INFANTS EXAMINED (FULL-TERM, PREMATURE AND TWINS, INCLUDING 21 ADDITIONAL TWINS – ALL WINTER BABIES)

	Number examined	*Mild rickets*	*Moderate rickets*	*Total rickets*	*%*
Full-term single infants	405	53	13	66	16·2
Premature single infants	41	6	4	10	24·3
Twins in series	7	1	1	2	32·1
Additional twins	21 (28)	4 (5)	3 (4)	7 (9)	

Nutritional State of Infants

The birthweights of 66 full-term infants with clinical rickets were in the same range as those of a control series of infants (obtained by random sampling of those infants who showed no signs of rickets). Their weight gains were also similar, except for 3 infants in the rickets group, one of whom was 50% below expected weight and two of whom were 30% below expected weight.

Age Incidence of Rickets

The infants were examined between the ages of 3 and 8 months. It will be noted from Table V that, although rickets was found between 3 and 4 months, it was milder

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than that seen between 4 and 5 months. This tied up with some of the infants seen monthly, who had minimal signs of rickets at 3 months of age and moderately severe rickets a month later.

TABLE V. AGE INCIDENCE OF RICKETS IN INFANTS EXAMINED (WINTER BABIES)

Age (months)	*Number examined*	*Mild rickets*	*Moderately severe rickets*	*Total rickets*	*%*
3–4	74	13	—	13	17·5
4–5	119	18	7	25	21·0
5–6	111	12	5	17	15·3
6–8	118	16	2	18	15·1
Exact age unknown	31	—	—	—	—

Incidence in Children over 1 Year of Age

It is difficult to get an unselected and representative group of preschool children. Fifty-two children between the ages of 15 and 22 months were examined at an immunization session. Of these, 15 had signs of having had rickets, e.g. Harrison's sulcus or widely open fontanelles, while 4 had active clinical rickets.

Malnourished Children

It has been noted that clinical rickets over the age of 1 year occurs mainly in children also suffering from protein-calorie malnutrition, particularly those children who are late in walking. These children are slow in development, and not mobile, and therefore continue in the 'baby' state, sitting indoors and getting no sunlight. It is in this group that severe bone deformity occurs—bending of the tibiae, knock-knees, pigeon chest and late closure of the fontanelle.

A very malnourished child of 5 years was seen with an open fontanelle, with no evidence of hydrocephalus.

An analysis of 112 malnourished children over 1 year of age with weights below the Boston 3rd percentile shows 31 who were treated for severe clinical rickets. Their weights, compared with those of other malnourished children, are shown in Fig. 1.

Sunlight

It is obvious that the degree of exposure to sunlight must have an effect on the incidence of rickets. A questionnaire was given to the health visitors to answer in respect of all cases of Coloured infants diagnosed as clinical rickets, and in respect of a control group. This group was a random sample of all infants in the Coloured group examined who showed no signs of rickets.

From the answers obtained (Table VI) it appears that the practice of leaving the infant lying in the room all day, as happens only too often when maternal care is poor, has a significant influence on the incidence of rickets.

PROPHYLAXIS

Vitamin-D Supplement Given

After clinical examination of the infant, mothers were questioned as to the amount of vitamin-D supplement given and the regularity with which it had been given in each case. From the replies received, only a rough

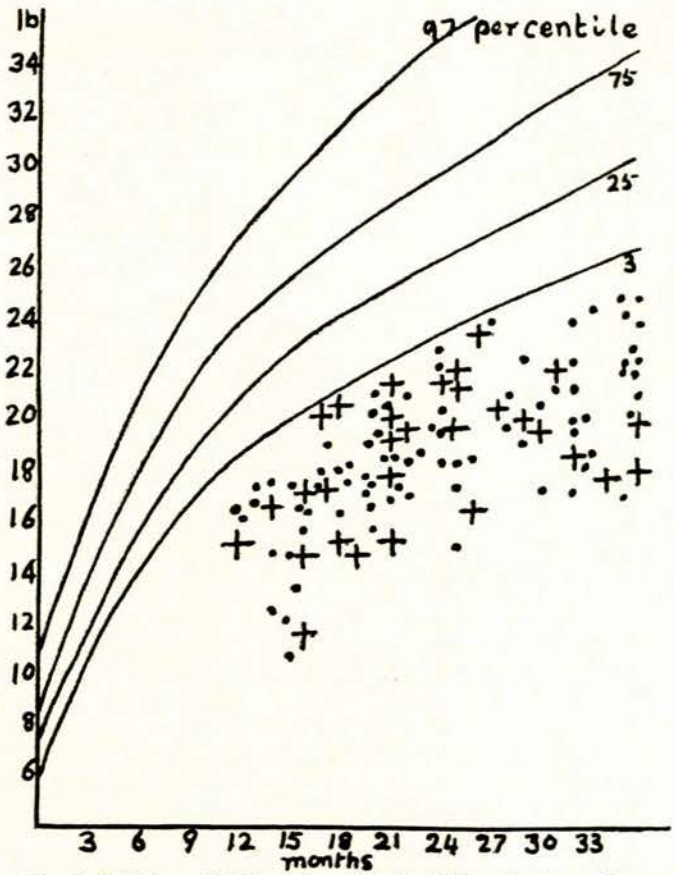

Fig. 1. Weights of 112 malnourished children between 1 and 3 years of age. Thirty-one (marked +) of these had severe clinical rickets.

TABLE VI. REPORTS BY HEALTH VISITORS ON 69 INFANTS WITH CLINICAL RICKETS AND 69 CONTROL INFANTS

Answers to questions	*Rickets group (%)*	*Control group (%)*
Child practically always kept indoors	67	12
Child very much wrapped up	35	8
Poor maternal care	40	12
Unhygienic, overcrowded home conditions	54	32

estimate could be made as to whether the infant had received a sufficient amount, as these statements could not be confirmed. However, the gradation from 29% of the infants having clinical rickets in a group who had no supplement to 9% in the group who probably had sufficient, is striking. This is shown in Table VII. The serious factor to note in this table is that out of 405 infants about whom information was obtained, only 100 probably had sufficient vitamin D. Premature infants and twins were not included here, as many of them had started life in hospital and had had intramuscular injections of concentrated vitamin D.

Vitamin-D Supplements—Premature Infants and Twins

This investigation was done for Coloured and Bantu infants together, as information about both was obtained

TABLE VII. INCIDENCE OF RICKETS IN FULL-TERM SINGLE INFANTS ACCORDING TO VITAMIN-D SUPPLEMENT GIVEN (WINTER BABIES)

Prophylaxis	*Number*	*Total rickets*	*%*
No vitamin-D supplement	70	20	28·6
Some vitamin D but insufficient	211	31	14·7
Probably sufficient	100	9	9·0
Unknown	24	6	25·0

from the hospitals.

Of the Coloured and Bantu infants examined, 49 were single premature, 34 were born in hospital and 3 were admitted to hospital from the surrounding district. The superintendents of the hospitals concerned kindly supplied information as to which of these infants (and the twins born in hospital) had received intramuscular injections of vitamin-D concentrate. The numbers of these premature infants who developed clinical rickets are shown in groups according to birthweight in Table VIII.

Thirty-seven twins and 1 set of triplets (Coloured and Bantu) were examined. Eleven pairs of twins and the triplets were born in hospital. Five of the twins (the twin of one baby having died) were born out of Cape Town and their birthweights were unknown, but they are reported as having been 'very small'.

The incidence of rickets according to birthweight is shown in Table IX. Of the 20 premature infants and low-weight twins who received intramuscular vitamin-D concentrate, 8 showed clinical rickets. Of these, only 2 appear to have had adequate and regular vitamin D by mouth after discharge from hospital, whereas, of the 13 who did not have rickets (including the triplets), 8 appear to have had adequate and regular amounts.

A similar picture was found in those premature infants and low-weight twins who did not receive intramuscular vitamin-D concentrate, namely that those who had regular supplies of vitamin D by mouth did not develop rickets. From these figures there seems to be a possibility that the regular and adequate administration of oral preparations of vitamin D to these low-weight infants is more effective in preventing rickets than a single dose of intramuscular vitamin-D concentrate.

The very high incidence of rickets in the twins over 5½ lb. should be noted. It is clear that all premature infants and twins need special care with regard to vitamin-D administration.

Use of Vitamin-D Concentrate by Intramuscular Injection

In July 1967, 103 Coloured infants aged under 1 month who were brought for BCG vaccination were given alternatively 1 ml. of Radiostol and a token dose of distilled water. The mothers of these infants were not told what injection they had had, and were given no instructions.

The infants were examined 'blind' 3, 4 and 5 months later, when they could be traced. One hundred and two were examined and 76 of these were re-examined a month or more later.

Radiostol series. Fifty-one infants were examined 3 months after injection. Two of these had small areas of craniotabes away from the sutures, but no other signs of rickets. The rest had no signs of rickets. They were all given a second dose of 1 ml. Radiostol. Thirty-seven were re-examined 1 - 2 months later. One of these had developed definite clinical rickets, with a marked rosary and craniotabes. This child weighed 5 lb. 4 oz. at birth, and so this finding fits in with the findings in the premature group.

Control group. Fifty-one infants were examined 3 months after injection: One had moderately severe rickets and was treated with Radiostol, and 5 others had mild clinical rickets.

Thirty-nine were re-examined 1 - 2 months later, when 2 of the infants seen with mild rickets on the first oc-

TABLE VIII. INCIDENCE OF RICKETS IN SINGLE PREMATURE INFANTS, ACCORDING TO BIRTHWEIGHT (COLOURED AND BANTU)

	Premature infants who received IM injection vitamin-D concentrate				*Premature infants who did not receive IM injection vitamin-D concentrate*			
Birthweight	*Total number*	*Mild rickets*	*Moderate rickets*	*Total rickets*	*Total number*	*Mild rickets*	*Moderate rickets*	*Total rickets*
4½–5½ lb.	4	—	—	—	26	1	—	1
3½–4½ lb.	7	3	2	5	6	2	1	3
Under 3½ lb.	4	—	2	2	2	—	—	—

TABLE IX. INCIDENCE OF RICKETS IN TWINS (AND 1 SET OF TRIPLETS) ACCORDING TO BIRTHWEIGHT (COLOURED AND BANTU)

	Twins who received IM injection vitamin-D concentrate				*Twins who did not receive IM injection vitamin-D concentrate*			
Birthweight	*Total number*	*Mild rickets*	*Moderate rickets*	*Total rickets*	*Total number*	*Mild rickets*	*Moderate rickets*	*Total rickets*
Over 5½ lb.	1	—	—	—	15	4	1	5
4½–5½ lb.	5	1	—	1	11	1	2	3
3½–4½ lb.	—	—	—	—	3	1	—	1
Unknown	—	—	—	—	5	1	2	3

casion now had moderately severe rickets and 3 others also showed mild rickets.

This gives a total of 9 infants seen to develop rickets out of the original 51, an incidence of 17·6%, similar to the general finding for Coloured infants.

Effects of Types of Milk Feeds Used

Every mother was questioned as to the type of milk feed her infant was receiving. The large majority were either breast fed or on unmodified dried milk. In order to assess the value of dried milk enriched with vitamin D, 20 infants were provided with Lactogen for 3 months, starting in their first month. No other vitamin D was given during this period. These infants were born at more or less the same time as the main group examined, therefore the incidence of rickets can be compared. A second group born in December 1967 and January 1968 were fed on specially enriched Pelargon.* They are compared with a group born in these months. In Table X the 20 infants in the Lactogen series are compared with other groups who had had no vitamin-D† supplements. Cases of rickets were found in all groups, but the breast fed infants and those fed on milk enriched with vitamin D had a very much lower incidence, with no severe cases, than the group fed on unmodified dried or fresh milk.

Taking all the infants together, according to their type of milk feeding, irrespective of the vitamin-D supplement received, there is still a marked difference, the incidence in infants on unmodified milk being double that in those on enriched milk or breast milk (Table XI).

*The amount of vitamin D added to the Pelargon for the purpose of this survey was 100 IU per oz. of powder or approximately 330 IU per pint of reconstituted Pelargon.

†The amount of vitamin D added to the Lactogen for the purpose of this survey was 113 IU per oz. of powder or approximately 339 IU per pint of reconstituted Lactogen.

CONCLUSION

The investigation demonstrates that rickets occurs in approximately 1 out of 7 infants in the Cape area and is most prevalent among premature infants and twins. Rickets at this age is dependent on the care the child receives, particularly whether it is taken out of doors or not and whether it receives regular supplement of vitamin D, and not on its general state of protein-calorie nutrition.

In the toddler group, on the other hand, active rickets was seen principally in children also suffering from protein-calorie malnutrition, particularly those who were late in walking.

The protective effect of intramuscular vitamin-D concentrate was demonstrated on a group of full-term infants.

TABLE X. INCIDENCE OF RICKETS IN COLOURED INFANTS ON DIFFERENT TYPES OF MILK FEEDS WHO HAD NO OTHER VITAMIN-D SUPPLEMENT

	No. of infants	*Mild rickets*	*Moderately severe rickets*	*Total*	%
Main series (winter babies)					
Special Lactogen series	20	2	—	2	10·0
Others fed on Lactogen or SMA	22	3	—	3	13·0
Unmodified dried milk and fresh milk	39	10	5	15	38·5
Wholly breast fed	39	4	—	4	10·0
Supplementary series (summer babies)					
Pelargon series	15	1	—	1	6·6
Unmodified dried milk	19	4	—	4	21·0

TABLE XI. OVER-ALL INCIDENCE OF RICKETS IN COLOURED INFANTS ON DIFFERENT TYPES OF MILK FEEDS IRRESPECTIVE OF AMOUNT OF ORAL VITAMIN-D SUPPLEMENT*

	No. of infants	*Mild rickets*	*Moderate rickets*	*Total*	%
Winter babies					
Milk enriched with vitamin D	99	10	1	11	11·0
Unmodified dried and fresh milk	202	34	9	43	21·0
Breast fed	115	11	2	13	11·0
Summer babies					
Milk enriched with vitamin D	18	1	—	1	5·5
Unmodified milk	48	9	—	9	18·7
Breast fed	20	2	—	2	10·0

* Infants whose feeds had been changed have been omitted.

It appeared to be largely ineffective in premature infants, unless it was followed up by a regular oral supply of vitamin D.

It was found that only 1 in 4 infants was receiving sufficient vitamin-D supplement.

The effect of using artificial feeds enriched with vitamin D was to reduce the incidence of rickets very considerably. It was noted, however, that rickets did occur even in babies fed on enriched milk when no extra supplement was given. This must be related to the fact that the amount of vitamin D in the various enriched milks works out at approximately 300 IU per pint of reconstituted milk. It would appear advisable to increase this amount to at least 400 IU.

The low incidence of rickets seen in breast fed infants deserves further study, as breast milk is reported to contain very little vitamin D. A possible explanation is that mothers who are breast feeding give their babies better care on the whole than those who bottle feed, and consequently the babies get more sunlight.

The time seems to be ripe to follow the overseas trend and use milk enriched with vitamin D for normal infant feeding. In addition, special care should be given to all premature infants and twins, to ensure continued and adequate dosage of vitamin D.

Similarly, breast fed infants should receive an adequate supply of vitamin D from birth. If these measures were applied, it should be possible to eliminate most of the infant rickets.

SUMMARY

A survey done on 600 infants and young children living in Cape Town in 1967 revealed that 1 in 7 infants developed clinical signs of rickets before the age of 6 months. Vitamin-D supplements, where given, reduced this incidence considerably, but only a small proportion of infants received these regularly.

A case is made for the use of dried milk, enriched so that a reconstituted pint contains 400 IU vitamin D, as the most effective method of preventing rickets.

I wish to thank the Medical Superintendents of Groote Schuur, Peninsula Maternity, Somerset, and Red Cross War Memorial Children's Hospitals for information relating to premature infants and twins in or admitted to these hospitals. The Lactogen and enriched Pelargon used in the experiments reported above were supplied by Nestlé (S.A.) (Pty) Limited.

HYPERTROPHY OF THE PYLORIC MUSCLE IN GASTRIC ULCERATION*

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Hypertrophic pyloric stenosis in the adult may be the end-result of conservative management of the infantile variety (in which cases there is an appropriate history) or it may commence in adult life. The latter variety may be the only lesion[1] or it may be associated with other abdominal disorders, of which gastric ulceration is one of the commonest. In such cases it is often difficult to decide whether the ulcer is the cause or the result of the muscle hypertrophy. There is no doubt that the stasis of pyloric hypertrophy can cause gastric ulceration,[1,2] but in many instances there is no history of preceding obstruction, which suggests that the ulcer was the primary event. This paper records some observations in this type of case.

Pyloric Muscle Thickness in Gastric Ulceration

A study was made of an unselected series of 31 stomachs resected for chronic gastric ulceration without any history of obstruction. At operation special care was taken to avoid injury to the pyloric muscle, and immediately after resection the stomach was opened along the greater curvature, pinned out on a board and fixed in 10% formalin in saline. After fixation, sections were cut of the whole thickness of the pyloric region of the stomach wall on the lesser and greater curvatures and anterior and posterior walls midway between the curvatures. The sections were stained with haematoxylin and eosin.

The thickness of the muscle coats was measured at the thickest part of the muscle, using a measuring device on the stage of a microscope.

Similar measurements were made on 10 normal stomachs removed at autopsy on patients without any evidence of abdominal disease and these were used as controls.

Measurements revealed no significant difference in thickness of the longitudinal layer of muscle of the ulcer cases as compared with the controls.

*Date received: 8 January 1969.

TABLE I. CIRCULAR MUSCLE THICKNESS

Site	*Mean of controls (mm.)*	*Mean of GU series (mm.)*	*Difference of means*	*t*	*N*	*P*
Greater curve	3·87	5·47	1·60	2·28	34	3%
Lesser curve	4·09	6·32	2·23	2·63	32	<2%
Anterior wall	4·10	5·02	0·92	1·78	34	10%
Posterior wall	4·06	5·43	1·37	2·04	35	5%

N = number of degrees of freedom.
P = level of significance.

The results obtained with the circular muscle are shown in Table I. The muscle in the ulcer group was significantly thicker than in the control group on the greater and lesser curvatures and on the posterior wall, but on the anterior wall this difference was not significant.

The Effect of Proximity of the Ulcer to the Pylorus

There was no significant correlation between the circular muscle thickness on the lesser curvature and anterior and posterior walls and the distance of the gastric ulcer from the pylorus, but on the greater curvature the coefficient of correlation is -0·66 (- indicates that distance is greater for thinner muscle). This is illustrated in Fig. 1. By the Standard t test this value is highly significant (probability of chance < 0·1% and 95% confidence limits as determined by Fisher's Z method are -0·38 and -0·84).

Time Relationship between the Onset of Muscle Hypertrophy and Gastric Ulceration

In the cases reported above it has been assumed that the ulcer preceded the pyloric hypertrophy because in none of them was there a history of pyloric obstruction preceding the ulcer symptoms. In one additional case this time relationship could be studied accurately.

Illustrative case report. A male, aged 27 years, developed a radiologically proved gastric ulcer in January 1964, when the pylorus appeared normal (Fig. 2). The ulcer