Among 460 preschool Negro children from low income families, 29 per cent were found to have low hemoglobin levels. Although these children had received well-child care, none had had a hemoglobin determination. The need for iron provided in dietary milk as a public health measure is emphasized.

THE PROBLEM OF IRON DEFICIENCY ANEMIA IN PRESCHOOL NEGRO CHILDREN

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In 1957 Guest and Brown[1] reported from Cincinnati that the prevalence of iron deficiency anemia in children under three years of age was the same or somewhat higher than it had been 20 years before. It is difficult to understand why a nutritional deficiency disease which is simple to diagnose and easy to cure should continue to flourish in an affluent country where well-baby clinics are available to all. The occurrence of nutritional anemia is not only an alarming statistic in its prevalence, but also is a cause of increased morbidity and probably of mortality in the early years of life.[2,3] It is primarily a disease of the lower socioeconomic groups and should be a prime target for improving community health.

The purpose of this report is to discuss the results found in a series of hemoglobin and hematocrit determinations performed on preschool Negro children during 1965 at the Child Health Center of Children's Hospital in Washington, D. C. Recent studies showing analogous results should stimulate public health programs similar to those that wiped out infantile rickets and scurvy in a preceding generation.

Clinical Material and Procedures

The patients were all Negro children from low income families, the majority of whom lived in the environs of the Child Health Center. This area is a Negro ghetto typical of those found in the central core of large northern cities. The families tend to be large, the mother is often head of the household, incomes are irregular and are derived from unskilled labor, the young child's diet consists to a great extent of the most easily prepared foods; this is especially true for milk from a bottle, which is often used as a pacifier. The excessive consumption of milk is abetted by the necessity for hiring baby sitters who are underpaid and untrained. About 15 per cent of the families in the study were supported by public assistance.

Hemoglobin and hematocrit readings were obtained on the preschool children over two months of age who attended the Child Health Center in 1965 and who had no record on their charts of a previous hemoglobin determination. Since no routine testing was performed before 1965, it was possible for some children to have reached five or six years of age without a test. On the other hand, many children had had hemoglobin levels taken in the past because the examining physician found some indication such as poor diet, prematurity, pallor, or frequent illness. Thus, many of the highest risk children had already been tested and

were not included in this series. Of course, an unknown number of the poorest and most disorganized families in the area did not come for well-baby care at all, a fact that would also tend to make the results reported here more favorable than the true prevalence of nutritional anemia in the total preschool population of this area.

Hemoglobin concentrations were measured by the cyanmethemoglobin method with the Coleman-Junior spectrophotometer. Microhematocrits were spun for five minutes in an International Micro-Capillary Centrifuge. When the hemoglobin level was below 10.0 gm per 100 ml, a smear for red cell pathology was examined. The presence of microcytosis and hypochromia was considered diagnostic of iron deficiency anemia. Any child with anemia of doubtful etiology was not included in the study but was referred to the Hematology Clinic for complete diagnostic work-up.

Results

A total of 460 preschool children were tested from approximately 800 who attended the clinics in 1965. The mean hemoglobin concentration was 10.40 gm per 100 ml; the mean hematocrit was 32.5. The lowest findings were in the children from 12 to 17 months of age whose mean hemoglobin level was 9.11 gm per 100 ml and the mean hematocrit 29.9 (Table 1).

Iron deficiency anemia, i.e., hemoglobin concentration below 10.0 gm per 100 ml with corroborative evidence on smear for red cell pathology, was found in 133 children, or 28.9 per cent of the total number. This group included only one pair of twins and nine patients with birth weights below five and one-half pounds; such patients would often have been tested previously. The incidence of iron deficiency anemia reached a peak of 65.0 per cent in the children from 12 to 17 months of age and then fell off rapidly in the older age groups (Table 2).

Those with anemia ranged in age from three months to five and one-third years. Relatively few cases of severe anemia were discovered; ten children, all under three years of age (or about 7 per cent of those with anemia) had hemoglobin levels below 7.0 gm. Among the 17 children tested under six months of age, over one-third had already developed iron deficiency anemia.

Table 1—Mean hemoglobin concentrations, range of concentrations and microhematocrit levels in 460 preschool Negro children

Age	No.	Hemoglobin average	Gm/100 ml range	Hematocrit average
0– 5 mo	17	10.29	8.9–11.9	32.4
6–11 mo	137	10.03	4.4–13.0	31.5
12–17 mo	46	9.11	6.0–12.6	29.9
18–23 mo	43	10.14	5.4–12.4	31.9
2–3 yr	73	10.54	6.5–12.7	33.1
3–4 yr	48	11.19	9.4–13.2	34.3
4–5 yr	68	11.21	8.5–14.5	34.1
5–6 yr	28	11.17	9.6–12.8	34.2
0–6 yr	460	10.40	4.4–14.5	32.5

Table 2 — Hemoglobin concentrations under 10.0 gm/100 ml in 460 preschool Negro children

Age	Total no.	Hemoglobin <10.0 gm/100 ml no.	%
0– 5 mo	17	6	35.3
6–11 mo	137	63	46.0
12–17 mo	46	30	65.2
18–23 mo	43	11	25.6
2–3 yr	73	14	19.2
3–4 yr	48	4	8.3
4–5 yr	68	4	5.9
5–6 yr	28	1	3.6
0–6 yr	460	133	28.9

When one follows the standard of Guest and Brown[1] and considers hemoglobin concentrations below 10.5 gm per 100 ml as indicating significant anemia, then almost half of the patients in the present series fell below this level (Table 3). The peak (84.8 per cent) was again found for the children from 12 to 17 months of age, but the per cent below 10.5 gm per 100 ml remained relatively high through the older age groups. This finding suggests that many children, though slowly overcoming their anemia, still had levels in the borderline range for many years. Seventy-seven per cent of the total series had hemoglobin concentrations below 11.5 gm per 100 ml (Table 3).

All of the cases which could be followed responded rapidly to oral iron medication and were found to have hemoglobin levels above 10 gm per 100 ml within a few months. There were 23 children who moved from the area and were lost to study at the Child Health Center. Ten cases required many months of follow-up, with two to four home visits, before the nurse could impress upon the parents and innumerable (as well as constantly changing) baby sitters the importance of giving the iron preparation regularly.

Comments

The exact percentages reported in the present study are not important but serious attention ought to be given to the implications of the findings. It should be emphasized again that the results do not indicate the true prevalence of iron deficiency anemia in Negro children from low income families of Washington, D. C. This report shows, more nearly, the prevalence in children considered normal by the pediatric resident staff and attending physicians!

Normal ranges for hemoglobin levels in the first years of life are not well established but no authority suggests that levels below 10 gm per 100 ml are normal. Guest, et al.,[4] propose 12.5 gm per 100 ml as desirable for all ages between three months and adolescence. Smith[5] considers the normal level for infancy between 11 and 12 gm per 100 ml, and that for children between 12.5 and 13.5 gm per 100 ml. A level of 11.5 gm per 100 ml may be accepted as optimal for infants, since it agrees well with the levels found in several studies

Table 3 — Per cent of 460 preschool Negro children with hemoglobin concentrations below 10.5, and below 11.5 gm/100 ml

Age	Total no.	% of hemoglobin concentrations <10.5 gm/100 ml	<11.5 gm/100 ml
1 yr	154	61.0	86.4
1–2 yr	89	68.5	87.7
2–3 yr	73	42.5	79.5
3–4 yr	48	25.0	60.4
4–5 yr	68	26.5	57.4
5–6 yr	28	21.4	60.7
0–6 yr	460	48.3	77.0

after prophylactic iron medication was given orally.[2,6-8,10]

Beside the high prevalence of anemia, two minor points of interest are emphasized by the present report. In population where iron deficiency is common, the condition develops early.[3,8,11] Over one-third of the infants tested in this series had already developed anemia by six months of age. While many more cases will be found by routine testing, delayed until one year of age, still one would tend to agree with Lahey[12] that infants who do not receive prophylactic iron medication should first be tested from five to six months of age.

Another point concerns the use of iron given intramuscularly — recommended for prematures[13]—that also seems to have valid use for the occasional child who cannot take iron by mouth or whose caretakers cannot be trusted to give it. For ten children in the present series, adequate treatment was delayed for many months, and many hours of nursing time were required in order to make certain that the medication was given. However, the necessity for giving iron intramuscularly should be evaluated carefully.

The sporadic reports made during the past four decades concerning the high incidence of iron deficiency anemia in underprivileged infants have, for the most part, been received with inertia and neglect. In 1928 an excellent and comprehensive study by MacKay[3] should have aroused the medical profession to wipe out this deficiency disease. MacKay found that the incidence of iron deficiency anemia was virtually universal in 541 "normal" infants of impoverished families that lived in the East End, London. The condition was prevented in a group of similar infants who were supplied with a formula to which the manufacturer had added an iron salt. Furthermore, the morbidity rate for total attacks of acute illness during a one year period was reduced by one-half in the treated cases. The same findings, in essence, were reported by Andelman and Sered[2] years later (1966) for a group of infants from a low socioeconomic population in Chicago.

Since 1928, the prevalence of iron deficiency anemia in children has been found to vary widely depending on the type of population studied. Andelman and Sered[2] reported the highest findings in 445 normal full-term infants—almost entirely nonwhite — who regularly attended Child Health Stations in Chicago; 76 per cent were shown to have hemoglobin levels below 10 gm per 100 ml by 18 months of age, and most of these had it by 12 months of age. This population was similar to that of the present study and, although the percentage with anemia was higher in Chicago, it was believed that the population studied in Washington would have shown about the same percentage with anemia if some high-risk children had not been eliminated from the series. In contrast to the findings in low income Negro populations, Guest and Brown[1] reported the most favorable picture for a series which they considered a cross section of the white population in Cincinnati; 30 per cent of the one- to two-year old children had hemoglobin levels below 10.5 gm per 100 ml. The prevalence cited in other reports has varied between these two extremes.[6,9,10,14,15] It is generally accepted that the prevalence is considerably higher in Negro children[11] and in low income groups of all races.[16]

One important fact has emerged from recent studies: diet, even a good diet, cannot be depended upon to prevent nutritional anemia in many instances. Shulman's excellent discussion[9] emphasized this point, and other authorities have agreed with it.[10,12,15,17] Certainly several investigations, including the present one, have shown that normal children under pediatric supervision are still in jeopardy of developing iron deficiency.[2,8,13,15]

The fact that the recent recommendation made by the American Academy of Pediatrics[18] for 1.5 mg of iron per kg of body weight per day in the first year of life is difficult to achieve with any diet that is not fortified with iron, supports this finding. Thus it follows that some prophylactic measure to augment iron intake must be undertaken. Many authorities in the field have suggested routine prophylaxis of some kind.[2-4,7-10,12,15,17]

The most simple, easy method, as well as the most certain—especially for the prevention of iron deficiency anemia in underprivileged families—is by the incorporation of iron into infant formulas. Several studies of the use of iron fortified formulas have shown most satisfactory results; good utilization of iron was found even in the earliest months of life.[2,3,8,9,19] Andelman's careful study[2] demonstrated that 12 mg of iron per quart of formula, beginning at birth (or at three months of age) and continued to nine months of age, decreased the incidence of nutritional anemia from the 76 per cent in 445 control infants to 9 per cent in the study group of 609 infants. The infants who received iron were protected through 18 months of age; moreover, those who had started the fortified formula at birth had significantly higher levels of hemoglobin at 18 months than did those who had started it at three months.

The amount of iron recommended daily for prophylaxis varies from 5 to 25 mg. Lahey[12] suggests 10 to 15 mg. In recent years many proprietary formulas have included 5 to 12 mg of iron per reconstituted quart. The addition of 12 mg per reconstituted quart has proved highly successful in prevention studies.[2,8]

One might ask, "Why worry about iron deficiency anemia since most children outgrow the condition between three and five years of age?" A generally accepted reason, though one of recent origin, concerns the fact that "anemia begets anemia," and children with a moderately low level of hemoglobin may be subject to occult gastrointestinal bleeding which can depress the level to a dangerous degree.[20]

Guest and his co-workers[4] list several other reasons for the prevention of iron deficiency anemia: (1) moderate anemia may become one of serious degree with superimposed infection; (2) there is less resistance to infection; (3) the habit of pica may be encouraged; and (4) when infection does occur, children with anemia are sicker and develop more complications.

The decreased resistance to infection in children with nutritional anemia is probably not proven to the satisfaction of skeptics. Certainly the reports of Andelman and Sered[2] and MacKay[3] that showed a statistically significant decrease during a period of a year or more, in the number of infections in a large series of treated cases as compared with an untreated series, should be given serious consideration. Andelman also noted several other minor studies with similar results. Large controlled series and prolonged observations are necessary for proof in a problem as complex as resistance to infection; however, it would seem that there is sufficient evidence to at least provide low income groups with the same preventive measure now supplied by many proprietary formulas for those who can afford them.

Several recommendations seem warranted, based on the present information on the high prevalence of iron deficiency anemia that is found in the well-baby clinics of large cities: (1) routine laboratory testing should be performed—by six months of age and again at 12 months of age—on those who do not receive prophylactic iron medication; (2) injections of iron by the intramuscular route should be considered for certain unusual cases; and (3) some form of prophylactic iron medication, preferably in the form of formulas fortified

with iron to the level of 12 mg per quart, should be made available in the first nine months of life.

Summary

In a series of 460 preschool Negro children from low income families, 133, or 29 per cent, were found to have hemoglobin levels below 10 gm per 100 ml and almost one half were below 10.5 gm per 100 ml. These children were a select group who had been receiving well-child care, but none had ever had a hemoglobin determination previously. The prevalence of anemia (hemoglobin levels below 10.0 gm per 100 ml) was high by six months of age, reached a peak of 65.0 per cent in those children who were 12 to 17 months of age, and then fell off rapidly in the older age groups.

Since individual supervision in well-child clinics has failed to solve the problem of iron deficiency anemia during the past 30 years, it seems imperative that 10 to 12 mg of iron per day should be provided in the dietary milk as a public health measure.

REFERENCES

1. Guest, G. M., and Brown, E. W. Erythrocytes and Hemoglobin of the Blood in Infancy and Childhood: III. Factors in Variability, Statistical Studies. Am. J. Dis. Child. 93:486–509 (May), 1957.
2. Andelman, M. B., and Sered, B. R. Utilization of Dietary Iron by Term Infants. Ibid. 111:45–55 (Jan.), 1966.
3. MacKay, H. H. M. Anemia in Infancy, Its Prevalence and Prevention. Arch. Dis. Childhood 3: 117–147 (June), 1928.
4. Guest, G. M.; Brown, E. W.; and Lahey, M. E. Normal Blood Values in Infancy and Childhood. Pediat. Clin. North America 93:362–363 (May), 1957.
5. Smith, C. H. Blood Diseases of Infancy and Childhood (Chap. 2, p. 29). St. Louis, Mo.: Mosby, 1960.
6. Colver, T. Anemia in Preschool Children. Lancet 1:145–147 (Jan.), 1938.
7. Sturgeon, P. Studies of Iron Requirements in Infants and Children. II. The Influence on Normal Infants of Oral Iron in Therapeutic Doses. Pediatrics 17:341–348 (Mar.), 1956.
8. Marsh, A.; Long, H.; and Stierwalt, E. Comparative Hematologic Response to Iron Fortifications of Milk Formula for Infants. Ibid. 24:404–412 (Sept.), 1959.
9. Shulman, I. Iron Requirements of Infancy. J.A.M.A. 175:118–123 (Jan.), 1961.
10. Moe, P. J. Iron Requirements in Infancy. Acta Pediat. Suppl. 150:1–67, 1963.
11. Munday, B.; Shepherd, M. L.; Emerson, L.; Hamil, B. M.; Poole, M. W.; Macy, I. G.; and Raiford, T. E. Hemoglobin Differences in Healthy White and Negro Infants. Am. J. Dis. Child. 55:776–783 (Apr.), 1938.
12. Lahey, M. E. Iron Deficiency Anemia. Pediat. Clin. North America 93:491 (May), 1957.
13. Elliott, W. D. Prevention of Anemia of Prematurity. Arch. Dis. Childhood 37:297–299 (June), 1962.
14. Baty, J. M. Classification of Anemia in Infants and Children. J.A.M.A. 134:1002–1003 (July), 1947.
15. Haughton, J. G. Nutritional Anemia of Infancy and Childhood. A.J.P.H. 53:1121–1126 (July), 1963.
16. Guest, G. M.; Brown, E. W.; and Wing, M. Erythrocytes and Hemoglobin of the Blood in Infancy and Childhood. Variability in Number, Size and Hemoglobin Content. Am. J. Dis. Child. 56: 529–549 (Sept.), 1938.
17. Schulz, J., and Smith, N. J. A Quantitative Study of the Absorption of Food Iron in Infants and Children. Am. J. Dis. Child. 95:109–119 (Feb.), 1958.
18. American Academy of Pediatrics: Report of the Committee on Nutrition. Trace Elements in Human Nutrition. Pediatrics 26:715–721 (Oct.), 1960.
19. Reedy, M. E.; Schwartz, S. O.; and Plattner, E. B. Anemia of The Premature Infant: Two Year Study of the Response to Iron Medication. J. Pediat. 41: 25–39 (July), 1952.
20. Hoag, M. S.: Wallerstein, R. O.; and Pollycove, M. Occult Blood Loss in Iron Deficiency Anemia of Infancy. Pediatrics 27:199–203 (Feb.), 1961.

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