

EDITORIAL : VAN DIE REDAKSIE

IRON-DEFICIENCY ANAEMIA IN CHILDHOOD

The application of the knowledge gained about vitamin-deficiency diseases and the widespread use of vitamins by child welfare clinics, hospitals, and physicians have almost eradicated these diseases. Long before the discovery of vitamins, however, iron was known to be necessary for the haemoglobinization of red blood corpuscles and known to be effective in the prevention and treatment of hypochromic anaemia. For various reasons, however, the value and efficacy of iron have on occasion been discredited.

It is surprising that, in a country such as America, where all aspects of nutrition are paramount in the minds of practising physicians, the incidence of iron-deficiency anaemia, in one centre at least, is as great today as it was two decades ago.¹ In Cape Town, Lanzkowsky and McKenzie,^{2,3} in a recent article in this *Journal*, have drawn attention to the high incidence of iron-deficiency anaemia in pre-school-going children attending various crèches in the Cape Town area. They also stressed the importance of maintaining an optimal haemoglobin level especially in the under-privileged sections of the community.

The association of an iron-deficiency anaemia with certain conditions, especially infections, occasionally results in a more serious prognosis and in some cases is an important contributory factor to the death of a child, e.g. when severe iron-deficiency anaemia is associated with bronchopneumonia, kwashiorkor, tuberculosis, etc. Many workers⁴⁻⁷ have recorded series of cases of lead poisoning and state that pica is an important aetiological factor in lead poisoning. Lanzkowsky⁸ has shown that all the children in his series of cases of pica had iron-deficiency anaemia. Iron-deficiency anaemia therefore, if associated with pica, predisposes to lead poisoning and thus in turn to a fatal outcome or to persistent neurological sequelae. Apart from these overt cases of iron-deficiency anaemia there exists a host of milder cases of iron-deficiency anaemia which remain undiagnosed and cause irritability, lassitude and anorexia and predispose the subject to infection.

Why is it then that iron-deficiency anaemia with its effective and cheap treatment is still prevalent today? Physicians, preoccupied with prescribing vitamin preparations, fail to insist on adequate prophylactic iron—particularly in cases where inadequate iron stores in the infant are anticipated. Adequate prophylactic iron therapy is indicated if the infant is prematurely born, one of a multiple birth or 'later born' compared with a first born, if the infant has lost blood at birth as a result of haemorrhagic disease or faulty ligation of the cord, or if less blood is replaced than is withdrawn in an exchange transfusion, etc. Prophylactic iron therapy is also necessary when maternal iron-deficiency anaemia exists as a result of frequent pregnancies or menorrhagia. The importance of maternal iron deficiency as a predisposing factor in the development of anaemia in infants has for a long time been debatable. The balance of opinion

in recent years favours maternal iron depletion as a definite aetiological factor in the development of iron-deficiency anaemia in infancy. It has been shown that while the mean haemoglobin levels of European and Cape Coloured infants at the time of birth are almost identical, the haemoglobin level of the Cape Coloured child at 3 months of age is statistically significantly below that of European infants of the same age.⁹ This may be a manifestation of inadequate maternal iron stores in Cape Coloured mothers.

Under these circumstances it is unsafe to rely upon dietary iron alone to prevent the development of iron-deficiency anaemia and it is wise to provide two to three times the usual dose of iron recommended for prophylaxis, i.e. 10-15 mg. of elemental iron daily rather than 5 mg., and therapy should be started at 6-8 weeks of age.

Post-natal factors such as dietary intake of iron, especially in prolonged breast feeding and excessive demand for iron because of rapid growth, are all potent factors in the development of iron-deficiency anaemia.

The presence of milder degrees of iron-deficiency anaemia is by no means always easy to recognize. The insidious onset of the condition, the remarkable ability of the body to adjust itself to a very low haemoglobin level, the vague symptomatology, the association with infection and the notoriously unreliable sign of pallor all combine to confuse the physician. McAlpine *et al.*,¹⁰ in a statistical investigation to determine the value of a clinical assessment of haemoglobin estimation based on facial colour, conjunctival inspection and inspection of the palmar creases, concluded that the clinical assessment of haemoglobin estimations above 60% (taking 14.8 g. as 100%) were so often erroneous that they could not be relied upon in the diagnosis of anaemia, whereas the error was small when the haemoglobin was 50% or less. This is in accordance with Lahey's statement¹¹ that pallor is rarely seen when the haemoglobin exceeds 7 g.% and only consistently appreciated when the haemoglobin is below 6 g.%. In view of the unreliability of the clinical determination of haemoglobin above 7 g.% it is a sad reflection on our standards of clinical accuracy that we should be satisfied to recognize haemoglobin levels below 7 g.% as the only significant criteria and pass the lesser degrees of anaemia as normal. The wider use of clinical haemoglobinometry is strongly recommended because this is the only way in which the all too frequent and important presence of subclinical anaemia can be recognized.

When iron-deficiency anaemia develops, treatment with an adequate dose of a suitable iron salt is indicated. Ferrous salts have an absorption of 10-15% in the usual doses given to anaemic patients in contrast to an absorption of 1.5-3.0% for iron and ammonium citrate.¹² For this reason ferrous salts in the form of sulphate or gluconate enjoy wide usage. A dose which furnishes 60-75 mg. of elemental iron per day,

given in 3 or 4 divided doses, is satisfactory for patients up to three years of age, after which 2-3 times this amount may be necessary. Four mg. of iron per kg. of body weight per day has been suggested as a minimal effective dose.¹¹ In addition to the rise in haemoglobin level induced by iron therapy, there are the less tangible effects of iron. Today, few physicians appreciate the marked tonic effect of iron therapy on the appetite, behaviour and sense of well-being of the child.

Until such time as iron is appreciated and made as readily available as certain vitamins, it will continue to be the physician's responsibility to recognize, prevent and treat this all

too prevalent deficiency state. This presents a perfect example of the scope of preventive medicine and deserves adequate recognition.

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