vitamin-B₁₂ levels decline through pregnancy, and after delivery infant levels are approximately twice those of the mother (Okuda *et al.*, 1956; Boger *et al.*, 1957; Dumont, 1957), and the low levels of serum vitamin-B₁₂ reported in the case of Kohler *et al.* (1960) and in the case recorded above are interesting in this respect. More recently, observations made in normal subjects indicate that in pregnancy there may often be an increased clearance of intravenously injected folic acid from the blood (Chanarin *et al.*, 1959b; Girdwood and Delamore, 1960).

The first patient described above presented in her first pregnancy with a megaloblastic anaemia that was unresponsive to folic acid therapy and with haemolytic anaemia in the second. It is possible that the prophylactic administration of folic acid in the second pregnancy may have prevented the development of megaloblastic erythropoiesis. Apart from the time of pregnancy the health of this patient has been entirely normal. In addition to this case, we have recently observed two other patients who were similar in that congenital spherocytosis was first suspected in pregnancy because on routine examination of the blood they were found to have anaemia and unusual reticulocytosis. In all of these patients the red-cell defect may well have remained unrecognized except for the advent of pregnancy.

The second patient is unusual in that we have not found records of any others in whom congenital spherocytosis produced megaloblastic anaemia in the absence of some other conditioning factor. Although the patient had a low level of vitamin B₁₂ in the serum he had hydrochloric acid in his gastric juice and normal absorption of vitamin B₁₂. He had a sudden crisis after an infection, and his blood picture was later characterized by leucopenia and thrombocytopenia. In the short period available for observation before operation, the administration of folic acid seemed to improve the white-cell and platelet counts.

It would appear that when haemolytic anaemia exists conditioned depletion of folic acid and perhaps also of vitamin B₁₂, as may occur in pregnancy, may aggravate the anaemia and lead to megaloblastic erythropoiesis; and that when a megaloblastic anaemia is discovered in pregnancy with an unusual reticulocytosis and is unresponsive to the usual therapy haemolytic anaemia should be considered. Moreover, as stated by Chanarin *et al.* (1959a), "a sudden increase in severity of anaemia or the onset of reticulocytopenia in a patient who is known to have a long-standing haemolytic anaemia should suggest the possibility that megaloblastic change is developing." We would add that in view of the appearance of leucopenia and thrombocytopenia in Case 2, sudden reduction in the white-cell and platelet counts may also indicate the need for examination of the marrow.

Summary

The cases of two patients in whom congenital spherocytosis is complicated by megaloblastic anaemia are described. In one patient it occurred in association with pregnancy, but in the other there appeared to be no complicating factor. It is suggested that if a patient suffering from chronic haemolytic anaemia suddenly becomes more anaemic, or develops leucopenia or thrombocytopenia, megaloblastic change should be suspected.

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CONGENITAL PYLORIC STENOSIS IN THE AFRICAN INFANT

BY

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In Europe and America congenital pyloric stenosis is regarded as the commonest condition requiring surgical treatment in infancy, and in Britain the incidence of the disease is usually estimated at three or four cases per thousand births. No case of this condition occurring in an African infant had been reported in the English literature until Petcher (1953) described two cases in Northern Nigeria. Since then, further cases have been reported from East, Central, and South Africa (Shepherd-Wilson and Gelfand, 1955; Luder, 1956; Griffiths, 1956; Hamilton, 1957; Menezes and Thethravusamy, 1957; Scragg, 1958; Boroda, 1960). A total of 11 cases of congenital pyloric stenosis affecting African infants have now been described, and their clinical details are summarized in Table I. In addition, Griffiths (1956) mentioned one and Scragg (1958) noted two further cases as having been diagnosed at their hospitals, but the cases were not described.

During the past three years six cases of congenital pyloric stenosis affecting Nigerian infants have been diagnosed and treated surgically in Ibadan, two cases in Lagos, and two cases in Enugu. They are reported briefly in Table II. In all these cases the typical pyloric tumour was found at operation and the standard Fredet-Rammstedt pyloromyotomy was carried out. Pre- and post-operative routines corresponded with the generally accepted techniques, and need no further consideration here.

Clinical Aspects.—A study of the case reports in Tables I and II shows that, with the exception of a rather late onset of vomiting in some cases, congenital pyloric stenosis, as so far reported in African infants, does not differ in its clinical aspects from the condition as it occurs in other races. The point is worth making, as cosmopolitan diseases do not always manifest themselves identically in different peoples. Two instances of

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this are peptic ulcer and intussusception, which in Southern Nigeria differ greatly in their natural histories from the same diseases as they occur in Europe and America.

Incidence in Africa

The question arises whether congenital pyloric stenosis is a rare disease in the African infant or whether it only seems to be so. In West Africa, and no doubt in many parts of Africa, it is almost impossible to assess the incidence of various diseases. A definite but unknown proportion of the population do not accept orthodox medical practice, and in the more remote areas a further unknown proportion are out of reach of such attention. Because of the widespread shortage of doctors, many hospital out-patient departments are grossly overcrowded, and the time the doctor can give to the individual out-patient is very limited.

The clinical picture of congenital pyloric stenosis is characteristic, and the diagnosis is based on the triad of signs and symptoms: projectile vomiting, visible gastric peristalsis, and a palpable pyloric tumour,

occurring in the early weeks of life. But much time and patience are often required to palpate the elusive tumour, without which a certain diagnosis cannot be made, while visible gastric peristalsis is often periodic in its appearance. It is all too easy to treat symptomatically the vomiting with which the infant presents, and to overlook the other signs, which, in any case, are not present in the early stages. Under the conditions prevailing in many African out-patient clinics, and bearing in mind that vomiting due to various causes is the presenting symptom in a large proportion of the infants attending them, the diagnosis of congenital pyloric stenosis is apt to be missed, and it may be assumed that many more cases occur than the literature would suggest.

With the expansion of medical services now taking place in most parts of Africa, and with greater awareness that the condition does occur in the African infant, there will certainly be an increase in the number of cases diagnosed and reported. On the other hand, the scarcity of reported cases to date is remarkable, and most of the authors quoted in Table I stated that the

TABLE I.—Previously Recorded Cases of Congenital Pyloric Stenosis in African Infants

Case	Author	Territory	Tribes	Sex	Birth Order	Age at Onset of Vomiting	Projectile Vomiting	Constipation	Dehydration	Malnutrition	Visible Gastric Peristalsis	Palpable Pyloric Tumour	Treatment	Result
1	Petcher (1953)	N. Nigeria	Laka	—	—	3 weeks	+	+	+++	+++	+	+	Pyloromyotomy	Cured
2	Petcher (1953)	"	Kilba	—	—	3 "	+	+	+++	+++	+	+	Operation refused	Died
3	Shepherd-Wilson and Gelfand (1955)	S. Rhodesia	—	M	2nd born	4 "	+	+	+++	+++	+	+	Pyloromyotomy	Cured
4	Luder (1956)	E. Africa	Ganda	M	3rd "	2 "	0	+	0	0	+	+	"	"
5	Griffiths (1956)	S. Africa	Bantu	M	—	3 "	—	—	—	—	+	+	"	"
6	Hamilton (1957)	Uganda	Dama	M	—	2 "	+	+	+	—	+	+	"	Cured
7	Menezes and Thehravusamy (1957)	Zanzibar	Nyagatwa	F	3rd born	3 "	+	+	++++	+++	+	+	Medical	"
8	Scruggs (1958)	S. Africa	Bantu	M	1st "	Unknown	+	+	+	++	+	+	Pyloromyotomy	Died from post-operative pneumonia
9	"	"	"	M	1st "	5 days	+	+	+	++	+	+	"	Cured
10	"	"	"	M	1st "	5 "	+	+	+	++	+	+	"	"
11	Boroda (1960)	E. Africa	Mudande	M	2nd "	3 weeks	0	++	0	0	+	0	"	"

+ = Positive finding. 0 = Negative finding. — = Finding not recorded. (Where more than one value is reported, the values given represent the extremes.)

TABLE II.—Newly Reported Cases of Congenital Pyloric Stenosis in Nigerian Infants

Case	Hospital and Date	Tribes	Sex	Birth Order	Age at Onset of Vomiting	Projectile Vomiting	Constipation	Dehydration	Malnutrition	Visible Gastric Peristalsis	Palpable Pyloric Tumour	Treatment	Result
1	Adeoyo Hospital, Ibadan. April, 1957	Bini	M	1st born	3 weeks	+	++	+	+	+	+	Pyloromyotomy	Cured
2	University College Hospital, Ibadan. April, 1958	Yoruba	M	1st "	7 "	0	++	+++	+++	+	+	"	Collapsed and died 24 hours after operation. P.M. did not reveal cause of death
3	University College Hospital, Ibadan. Aug., 1959	Ibo	M	5th "	8 days	0	+	+	0	0	+	"	Cured
4	University College Hospital, Ibadan. Feb., 1960	Yoruba	M	1st "	6 weeks	+	+	++	++	+	+	"	"
5	University College Hospital, Ibadan. April, 1960	"	M	1st "	2 "	+	+	0	0	+	+	"	"
6	University College Hospital, Ibadan. June, 1960	Urhobo	M	3rd "	4 "	+	+	0	0	+	+	"	"
7	General Hospital, Lagos. Nov., 1958	Yoruba	M	4th "	5 "	+	++	+++	+++	+	0	"	"
8	General Hospital, Lagos. Nov., 1959	"	M	—	1 week	0	+	+	0	+	+	"	"
9	General Hospital, Enugu. Jan., 1959	Ibo	M	—	10 weeks	—	—	—	—	—	—	"	"
10	General Hospital, Enugu. Sept., 1959	"	M	—	—	—	—	—	—	—	—	"	"

+ = Positive finding. 0 = Negative finding. — = Finding not recorded. (Where more than one value is reported, the values given represent the extremes.)

cases they recorded were unique in their experience. Bowesman (1960), in his book on surgery in the tropics, remarks that in twenty years of surgical practice in Africa he has operated on only six African babies for congenital pyloric stenosis. The town of Ibadan in Western Nigeria, with a population of over 750,000, is served by two large public general hospitals—University College Hospital and Adeoyo Hospital. The former has a large specialist paediatric unit, and in the latter separate children's out-patient clinics are held. If the incidence of congenital pyloric stenosis in Ibadan approached the European level of three or four cases per thousand births, it seems likely that many more than six cases would have been diagnosed between the two hospitals during the past three years. While the evidence is inconclusive, it suggests that the condition is relatively uncommon in the African infant.

Incidence in American Negroes

A consideration of the incidence of the disease in American negro infants is of obvious interest. The American literature does not appear to contain any study devoted to the subject, though various authors refer to it. Ward (1927) found no cases reported in American negro infants before 1927. Gleich and Goodman (1930) reported two cases in negro infants, and Donovan (1932) found two such infants in a series of 119 cases. Of 76 cases of congenital pyloric stenosis treated at the Charity Hospital, New Orleans, between 1916 and 1936 Romano and McFettridge (1938) found 30% to be negro infants. Ladd, Ware, and Pickett (1946), reporting on a series of 380 cases of the condition operated on between 1939 and 1945 at Harvard Medical School and the Children's Hospital, Boston, noted no racial disposition in their series. They ascribed the fact that they had never seen a case in a negro infant to pure chance. Mitchell-Nelson (1946) stated that the condition was apparently rare in negro infants. Gross (1953), in a presentation of the subject of congenital pyloric stenosis based on experience gained from the operative treatment of 1,787 cases up to December, 1952, stated that there was no racial predisposition to the condition, but did not state what proportion, if any, of his cases occurred in negroes. English *et al.* (1954) found 7 negro infants in a series of 41 cases operated on at the Boston City Hospital between 1949 and 1952. Benson and Warden (1957) reported from Michigan that, of their consecutive series of 707 cases operated on between 1940 and 1955, 63 were coloured infants, and that in their hospital admissions the ratio of coloured to white patients was roughly one to two.

These are all the references to the subject that a search of the available American literature reveals, and it is difficult to draw firm conclusions from them. They show that an increasing number of cases of congenital pyloric stenosis occurring in American negro infants have been reported over the past three decades, but no figures are given to show what relation the incidence of the condition bears to total births in this racial group. There are therefore no grounds for supposing that there has been an actual increase in incidence, and it seems likely that the increase in the number of cases reported is related directly to the expansion of medical services in America during the period concerned. In fact, the number of cases reported seems very small in relation to the large negro population in America.

In conclusion, it is suggested that congenital pyloric stenosis is a relatively rare condition both in the African and in the American negro infant, and that this may be due to a common genetic factor.

Summary

A total of 11 cases of congenital pyloric stenosis occurring in African infants have previously been described. A further 10 cases diagnosed in Nigerian infants over the past three years are reported.

The literature dealing with the occurrence of the condition in African infants and in American negro infants is reviewed.

The condition, as so far reported in African infants, does not appear to differ in its clinical aspects from the disease as it occurs in other races.

Congenital pyloric stenosis appears to be relatively rare both in the African and in the American negro infant, and this may be due to a common genetic factor.

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The proposed hospital at Lusaka, which is to be built at a cost of around £190,000, will provide accommodation for mentally defective Africans for whom there are no specialist hospital facilities at present in Northern Rhodesia. There will be three quadrangular blocks, housing altogether 316 patients, in the form of dormitories accommodating up to 25 patients. Each block has its own treatment, recovery, and occupational therapy departments. A central boiler-house supplies steam to each block to give hot water by means of calorifiers. There is a central kitchen with subsidiary kitchens in each block. The separate administrative block contains the pharmacy and a laboratory, as well as the various offices. Other buildings are a central laundry, stores, and mortuary. The hospital will be staffed by members of religious orders, and the male and female quarters have been designed accordingly. Separate housing is being provided for the medical superintendent and the hospital secretary. A hostel for female African staff and 26 houses for the male African staff are also being provided.