

last one. It died on the fourth day, from septicemia. The urine examined before inoculation showed no tubercle bacilli. The feces also gave no bacilli. The autopsy gave the same findings as in the preceding animal, except that the suppurative process was not so far advanced. The lungs were congested, but showed no purulent metastases. A smear from the peritoneal pus gave myriads of small diplococci and no tubercle bacilli. The bladder contained urine, which showed in the dried and stained specimen a few scattered tubercle bacilli. The feces also contained scattered single tubercle bacilli.

CONCLUSIONS

1. Intrauterine tuberculous infection of the ovum through the semen is probably a frequent event.

2. Tubercle bacilli are present in the feces and in the urine of many, and probably all cases of active local and systemic tuberculosis of the human being.

3. Tubercle bacilli appear in the feces and urine of tuberculous subjects within the first few days following an acute infection, and in certain instances long prior to the appearance of physical signs.

4. The only likely means of rendering tuberculous excreta (sputum, feces, urine) innocuous is the direct and effectual application of heat. They can not be expected certainly to overgrow or die out or surely to succumb to sunlight or drying.

5. Excreta from which formerly demonstrable tubercle have apparently disappeared may still prove destructive to animal life, and the bacilli again be recovered from the test animals in a demonstrable form, both by culture and by tinctorial methods of examination of the excreta.

6. The routine examination of the stools for tubercle bacilli should be more invariable even than that of the sputum. It more often yields a positive result. In occasional instances the microscopic field is as thickly spread with the bacilli as any sputum. Often the search must be painstaking and extended until a few scattered bacilli have been detected.

7. The examination of the urinary sediment for tubercle bacilli is not adapted to the use of the general practitioner. It should be resorted to whenever necessary to a thorough confirmation of suspected and doubtful or local (genitourinary) infection. The inoculation of guinea-pigs with the urinary sediment is a surer though slower method of demonstrating the bacillus, provided the test animal escape early death from mixed infection.

8. Probably all infections will open up to similar methods of study their causal organism when once we have determined upon specific stains and differential methods. There appears to us to be little doubt that every bacterial infection is a bacteriemia, and it seems not improbable that in every bacteriemia the causal organism is excreted through the urine and the feces.

SIGNIFICANCE OF THE FOREGOING

The import of this and of similar studies by other investigators is at once vital in its bearing on society at large. The physician is confronted with the certainty that he has no more than begun the crusade against tuberculosis. He has perhaps even overestimated the possibility of its complete ultimate control. Without the slightest desire of uttering a pessimistic word, and with the knowledge that in the clearest understanding of the truth lies the best hope of ultimate accomplishment—on the basis of this study we feel dubious as to the extinction of the disease by human means. No more likely is it that the discharges from the typhoid or cholera or dysenteric patient will always

or even usually (except in hospitals) be effectually destroyed, and thus prevented from polluting the air we breathe and the drinking supply, than that tuberculosis shall more than be inhibited in its spread. Granting as we now must that the infection is scattered broadcast for days and weeks, and perhaps months, before signs and symptoms demand a study of the case, it will be seen at a glance into what a labyrinth wanders the quest for its extermination and control.

Urine, sputum and feces are virulent, whether wet or dry, and for an indefinite time, the very indefiniteness of which renders them positively antagonistic to the health of the community until the bacillus or some equally positive destructive force places the sufferer under the sod.

The studies of Lord¹¹ have shown us that one fly-speck may contain as many as 5,000 tubercle bacilli; further, that thirty infected flies deposit in three days from 6,000,000 to 10,000,000 tubercle bacilli. We are, therefore, starting some centuries late in the destruction of flies.

Friedmann has injected tubercle bacilli into the vagina of a guinea-pig just after coitus, and later demonstrated the bacillus in the embryo. We, therefore, not only have on our hands the destruction of insects, but the prevention of marriage among the tuberculous. And yet, in spite of this knowledge that urine and feces and sputum and semen all carry, under favorable conditions, the certainty of a spread of the disease—in future rather because than in spite of this—there will be disinfected many an otherwise ignored or neglected source of contagion.

In so far as we arrive a step nearer a comprehensive realization of the armament, the subtlety, and the ubiquity of the tubercle bacillus; in just the proportion that our effort at control is relentless and systematic; in just that measure will we render a dayspring of true health conceivable, and lighten with hope this toilsome burden of man.

ANEMIAS OF INFANCY *

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The blood of infants under 2 years differs normally in certain of its characteristics from that of adults. The hemoglobin, relatively high for a time after birth, rapidly falls to a minimum of 55 or 60 per cent. in the first three weeks, then gradually rises to 70 per cent. at about six months, where it remains during the rest of infancy. There are, moreover, normally great variations in different individuals and in the same individual at different times. The number of red corpuscles is somewhat larger than in adults, averaging between 5,500,000 and 6,000,000 per c.mm. During the first few weeks of life there is more or less variation in the size and shape of the red cells, and nucleated forms, are not very unusual during the first few days.

11. Pub. Mass. Gen. Hosp., 1906, 1.

* Read in the Section on Diseases of Children of the American Medical Association, at the Fifty-ninth Annual Session, held at Chicago, June, 1908. Owing to lack of space, the article is here abbreviated by omission of the portion relating to leukemia. The complete article appeared in the Transactions of the Section and in the author's reprints.

The number of white corpuscles per c.mm. is somewhat larger than in adults, averaging from 10,000 to 14,000. The relative proportions of the various forms of leucocytes are also considerably different. The average figures are roughly as follows: Small mononuclear, 40 to 50 per cent.; large mononuclear, 10 per cent.; polynuclear neutrophils, 35 to 45 per cent.; eosinophiles, 1 to 5 per cent. That is, the proportion of mononuclear forms is considerably greater, and that of the polynuclear neutrophils considerably smaller than in adult life. The mononuclear cells, moreover, are not merely lymphocytes, but vary much, not only in the size of the cell as a whole, but also in the size of the nucleus and in the amount of protoplasm. Myelocytes are occasionally found under normal conditions in the first few weeks of life, but never later (Zelenski and Cybulski¹). The neutrophilic blood picture after the first few weeks is much like that of the adult, except that there are rather more corpuscles at the extreme right. Esser thinks that the blood picture shifts to the left when infants are artificially fed, but Zelenski does not agree with him.

Blood changes develop more easily and more frequently as the result of various morbid conditions and disease in infants than in adults. All the changes seen in the blood of adults as the result of disease are aggravated in infancy. The tendency is always to revert to a younger or to the fetal type of blood. All forms of blood disorders in infancy are apt to be associated with splenic enlargement. As the result of this tendency to aggravation of changes and to reversion to a younger type of blood, the red corpuscles show much greater variety in size and shape and many more nucleated forms are seen than under similar pathologic conditions in the adult. If, as seems probable, the red color of the marrow in infancy indicates that the whole marrow of the long bones is already occupied in the manufacture of red corpuscles, there can be no reserve power of formation at this age, as there is in the adult. This may explain why children bear hemorrhage so badly and why it is that they become anemic on such slight provocation.

Leucocytosis also develops more rapidly and to a greater degree, both in normal and abnormal conditions, than in later life. The physiologic leucocytosis of digestion, for example, is decidedly greater in infancy than later. The type of leucocytosis, moreover, is not as constant as in adults, in whom the increase of white cells is largely in the polynuclear neutrophils. In infancy the increase is sometimes in the lymphocytes, sometimes in the large mononuclear forms, sometimes in the polynuclear neutrophils, and sometimes, even, in the eosinophiles. Leucocytosis is more apt to occur in blood conditions associated with splenic enlargement than in those without it. Myelocytes occur in less severe conditions and in greater numbers than in adults (Zelenski and Cybulski¹). An increase in the number of eosinophilic cells, even if considerable, is not uncommon and seems to be of little significance.

The present theory as to the development of the leucocytes explains quite satisfactorily the peculiar type of leucocytosis in infancy. It is now believed that both the granular and the non-granular cells are derived from one common parent cell from which the red corpuscles are also derived. This parent cell is almost

identical in character with the large lymphocyte, and is the first to appear in the fetal blood.

During the process of development a gradual differentiation of function takes place in the blood-forming organs, as the result of which the lymphoid cells of the marrow produce only granular cells and those of the adenoid tissue only non-granular cells, or lymphocytes. This specialization, however, is never so complete but that when there is a demand for a greater number of cells of any type the lymphoid cells of any of the blood-forming organs are able to produce this type. Although it is probable that in the infant, as in the adult, the granular cells are derived exclusively from the marrow and the non-granular cells almost, if not absolutely, exclusively from adenoid tissue, yet the nearer the organism stands to the fetus the more easily can a return to fetal conditions take place, and the lymphoid cells of the marrow assume vicariously the function of producing lymphocytes and those of the adenoid tissue that of producing granular cells. There is, moreover, considerable pathologic evidence to show that such a vicarious function does actually take place in some of the blood disorders of infancy.

The relative and absolute excess of the non-granular cells which is characteristic of the blood in infancy indicates an unusual degree of activity of the adenoid tissue at this time of life. This activity of the adenoid tissue is of great importance pathologically because it is a well-known fact that the more active a tissue is the more likely is it to become diseased. Moreover, it is a fact that in the blood disorders of infancy affections of the adenoid tissue dominate the picture, while those of the marrow are extremely rare.

The percentage of hemoglobin is almost always relatively low in the blood disorders of infancy. This is presumably due to the facts that the infant normally receives an insufficient supply of iron in its food, and that the reserve of iron present in the liver at birth is not large enough to keep the percentage of hemoglobin at the adult standard. This reserve of iron may, moreover, be insufficient and in any event is comparatively easily exhausted.

In all the blood disorders of infancy, therefore, the most characteristic features are the relatively low percentage of hemoglobin, the relatively large number of non-granular cells and the marked morphologic changes in the red corpuscles. The blood picture may change very rapidly in children, corresponding to several types in a few weeks, and thus rendering the diagnosis extremely difficult.

It is customary to divide the diseases of the blood in adults into primary and secondary anemia and leukemia, including chlorosis and pernicious anemia among the primary anemias. The classification of the diseases of the blood in infancy is, however, not so simple, several of the types met in later life being entirely or apparently entirely lacking and other types not seen in later life being very common.

CONGENITAL ANEMIA

It is probable that Hutchison is right in recognizing a congenital anemia. In some of these cases it seems as if the reserve of iron at birth is insufficient. In others it is possible, but not proved, that there is a congenital defect in the bone marrow. In still other instances, it has followed a marked icterus neonatorum, this, perhaps, being a sign of excessive blood destruction at birth.

1. Jahrb. f. Kinderh., 1904, ix, 884.

CHLOROSIS

Much has been written recently, especially by the French, with regard to chlorosis in infancy. Some authors speak of it as chlorosis and apparently consider it the same disease as the chlorosis of young girls. Others doubt that it is chlorosis and prefer the term oligosideremia (Rist and Guillemot²), while still others speak of it as the chlorotic type of anemia in infancy. There can be no doubt that cases showing the type of blood seen in the chlorosis of young women are found not infrequently in infancy. There can be no doubt, too, that the clinical picture in these cases is somewhat different from that seen in the average secondary anemia at this age. The babies have a yellowish pallor; they are fat instead of thin; they are apathetic instead of irritable; the symptoms of disturbed digestion are comparatively slight; cardiovascular murmurs are common. There is also no question but that they improve very rapidly when iron is given. The following case is an example of this type of anemia:

CASE 1.—History.—No. 2,592 was seen when 23 months old. Her mother had died of cancer which she had during the pregnancy. The baby had always been fed on modified milk. She had had a number of digestive upsets when about a year old. She often seemed nauseated, but did not vomit. The bowels had always been regular. She had never had any other illnesses.

Examination.—She was well developed and nourished and moderately pale. There was a venous hum in the neck and a faint systolic murmur at the base of the heart. There was a slight rosary but no other signs of rickets. The liver was palpable 1 cm. below the costal border. The spleen was not palpable. There was no enlargement of the peripheral lymph nodes. The lungs were normal. The urine was normal.

Blood: The blood showed hemoglobin, 50 per cent.; red corpuscles, 5,122,000; white corpuscles, 11,300; mononuclear, 31 per cent.; polynuclear neutrophils, 65 per cent.; eosinophiles, 1 per cent.; mast cells, 3 per cent. The red corpuscles showed some variation in size and shape and moderate achromia. There were no nucleated forms.

She took her food well, did not vomit and had normal movements. She was listless and quiet and her temperature was subnormal. She improved very rapidly when iron was given.

There seems to be no justification for considering these cases to be chlorosis. They occur indifferently in boys and girls and have no pathologic connection with the nervous or genital systems. They are in most cases digestive in origin, but are apparently more likely to develop in infants whose reserve supply of iron was, as the result of premature or twin birth, deficient at birth or has been exhausted from some cause, such as hemorrhage, or in those who have been kept too long on a diet deficient in iron, such as milk.

The resemblance of the blood picture to that of chlorosis is easily explained by the facts that the percentage of hemoglobin is normally relatively low in infancy and that the number of red corpuscles is larger at this age than later. A diminution in both, therefore, gives a combination like that found in chlorosis in later life. This leads to confusion unless these facts are taken into consideration. Moreover, owing to the comparatively small reserve of iron, almost all anemias in infancy have a relatively low percentage of hemoglobin.

It is also not uncommon to see the chlorotic type of blood develop in the course of anemias in infancy and

disappear later; in fact, this type of blood is almost always present to a greater or less degree at some time or other in the course of every anemia in infancy. This is especially true in the early convalescent stage of secondary anemia. The ready response to iron in these cases is no proof of their essential nature, as all but the severest forms of anemia in infancy are very favorably influenced by iron and many of them do not respond materially to treatment until iron is given.

It is safe to conclude, then, that while anemia of the chlorotic type is common in infancy, the disease chlorosis probably does not occur at this age.

PERNICIOUS ANEMIA

There is great difference of opinion as to whether or not pernicious anemia occurs in infancy; all agree, however, that if it does occur it is extremely unusual. Hutchison³ could find but one case, that of Rotch and Ladd, which he considered genuine. The subsequent course of this case, however, shows that it was certainly not one of pernicious anemia. Simon⁴ accepts but four cases as genuine.

The diagnosis of pernicious anemia at this age is extremely difficult, if not impossible, because at this period any secondary anemia may, for the reasons already detailed, take on the characteristics which are considered pathognomonic of this disease in later life. The presence of marked variation in the size, shape and staining characteristics of the red corpuscles, of nucleated forms, whether large or small, of myelocytes, even if in considerable numbers, and of a relative lymphocytosis are, therefore, of little assistance in diagnosis at this age. Not even an increased globular value is pathognomonic. A fatal termination is also of little value in diagnosis because many of the severe secondary anemias are also fatal. The fact that the blood plates are diminished in pernicious anemia and increased in secondary anemia in adults, is of some importance in diagnosis at this age. So far as I know, there are no data as to this point in relation to the anemias of infancy. It is safe to assume, however, that if pernicious anemia does occur in infancy the characteristics of the blood are the same as in later life, and that the metaplastic type is by far the more common. Petrone has reported the only case of the aplastic type of which I am aware. This was in a baby of ten months in whose blood there were no nucleated red corpuscles or megalocytes, although the hemoglobin was only 12 per cent. and the red corpuscles numbered 1,400,000.

The conclusion seems justified, therefore, that pernicious anemia rarely, if ever, occurs in infancy, and that if it does the blood shows the same peculiarities as in later life and that it is almost universally of the metaplastic type.

SECONDARY ANEMIA

The characteristics of the blood and blood-forming organs in infancy, which have already been mentioned, make the infant especially liable to become anemic from the action of all sorts of debilitating and injurious conditions. These conditions may act either on the blood-forming organs or on the blood itself, rendering the former less capable of forming blood or increasing the destruction of the latter. However produced, secondary anemia in infancy is apt to assume a severity rarely seen

2. Bull. de la Société Méd. des Hôpitaux, 1906, Nov. 9; Abstr. in Arch. de Méd. des Enfants, 1907, x, 312.

3. Lancet, London, 1904, i, 1,253.

4. Rev. mensuelle des mal. de l'enfance, 1907, xxv, 145.

in adult life and in the most pronounced forms may present a picture which in the adult would justify the diagnosis of pernicious anemia.

The hemoglobin always suffers first, and in the mildest cases the blood shows merely the characteristics of a mild type of chlorosis. This is not surprising when the normally low percentage of hemoglobin is remembered. When the injurious influences are more powerful there is an increased destruction of red corpuscles and an impairment of the reproductive power of the bone marrow with a consequent diminution in the number of red corpuscles as well as in the hemoglobin. Abnormalities in the size, shape and staining qualities of the red corpuscles develop and nucleated red corpuscles appear. It is the pronounced tendency toward the development of these abnormalities which especially distinguishes the secondary anemias of infancy from those of adults. The explanation of this tendency is presumably to be found in the fact that the bone marrow in infancy is, under normal conditions, working close up to its limit, and thus, being unable to increase its productive power by hyperplasia, meets the demand for new cells by a more rapid division of its constituents instead of by an extension of the area of production. Such rapid production necessarily leads to imperfect formation of the corpuscles which are thus sent into the blood in an only partially completed condition.

In all but the mildest forms, leucocytosis is likely to develop in addition. The type of the leucocytosis varies according to the cause of the anemia, the increase depending on the chemotactic influence of the special causative condition and bearing no direct relation to the changes in the red corpuscles. It is most often of the polynuclear type, but may be of the lymphocytic, especially when the anemia is due to diseases of the gastroenteric tract. It may, however, be eosinophilic when the anemia is due to helminthiasis or of the large mononuclear type when it is due to malaria.

(I have quoted Hutchison freely in the past paragraph, as well as in other parts of this paper, and wish to acknowledge my indebtedness.)

ILLUSTRATIVE CASES

CASE 2.—History.—No. 2,520, aged 3 months. Proprietary foods. Appetite good. No vomiting. Slight constipation.

Physical Examination.—Fairly developed and nourished. Fair color. Anterior fontanelle depressed. Slight rosary. Faint systolic murmur at base. Lungs normal. Edge of liver 1 cm. below costal border. Spleen not palpable. Slight puffiness of hands and eyelids. Slight general enlargement of peripheral lymph nodes.

Urine: Pale, acid, 1006, no albumin.

Blood: Hemoglobin, 65 per cent.; red corpuscles, 4,064,000; white corpuscles, 16,800; small mononuclear, 55 per cent.; large mononuclear, 25 per cent.; polynuclear neutrophiles, 20 per cent. No morphologic changes in red corpuscles.

CASE 3.—History.—No. 1,982, aged 18 months. Nothing known as to first nine months. Last nine months fed on milk and water. Digestion had been good.

Physical Examination.—Small and poorly nourished. Moderate pallor. Top and back of head flattened. Frontal eminences enlarged. Three teeth. Slight rosary. Chest flattened. Heart and lungs normal. Edge of liver 3 cm. below costal border. Spleen not palpable. Abdomen and extremities normal. No enlargement of peripheral lymph nodes.

Urine: High colored, acid, 1018, slightest possible trace of albumin.

Blood: Hemoglobin, 35 per cent.; red corpuscles, 4,336,000; white corpuscles, 21,000; small mononuclear, 19.5 per cent.; large mononuclear, 14.75 per cent.; polynuclear neutrophiles, 63.25 per cent.; eosinophiles, 1.25 per cent.; myelocytes, 0.5

per cent.; mast. cells, 0.75 per cent. Slight irregularity in the size, shape and staining qualities of the red corpuscles and a very few normoblasts.

CASE 4.—History.—Sally L., age 12 months, infectious diarrhea at 9 months. After recovery, began to vomit occasionally and to be somewhat constipated. Vomiting increased, but was quickly relieved by careful feeding.

Physical Examination.—Fairly developed and nourished. Marked pallor. Six teeth. Slight rosary. Heart and lungs normal. Edge of liver 1 cm. below costal border. Spleen not palpable. Abdomen negative. Slight enlargement of epiphyses at wrists and ankles. Slight general enlargement of peripheral lymph nodes.

Urine: Pale, no albumin or sugar.

Blood: Hemoglobin, 55 per cent.; red corpuscles, 3,700,000; white corpuscles, 5,500. Slight variation in the size and shape of the red corpuscles but no nucleated forms.

CASE 5.—History.—No. 1,505, age 10 months. Premature. Fed on undiluted cows' milk. Progressive failure; vomiting; constipation.

Physical Examination.—Emaciated. Marked pallor of skin and mucous membranes. Slight rosary. Heart and lungs normal. Edge of liver 2 cm. below costal border. Spleen not palpable. Slight enlargement of epiphyses at wrists and ankles. Slight general enlargement of peripheral lymph nodes.

Urine: Normal color, acid, no albumin.

Blood: Hemoglobin, 25 per cent.; red corpuscles, 2,600,000; white corpuscles, 15,000; small mononuclear, 61 per cent.; large mononuclear, 3 per cent.; polynuclear neutrophiles, 35 per cent.; eosinophiles, 1 per cent. Marked variation in the size of the red corpuscles, but none in their shape or staining reaction. No nucleated forms.

Blood after a month of treatment with modified milk, beef juice and iron: Hemoglobin, 35 per cent.; red corpuscles, 3,600,000; white corpuscles, 14,000.

CASE 6.—History.—No. 2,395, age 14 months. Imperforate anus with successful operation. Breast for three months, then cow's milk, then milk and barley water. Otitis media at 10 months. Digestion disturbed the last six months. Feet swollen for a week, hands and scrotum for several days.

Physical Examination.—Small and poorly nourished. Marked pallor. Marked edema about eyelids and a little of cheeks. Four teeth. Slight rosary. Venous hum in neck. Heart and lungs normal. Abdomen distended. Liver 2 cm. below costal border. Spleen not palpable. Slight edema of scrotum. Some edema of feet and moderate edema of legs below the knees. Slight enlargement of cervical lymph nodes. No evidences of scurvy.

Urine: Pale, acid, 1020, no albumin.

Blood: Streaky. Hemoglobin, 25 per cent.; red corpuscles, 2,300,000; white corpuscles, 10,400; small mononuclear, 8 per cent.; large mononuclear, 18 per cent.; polynuclear neutrophiles, 73 per cent.; eosinophiles, 1 per cent. Marked variation in the size, shape and hemoglobin content of the red corpuscles, but no polychromatophilia or stippling. One megaloblast seen in counting 120 white corpuscles.

ANEMIA WITH SPLENIC TUMOR

In many instances anemia in infancy is associated with enlargement of the spleen. This enlargement may be of all grades, from one so slight that the spleen is barely palpable to one in which it reaches the anterior superior spine of the ilium and fills up the whole flank. Enlargement of the liver is also not uncommon. Leucocytosis may or may not be present, and if present may, as in the secondary anemia just mentioned, be of any type. All grades of morphologic changes may be found in the red corpuscles and nucleated red cells of various types may or may not be present. Much difference of opinion has arisen as to the classification of these cases and as to the relation between the splenomegaly and the anemia, some believing that the primary seat of the trouble is in the spleen, others that the

splenomegaly is secondary to the anemia, and still others that neither the splenomegaly nor the anemia is dependent on the other, but that both are the manifestations of some other diseased condition. Still others believe that the marked cases are leukemia modified by the physiologic conditions peculiar to infancy, and others place them in the indefinite class of adult splenic anemias.

Before discussing these different theories it may be well to cite a few cases to illustrate some of the various grades of severity of this condition.

CASE 7.—History.—Fred A., age 2 years. Always perfectly well, except for occasional very slight digestive upsets, and pneumonia four months previously. A month before had a slight digestive upset from which he apparently quickly recovered. Temperature, however, continued to be a little elevated from time to time. Appetite good. Tongue a little coated. Digestion good except for slight constipation. Slight loss of color and circles under eyes.

Physical Examination.—A little pale. Small circles under eyes. Tongue slightly coated. Loud venous hum in neck. Heart normal except for slight systolic murmur at the base. Lungs normal. Abdomen negative. Edge of liver 1 cm. below costal border. Spleen palpable 1 cm. below costal border. Extremities normal. No enlargement of peripheral lymph nodes.

Urine: Pale, acid, 1024, no albumin or sugar.

Blood: Hemoglobin, 50 per cent.; red corpuscles, 5,088,000; white corpuscles, 10,900; mononuclear, 59.75 per cent.; polynuclear neutrophils, 36.5 per cent.; eosinophiles, 3.25 per cent.; myelocytes, 0.5 per cent. Slight macrocytosis and poikilocytosis. Moderate achromia. No nucleated corpuscles. No malarial organisms.

CASE 8.—History.—No. 760, age 11½ months. Nothing known as to the history except that she had had whooping-cough three months before, that her appetite was poor, that she had vomited occasionally and had had a sore mouth.

Physical Examination.—Markedly emaciated and very pale. Systolic murmur over whole precordia. Occasional râles in both chests. Edge of liver 1 cm. below costal border. Spleen palpable 3½ cm. below costal border. Slight general enlargement of peripheral lymph nodes. Body and extremities covered with a fine purpuric eruption. Eyelids puffy. Few hemorrhagic spots in mouth.

Blood: Markedly pale and yellow. Hemoglobin, 19 per cent.; red corpuscles, 950,000; white corpuscles, 27,000; small mononuclear, 52 per cent.; large mononuclear, 13 per cent.; polynuclear neutrophils, 32 per cent.; eosinophiles, 0.5 per cent.; myelocytes, 2.5 per cent. Marked variation in size and moderate variation in shape of red corpuscles; 22 megaloblasts, 8 microblasts, 25 normoblasts and 12 atypical nucleated red cells seen in counting 500 white cells.

CASE 9.—History.—No. 1,427, age 18 months. No history.

Physical Examination.—Flabby and rather pale. Fontanelle nearly closed. Parietal eminences enlarged. Twelve teeth. Slight rosary and flaring of the lower ribs. Heart and lungs normal. Upper border of liver flatness at fifth rib, lower border palpable 6 cm. below costal border. Spleen palpable 3 cm. below costal border; surface smooth, consistency firm, edge rounded, notch distinct. Epiphyses at wrists and ankles enlarged. Slight bow-legs. Slight general enlargement of peripheral lymph nodes.

Urine: Normal color, acid, 1012, no albumin.

Blood: Hemoglobin, 75 per cent.; red corpuscles, 5,650,000; white corpuscles, 11,800; small mononuclear, 32 per cent.; large mononuclear, 8 per cent.; polynuclear neutrophils, 51 per cent.; eosinophiles, 9 per cent. A very little variation in the size of the red corpuscles, but none in their shape or staining reaction. No nucleated cells seen.

CASE 10.—History.—No. 1,538, age 17 months. Breast-fed for five months; then strained, rolled oats. Had vomited occasionally and lost some weight. History very indefinite.

Physical Examination.—Well developed and fairly nourished. Color good. Fontanelle closed. Slight rosary. Heart and lungs normal. Lower border of liver almost touched the anterior superior spine, reached to within 2 cm. of the navel and

passed under the left costal border in the left nipple line. Spleen palpable running out from beneath the costal border in the anterior axillary line, directly downward to the level of the anterior superior spine and backward into the flank; surface smooth, density increased, edge sharp, notch not palpable. Abdomen large and lax. Epiphyses considerably enlarged at wrists and ankles. Legs slightly bowed. Slight general enlargement of peripheral lymph nodes.

Urine: Pale, 1008, no albumin.

Blood: Hemoglobin, 65 per cent.; red corpuscles, 3,720,000; white corpuscles, 10,700; small mononuclear, 30 per cent.; large mononuclear, 4 per cent.; polynuclear neutrophils, 65 per cent.; eosinophiles, 1 per cent. Little variation in the size and none in the shape of the red corpuscles. No nucleated forms seen.

CASE 11.—History.—Christine G., age 12 months. Breast-fed for two months; after that extremely badly fed. Much vomiting and undigested movements. Steady loss of weight. Edema began at 10 months. Never any fever.

Physical Examination.—Marked emaciation. Very marked pallor. Fontanelle depressed. Six teeth. Gums and mouth normal. Venous hum in neck. Systolic murmur at base of heart. Lungs normal. Moderate rosary. Abdomen negative. Lower border of liver 2 cm. above navel. Spleen felt running out from under costal border downward to level of navel and backward into flank; surface smooth, edge sharp, notch distinct. Extremities normal. Slight general enlargement of lymph nodes. Slight edema of hands and eyelids.

Blood: Hemoglobin, 20 per cent.; red corpuscles, 952,000; white corpuscles, 10,320; mononuclear, 60 per cent.; polynuclear neutrophils, 37 per cent.; eosinophiles, 1 per cent.; basophiles, 2 per cent. Very marked variation in size and shape of red corpuscles, many large forms, also very marked polychromatophilia. Four normoblasts and 11 megaloblasts seen in counting 200 white cells, as well as 3 red corpuscles with stippling.

Under careful feeding and citrate of iron subcutaneously the general condition improved somewhat, although petechial spots appeared from time to time and edema of the face and extremities increased.

Blood at 13 months: Hemoglobin, 35 per cent.; red corpuscles, 1,164,000; white corpuscles, 11,800; mononuclear, 50 per cent.; polynuclear neutrophils, 44 per cent.; eosinophiles, 2 per cent.; basophiles, 3 per cent.; myelocytes, 1 per cent. Marked variation in size and shape of red corpuscles; marked polychromatophilia, 7 normoblasts and 13 megaloblasts and 2 red corpuscles with stippling seen in counting 300 cells.

A week later she had an acute intestinal upset from which she rallied, to die, after progressive failure, at 14 months.

Autopsy (By Dr. Bassoe, Chicago).—General anemia; emaciation; catarrhal gastroenterocolitis; edema of face and lungs; slight hydroperitoneum; bilateral hydrothorax; slight hyperplasia of spleen; few retroperitoneal hemolymph glands; slight bronchopneumonia. Bacterial examination showed pneumococci in cultures from the heart's blood.

Microscopic examination of spleen, by Dr. Wolbach, Boston): The spleen showed a slight increase in the reticulum and a slight amount of pigment within the cells about the trabeculae. The pulp also contained an excess of polynuclear leucocytes. The condition is presumably one of chronic passive congestion.

CASE 12.—History.—No. 2869. Age 12 months. No history of importance.

Physical Examination.—Small and poorly nourished. Moderate pallor. Serous discharge from right ear. Frontal eminences enlarged. Head flattened on top. Fontanelle depressed. Two teeth. Venous hum in neck. Marked rosary. Heart and lungs normal. Edge of liver 2 cm. below costal border. Spleen palpable running out from costal border in the left anterior axillary line downward and forward to navel, downward and backward to anterior superior spine, then backward along crest of ileum and under ribs in scapular line. Surface smooth, edge a little rounded, notch indistinct. Marked enlargement of epiphyses at wrists and a little at ankles. Inguinal and cervical lymph nodes the size of peas.

Urine: Clear, no albumin.

Blood: April 4: Hemoglobin, 40 per cent.; red corpuscles, 4,054,000; white corpuscles, 13,000; mononuclear, 91.7 per cent.; polynuclear neutrophiles, 4.2 per cent.; eosinophiles, .5 per cent.; myelocytes, 3.1 per cent.; mast cells, .5 per cent. One normoblast to 250 white cells.

April 13: Hemoglobin, 45 per cent.; red corpuscles, 3,094,000; white corpuscles, 24,000; mononuclear, 41 per cent.; polynuclear neutrophiles, 38 per cent.; myelocytes, 18 per cent.; mast cells, 3 per cent. Marked poikilocytosis. Moderate achromia. Some stippling. Three normoblasts seen in counting 200 white cells.

April 24: Hemoglobin, 50 per cent.; white corpuscles, 45,000; mononuclear, 37.5 per cent.; polynuclear neutrophiles, 40.5 per cent.; eosinophiles, 2 per cent.; myelocytes, 20 per cent. Nine megaloblasts and 12 normoblasts seen in counting 200 white cells.

May 9: Hemoglobin, 60 per cent.; red corpuscles, 3,789,000; white corpuscles, 23,000; mononuclear, 62 per cent.; polynuclear neutrophiles, 29 per cent.; eosinophiles, 3 per cent.; myelocytes, 6 per cent. Moderate macrocytosis and slight poikilocytosis. Sixteen megaloblasts and 8 normoblasts seen in counting 300 white cells; also an occasional nuclear figure.

May 27: Hemoglobin, 80 per cent.; red corpuscles, 3,440,000; mononuclear, 55 per cent.; polynuclear neutrophiles, 40 per cent.; eosinophiles, 4.5 per cent.; myelocytes, .5 per cent. Slight poikilocytosis; 6 normoblasts seen in counting 200 white cells.

June 24: Hemoglobin, 80 per cent.; red corpuscles, 5,600,000; white corpuscles, 22,000; mononuclear, 63.3 per cent.; polynuclear neutrophiles, 32.6 per cent.; eosinophiles, 2.1 per cent.; myelocytes, 2 per cent. Slight poikilocytosis. No macrocytosis; 3 megaloblasts and 8 normoblasts seen in counting 300 white cells.

During this period the child had taken its food well and rarely vomited. The movements had been fair, better toward the end. The weight had increased from 5,340 grams to 5,910 grams. The treatment had been buttermilk and citrate of iron, gr. $\frac{3}{4}$, subcutaneously, every other day. The baby then left the hospital, but reappeared at the Floating Hospital a week later. The physical examination was the same as when she was first seen.

Urine: Normal color, acid, slightest possible trace of albumin, a few leucocytes and squamous cells, an occasional red blood corpuscle, no casts.

Blood: July 2: Red corpuscles, 5,392,000; white corpuscles, 51,400; mononuclear, 41 per cent.; polynuclear neutrophiles, 59 per cent.

Died July 8.

Autopsy (by Dr. Wohlbach).—There were marked signs of rickets. The spleen was long and narrow. The estimated weight was 250 grams. The color was deep brownish-red, the consistency firm. On section the cut surfaces were smooth and deep red. The corpuscles and trabeculae were easily visible, as in the normal spleen. A very small amount of pulp was obtained by scraping. The liver was normal in size. The capsule was smooth, the consistency normal, the color pale brownish-red. On section the lobular markings were barely visible. Bone marrow: That in the vertebrae was deficient in color and amount; that from the femur abundant, dark grayish and fairly firm in consistency. There were in addition an organized pneumonia, acute bronchitis, chronic fibrous pleuritis, lymphatic hypertrophy, atrophy of the thymus, dilatation of the stomach and subpericardial ecchymoses.

Microscopic examination of spleen: The splenic nodules were numerous, large and irregular in shape. The germinal centers were very large and showed numerous mitotic figures. The pulp showed a slight increase in the reticulum. The sinuses and reticular tissue contained many large cells with granular protoplasm resembling myelocytes. These cells had either blue or purple staining protoplasm, the stain used being methylene blue and eosin. Groups of such cells were found surrounded by nucleated corpuscles, forming foci similar to the erythroblastic centers of the bone marrow. There were also many mononuclear eosinophiles with large nuclei, cells resembling those at the germinal centers of the nodules, and plasma cells. There was moderate injection of the sinuses with blood. There

were many polynuclear leucocytes in the pulp and sinuses. Many were fixed in the act of migrating from the sinuses.

Liver: The general arrangement of the lobules and columns of cells was normal. The liver cells were normal, except in places where they were slightly compressed by the crowding of the sinusoids with myelocytes. This was most marked just beneath the capsule. There were both basophilic and neutrophilic myelocytes singly and in groups. There were also many nucleated red blood corpuscles, often in close relation to clumps of myelocytes. Various types of nucleated reds were found, so that these collections of cells resembled those found in the spleen and in the bone marrow. The sinusoids contained everywhere much blood. The blood in the large vessels contained an excess of white corpuscles, in which large cells resembling myelocytes predominated. Many mononuclear eosinophiles were present in the sinusoids and in the blood of the large vessels.

Kidney: There was marked cloudy swelling of the convoluted tubules with slight fat vacuolation at the bases of the cells. The glomeruli, except for a few of fetal type, were normal. There were in the cortex a few elongated radial areas of connective tissue increase. These regions contained glomeruli of the fetal type and atrophic tubules. There was marked lymphoid and plasma cell infiltration and a few mononuclear and polynuclear eosinophiles.

Ileum: The solitary follicles were large with large and active germinal centers.

Tonsils and mesenteric and cervical lymph nodes: These all contained large and very active germinal centers. They all contained numerous mononuclear and polynuclear eosinophiles.

Bone marrow: That from the femur and vertebra showed marked activity. There were in both many erythroblastic centers, and nucleated red corpuscles were everywhere abundant. There was a great increase in the number of eosinophilous myelocytes. Megalokaryocytes were present in about normal numbers.

Diagnosis: Hematopoietic spleen and liver. Chronic focal nephritis and parenchymatous degeneration of kidneys. Lymphoid hyperplasia.

In spite of the frequency of these conditions, and in spite of the frequency of a fatal termination, pathologic data are comparatively few. The chief changes in the spleen are an increase in the connective tissue, usually associated with a diminution in the pulp. It never shows any changes in any way resembling those of leukemia, the changes being of the same sort as those found in other cachectic conditions, such as rickets (Sasuschin⁵) and syphilis (Hutchison³). The liver shows simple parenchymatous changes, fatty degeneration, such as is seen in all severe anemias, or simply lymphoid infiltration. The bone marrow shows nothing abnormal, merely the signs of increased activity. There are no changes suggestive of leukemia or in any way characteristic. The lymph nodes, if not normal, show merely the changes of chronic adenitis. Lymphoid infiltration has been occasionally found in the intestine.

Certain authors believe that anemia with splenic tumor in infancy is an aleukemic stage of leukemia or that it is leukemia modified by the peculiarities of the infant's blood and blood-forming organs. Aleukemic stages of leukemia certainly do occur, even in infancy, as is shown by the case reported in this paper. No one, however, has ever satisfactorily described a case of anemia with splenic tumor in infancy which has later changed to a true leukemia. No pure case of myelogenous leukemia has ever been reported, moreover, although anemia with splenic tumor is not at all uncommon at this age. Finally, the pathologic changes are radically different. The view that anemia with splenic tumor in infancy is merely leukemia modified by the

5. Jahrb. f. Kinderh., 1900, 11, 297.

age of the patient is hardly tenable, not only because of the reasons just given, but also because true leukemia does occur at this age. Moreover, many of these cases recover entirely and the splenic tumor disappears.

It has been stated that these cases do not respond to treatment with the x-ray while cases of true leukemia do, and this statement has been advanced as a further argument against their leukemic nature. A case of Lommel's,⁶ however, improved markedly under treat-

the changes in the bone marrow are not those of pernicious anemia. Moreover, it is extremely doubtful if pernicious anemia occurs at this age.

A study of a large number of cases of anemia with splenic tumor shows that all grades of anemia, with or without leucocytosis, may be found when the enlargement of the spleen is inconsiderable. The cases analyzed in Table 1 illustrate this point.⁷

Patient 2204 had a systolic murmur in the heart;

TABLE 1.—ANALYSIS OF CASES OF ANEMIA WITH SLIGHT SPLENIC ENLARGEMENT

No.	Age in Months.	Spleen. Cm. below ribs.	Per cent. Hemoglobin.	Red Corpuscles.	White Corpuscles.	Small Mononuclear.	Large Mononuclear.	Polynuclear Neutrophils.	Eosinophiles.	Myelocytes.	Mast Cells.	Morphologic Changes.	Etiology.
2526	18	2	65	5,872,000	22,600	32	65	1	..	2		Slight poikilocytosis. Some macrocytes, 1 normoblast, 1 megaloblast to 400 white cells.	Tuberculosis.
2019	16	2	45	4,700,000	21,900	67	5	26	1	1	..	Marked variation in size, shape and amount of hemoglobin in red cells. No nucleated forms.	Milk too long.
2204	11	2	20	3,083,000	23,000	55	43	0.5	1	0.5		Slight variation in size and shape of red cells. Slight polychromatophilia. 1 megaloblast to 500 white cells.	?
2608	6	3	45	2,499,000	8,000	36	60	4		Macrocytes predominate. Some microcytes. Slight poikilocytosis. Moderate achromia. 7 megaloblasts and 10 normoblasts to 200 white cells.	Improper feeding.
2899	13	2	25	1,256,000	9,000	47.5	49	..	3.5	..		No achromia. Slight poikilocytosis. 10 megaloblasts and 7 normoblasts to 200 white cells.	?
2917	11	1	30	1,296,000	7,500	69.5	28	1	1.5	..		Marked achromia and poikilocytosis. Excess of macrocytes, 3 megaloblasts and 5 normoblasts to 400 white cells.	Improper feeding. Bronchopneumonia.

TABLE 2.—ANALYSIS OF CASES OF ANEMIA WITH MARKED SPLENIC ENLARGEMENT

No.	Age in Months.	Per cent. Hemoglobin.	Red Corpuscles.	White Corpuscles.	Small Mononuclear.	Large Mononuclear.	Polynuclear Neutrophils.	Eosinophiles.	Myelocytes.	Mast Cells.	Morphologic Changes.	Etiology.
2020	16	55	6,000,000	15,000	50	6	40	3	1	..	Moderate variation in size and shape of red cells. No nucleated red cells.	?
1300	10	40	4,000,000	18,700	34	13	51	..	2	..	Marked variation in size, shape and staining reaction of red cells. 9 megaloblasts and 16 normoblasts to 500 white cells.	Gastrointestinal.
2363	20	50	4,000,000	15,000	34	4	50.5	0.5	1.5	0.5	Slight variation in size and shape of red cells. Marked polychromatophilia. 8 megaloblasts and 2 normoblasts to 200 white cells.	?
1818	21	55	3,800,000	37,600	16	5	77	0.5	1.5	..	Moderate variation in size and shape of red cells. Slight polychromatophilia. 1 megaloblast to 500 whites.	?
1775	16	25	2,392,000	34,400	55	10	35.5	1	1	0.5	Some polychromatophilia. No nucleated red cells.	Prematurity. Gastrointestinal.
2827	19	30	2,344,000	7,000	64.5	34.5	1	Marked achromia and poikilocytosis. No nucleated red cells.	Syphilis (?).
2404	14	35	1,992,000	13,400	36	4	56	2	1	1	Character of red cells? 4 normoblasts to 100 white cells.	Gastrointestinal.
1952	16	15	904,000	9,600	28	8	62	..	2	..	Watery and streaky. Considerable variation in size and shape of red cells. Marked polychromatophilia. 4 megaloblasts, 5 normoblasts and 1 microblast to 100 white cells.	Gastrointestinal.

ment with the x-ray, and the autopsy a year later showed a normal spleen and no evidences of leukemia.

The theory that anemia with splenic tumor in infancy occupies a position midway between pernicious anemia and leukemia was advanced at a time when the morphologic changes in the blood were not as well understood as at present and hardly deserves serious consideration. The postmortem appearances are not characteristic of pernicious anemia, iron is not found in the liver and

2608 a very large liver and marked edema; 2899 considerable edema; 2917 slight edema, ecchymoses and hemorrhage from the nose. The first three of these patients improved, two were discharged unimproved and the last died.

Further study shows that there may be, in some instances, extreme enlargement of the spleen with almost no changes in the blood, while in others the blood shows

6. München. med. Wehnschr., 1905, III, 904.

7. Boston Med. and Surg. Jour., 1894, cxxxi, 133; 1903, cxlviii, 573.

most marked changes. The cases analyzed in Table 2 illustrate these points. In all of these the spleen reached to the anterior superior spine, filling the flank.⁷

The liver was enlarged in 2020, 1775, 2827 and 1952, but not in the others; 1952 had edema, ecchymoses and hemorrhages from the nose. Systolic murmurs were heard in 1818 and 2827; 2363, 1300, 1818, 2355, 2827 and 2404 improved under treatment; 1300 was well and the splenic tumor was gone in eighteen months; 1775 and 1952 died.

Splenic tumor is not uncommon in infancy when there is no anemia. The anemia may improve or entirely disappear without any change in the size of the spleen. The following cases are examples:

CASE 13.—No. 2827. February 19, hemoglobin, 30 per cent.; red corpuscles, 3,344,000.

March 3: Hemoglobin, 40 per cent.; red corpuscles, 3,200,000.

March 16: Hemoglobin, 45 per cent.; red corpuscles, 3,408,000. Size of spleen remained the same.

CASE 14.—No. 1818. May 26: Hemoglobin, 35 per cent.; red corpuscles, 3,800,000; white corpuscles, 37,600.

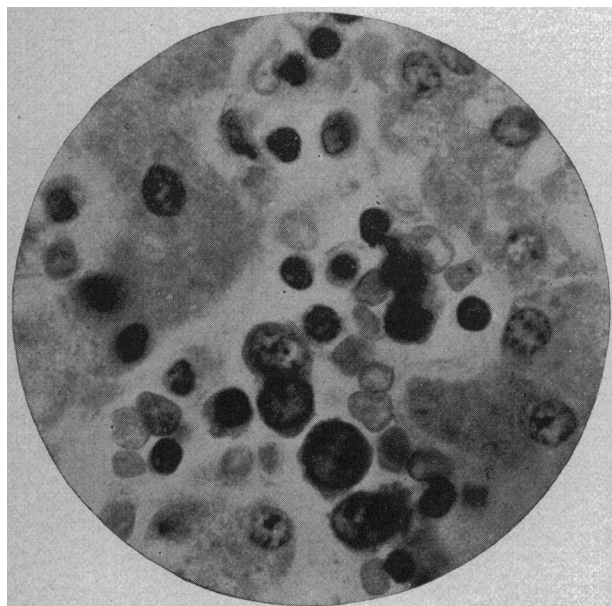


Fig. 1.—Liver (Case 12, No. 2,869) multiplied about 1,000 diameters. Hematopoietic center, showing myelocytes and nucleated red corpuscles.

June 18: Hemoglobin, 65 per cent.; red corpuscles, 5,104,000; white corpuscles, 28,800. Size of spleen remained the same.

CASE 15.—No. 2363. May 3: Hemoglobin, 50 per cent.; red corpuscles, 4,000,000.

May 20: Hemoglobin, 75 per cent.; red corpuscles, 4,800,000. Size of spleen remained the same.

The size of the spleen may diminish without any improvement in the anemia. The following case is an example:

CASE 16.—No. 3171. April 13: Hemoglobin, 60 per cent.; red corpuscles, 1,640,000; white corpuscles, 20,100. Spleen reached to navel.

April 17: Hemoglobin, 65 per cent.; white corpuscles, 40,200. Spleen about one-half as large.

April 22: Hemoglobin, 50 per cent.; red corpuscles, 1,304,000; white corpuscles, 50,600. Spleen 3 cm. by 4 cm.

The anemia responds to the same treatment and in the same way as do secondary anemias without splenic tumor.

It seems evident, therefore, that there is no relation whatever between the size of the spleen and the presence,

absence or degree of anemia. Having no causal relation to each other, they must, when associated, be due to some common cause. They are both found independently as the result of disturbances of nutrition of various sorts. The pathologic changes in the spleen are the same whether the enlargement is or is not associated with anemia. The same blood changes are found both with and without splenic tumor. These blood changes are the same as those found in the ordinary secondary anemias without splenic tumor. It seems reasonable to assume, therefore, that when they are associated they are both manifestations of disturbances of nutrition. There is, therefore, no justification for putting cases of anemia with splenic tumor in a class by themselves under the head of splenic anemia, anemia infantum pseudo-leukemica, and so on. They should rather be classed as cases of secondary anemia with splenic tumor. It may be, however, as Rist suggests, that this, although the most satisfactory, is too simple an explanation and that the future will justify some other classification.

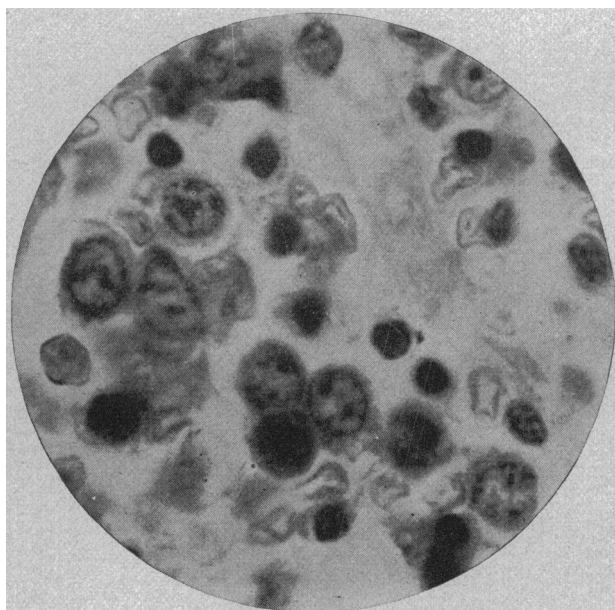


Fig. 2.—Spleen (Case 12, No. 2,869) multiplied about 1,400 diameters. Hematopoietic center, showing myelocytes and nucleated red corpuscles.

Secondary anemia with splenic tumor may or may not be associated with leucocytosis. A division into two classes on this basis, as suggested by some Italian authors (Petrone⁸), hardly seems necessary or justifiable, however. The division of the cases of secondary anemia with splenic tumor and leucocytosis into splenic anemia with myelémia, splenic anemia with lymphocythemia and even splenic anemia with eosinophilia (Labbé and Aubertin⁹), according to the predominance of the various forms of leucocytes, has been suggested. This division seems rather unnecessary and far-fetched, however, as the proportions of the leucocytes depend in these cases, as in the secondary anemias without splenic tumor, on the special chemotactic action of the causative condition.

Wolff,¹⁰ basing his opinions on an unique case of his own, has come to very different conclusions as to the

8. Arch. gen. de méd., June, 1907; abstr. in Arch. de méd. d. enfants, 1907, x, 752.

9. Rev. mens. d. mal. de l'enfance, 1907, xxv, 69.

10. Berl. klin. Wchnschr., 1906, xliii, 1565.

causation of these cases and as to the relation of the splenic tumor and the anemia. He removed the spleen from an 18-months-old baby with marked anemia. The blood before the operation showed 40 per cent. of hemoglobin, 467,000 red corpuscles and 37,800 white corpuscles. The infant gained two pounds during the next ten days and the blood then showed 51 per cent. of hemoglobin, 2,500,000 red corpuscles and 36,000 white corpuscles. The baby recovered entirely, and at the end of a year the blood contained 2,665,000 red corpuscles and 25,800 white corpuscles.

The spleen weighed 500 grams. There was marked induration due to thickening of the walls of the lacunæ of the pulp but no enlargement of the Malpighian corpuscles. There was an infiltration with large cells of various sorts free from hemoglobin but no increase of the lymphocytes. There was, however, a large number of lymphocytes in the larger blood vessels.

He believes that as the improvement in the general condition and blood followed immediately on the extirpation of the spleen that this organ stood in a causal connection, that these cases originate in a primary disease of the spleen and that they, therefore, present a completely characteristic disease picture. He explains the anemia by an increased destruction of blood corpuscles in the spleen or by the production of substances in the spleen which cause an increased destruction elsewhere.

While he does not identify this disease with Banti's symptom-complex he thinks that they have so many points in common—splenic tumor, trabecular hyperplasia of the spleen, cirrhosis of the liver, improvement after splenectomy—that there is probably some connection between them. While there are many points of difference between the two conditions, the greatest obstacle to considering them identical is the fact that in Banti's symptom-complex the course is slowly progressive to death, while very many of the cases of anemia with splenic tumor in infancy entirely recover. It is very possible, however, that while the physical signs and blood picture are essentially the same in all cases, the pathologic processes are different in different cases and that certain of them are of the same nature as Banti's symptom-complex. In such cases removal of the spleen will undoubtedly do good. The great difficulty at present is to know how to recognize these cases.

ABSTRACT OF DISCUSSION

DR. J. F. HULTGEN, Chicago: I should like to know how many children Dr. Morse examined in all and whether the children were absolutely normal or whether they were just recovering from some attack of gastric disturbance or some other condition. We know that there is a change in the blood picture following any disease, infectious or toxic, namely, a postinfective or post-toxic lymphocytosis. As to the blood picture returning to the infantile condition, I believe that Domenici of Paris invented that term and started a new line of investigation. This is the same as what happens when healing of any wound occurs. The attempt at repair of an injury is in the direction of a return to the fecal condition. As long as the body is in a vigorous condition the large mononuclear cells are increased in proportion to the degree of irritation. The predominance of lymphocytes in the hematology of the child is an exceedingly interesting and important question, one that has not been settled yet. We need young men to go into the study of hematology to work on it for years. The probability is that the lymphocytes do predominate in the normal infantile blood, but it should be demonstrated whether those children are perfectly normal at the time of examination.

Dr. Morse's statement that he sees no reason for calling "chlorosis" a number of cases that look like chlorosis at a later age pleases me very much. That the patients improve under iron is no proof. Iron is a specific for any secondary anemia. I think that chlorosis is an obsolete term and the quicker we discard it the better. As to the relation between iron and the blood picture, if we consider the nature of the ferments and proferments we can imagine that the iron excites a particular part of the hematopoietic system, whence the production of blood corpuscles. I think that Dr. Morse has slighted the eosinophiles somewhat. I think it is likely they are derived from the large mononuclear cells, but, just how, we do not know. But there is a distinct relation between the eosinophiles and digestion and there is a distinct and established relation between the infections and the eosinophiles. The eosinophiles are in the vanguard of the leucocytes. They disappear very early in infectious diseases and they stay away very long.

DR. J. H. MASON KNOX, Baltimore: That there is no definite line of cleavage between these blood conditions is most important. One thing that should be considered in determining the degree of anemia in children or others, where there has been great loss of weight, is the concentration of the blood. Such children have anemia, but have a high percentage of hemoglobin and a large number of corpuscles, as determined by the usual method, because of the great loss of fluid. In such cases the determination of the specific gravity of the blood would be helpful in judging the degree of anemia.

DR. JOHN LOVETT MORSE, Boston: My figures regarding the normal blood in infancy were based partly on my own observations and partly on the literature. I have been working on this subject for the last fifteen years and think that I have the cases to prove the points which I have made. The hemoglobin and the specific gravity go hand in hand. What the last speaker said about the concentrated blood in these babies is perfectly true. One has to guess at the degree of anemia in such cases.

Clinical Notes

OVARIAN CYSTOMA OBSTRUCTING PELVIS AT TERM

WITH RECOVERY AFTER CESAREAN SECTION

A. BELCHAM KEYES, M.D.

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Patient.—Mrs. C. F. H., aged 33, German, primipara, married. Since puberty the menses had been regular (every 28 days) till pregnancy began. The last menstrual period (remembered) began about August 25 and supposed quickening was first noticed about January 18.

Labor.—The patient entered hospital in labor June 19, 1908, complaining of having had violent labor pains for fifty-three hours. The uterine contractions were strong and rapidly increased in frequency to one in every two minutes. Abdominally the child's lower limbs could be distinctly palpated near the fundus uteri. The child's head was easily palpated above the pelvic brim and apparently not larger than the average. The fetal heart tones were from 180 to 200 and weak. The external pelvic measurements of the mother were normal. Vaginally the cervix uteri was found dilated enough to admit two fingers easily. The head was high up and could be reached with difficulty, position left occipito-anterior. The bag of waters was still intact but accidentally ruptured during the examination, resulting in the discharge of the liquor amnii with much meconium. The posterior vaginal fornix was bulged forward by something giving at first the impression of a loaded sigmoid and rectum, though it had not the doughy feel of feces but rather the elastic sensation of a cyst. So far as one could judge, the internal pelvic measurements were normal, though the promontory was covered by the