ing repeatedly with alcohol and ether, the rats gained for a very short time or not at all, the cessation of growth occurring without evident ophthalmia. In all cases, if loss of weight on diets low in the A factor was allowed to proceed longer than a week, pulmonary affections, sore feet, diarrhea and extreme emaciation followed, and death could not be prevented by feeding vitamin A. It is important, in such feeding experiments, to give the source of vitamin A when the animal has merely ceased growing on the vitamin-low diet rather than after decline in weight has definitely set in.

Mendel³ has reported that 100 mg. of butter fat daily will provide fat soluble vitamin for growth to maturity of a rat on otherwise adequate diet. In the present series of experiments, 1 gm. and 0.5 gm. a day of the unfrozen and of the frozen ice cream, respectively, were fed in different experiments. When 1 gm. of ice cream (100 mg. of butter fat) furnished the sole source of vitamin A, normal growth was obtained. The same was true of the unfrozen mixture. When, however, 0.5 gm. of ice cream (50 mg. of butter fat), either frozen or unfrozen, was fed, subnormal growth was obtained. Here, then, is evidence that freezing has no effect on the growth-promoting power of the fat soluble vitamin and, basing our statement on Mendel's figures for butter fat, the preliminary pasteurization of the unfrozen mixture at 180 F. for one-half hour had no marked deteriorating effect on vitamin A in the mixture.

Rats, with ophthalmia of varying degrees of severity, on a diet low in fat soluble vitamin were cured by 1 gm. daily of either the unfrozen or the frozen ice cream. The ophthalmia in two rats was cured with as little as 0.25 gm. of frozen ice cream a day, which represents 25 mg. of butter fat. Growth was not obtained with this quantity of ice cream, however, until the eyes were entirely clear, and then the animals grew only at a subnormal rate.

VITAMIN B

Milk has not been considered a rich source of the water soluble vitamin. American investigators have never been able to duplicate the results with the small dosages found adequate by Hopkins.⁴ Osborne and Mendel⁵ obtained normal growth to maturity with 16 c.c. of fresh milk as a minimum. In ice cream, the milk and possibly the eggs used constitute the source of vitamin B. In the present series of experiments, 7.7 gm. of either the unfrozen or the frozen ice cream (equivalent to 15 gm. of milk) was the minimum satisfactory amount for normal growth, and some rats required 10.2 gm. (equivalent to 20 gm. of milk). Doses of both the unfrozen and the frozen ice cream equivalent to 5 and 10 gm. of milk were found to be distinctly inadequate as a source of vitamin B for inducing normal growth.

VITAMIN C

The potency of milk as an antiscorbutic varies with the feed of the cows and the treatment of the milk. The obscure history of the milk used in commercial ice cream and the pasteurization of the mixture raises doubt as to the value of the product as an antiscorbutic. Again, the high calorific value of the ice cream mixture prevents the ingestion of the large amount theoretically necessary even when resorting to forced feeding. Doses equivalent to 60 c.c. of milk were given, but as soon

as scurvy developed the guinea-pigs refused to eat this amount. Guinea-pigs on the soy bean diet usually showed the first clinical signs of scurvy in from fourteen to sixteen days. When the animals were forced to eat the ice cream, the first appearance of the disease was delayed for one to seven days. Apparently, the animal was sustained by the forced feeding long enough for severe scurvy to develop; for classic clinical and postmortem pictures of the disease were obtained in practically all of the animals. Lack of appetite, lethargy, bloody diarrhea, prolapse of the rectum, sore joints and muscles, costochondral enlargements, sore eyes and loose teeth were observed, and on necropsy, extensive hemorrhages under the muscle sheaths and in the joints, gums and costochondral junctions.

Neither frozen nor unfrozen ice cream in the maximum doses possible caused more than the slightest delay in the appearance of scurvy. In no case was protection afforded.

SUMMARY

Vitamin A was present in the typical samples of ice cream in such concentration that one may conclude that no noteworthy alteration in its potency is caused by pasteurizing or freezing. Normal growth was induced by 1 gm. of the ice cream, and ophthalmia was cured by 0.25 gm., containing 25 mg. of butter fat.

The vitamin B of the ice cream can be accounted for by the equivalent quantity of milk used in it. Freezing had no effect on the vitamin B in the ice cream used. The ice cream, which was made from pasteurized products, contained no significant quantity of vitamin C.

INFLUENCE OF DIET ON BLOOD GROUPING *

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Since their discovery by Landsteiner and Shattock, working independently in 1900, isohemagglutinins have been studied extensively by Hektoen, Ottenberg, Moss, Jansky, Gay, Brem and many others. Their practical application in blood transfusion requires no comment here. During this time, the investigators have uncovered many interesting and practical facts in regard to the character and mechanism of these isohemagglutinins. Among the influencing factors may be mentioned heat, desiccation and disease. No reference in regard to diet as a definite factor in this respect has been noted.

We believe that diet does play a part in influencing isohemagglutinins, especially in Group III blood, and that it should be taken into consideration to avoid untoward results. The question of diet was brought to our attention in the following manner: This laboratory, like others, keeps on hand for blood grouping purposes the serums of Groups II and III. The serums are obtained from the laboratory personnel. Occasionally, difficulty was experienced in securing a Group III serum that was high in isohemagglutinin or was of good keeping quality, notwithstanding the fact that the same blood had yielded an excellent serum on a previous occasion.

* From the U. S. Naval Medical School.

3. Mendel, L. B.: New York State J. Med. **20**: 212 (July) 1920.

4. Hopkins: J. Physiol. **44**: 425, 1912.

5. Osborne, T. B., and Mendel, L. B.: J. Biol. Chem. **34**: 537 (June) 1918.

Then, again, it was noticed that, in attempting to group such a blood, unsatisfactory results were encountered: the cells of this Group III blood would not properly agglutinate with a definite Group II serum; so that, unless one was alert, a Group III would be classified as a Group I (Jansky) or a Group IV (Moss). This difficulty was never experienced with groups other than Group III. Several instances occurred in which a donor, classified under Group I (Jansky) or Group IV (Moss) was subsequently

TABLE 1.—ORDINARY DIET

	Group II (Serum)	Group III (Serum)
Group I (Cells) ..	A. No agglutination	No agglutination
	B. No agglutination	No agglutination
	C. No agglutination	No agglutination
Group II (Cells) ..	Group II (Serum)	Group III (Serum)
	D. No agglutination	Strong agglutination
	E. No agglutination	Strong agglutination
Group III (Cells) ..	F. No agglutination	Strong agglutination
	Group II (Serum)	Group III (Serum)
	G. Weak agglutination	No agglutination
	H. Weak agglutination	No agglutination
	I. Weak agglutination	No agglutination

TABLE 2.—GREEN-FREE DIET

	Group II (Serum)	Group III (Serum)
Group I (Cells) ..	A. No agglutination	No agglutination
	B. No agglutination	No agglutination
	C. No agglutination	No agglutination
Group II (Cells) ..	Group II (Serum)	Group III (Serum)
	D. No agglutination	Strong agglutination
	E. No agglutination	Strong agglutination
Group III (Cells) ..	F. No agglutination	Strong agglutination
	Group II (Serum)	Group III (Serum)
	G. Very weak agglutination	No agglutination
	H. Weak agglutination	No agglutination
	I. Very weak agglutination	No agglutination

TABLE 3.—GREEN DIET *

	Group II (Serum)	Group III (Serum)
Group I (Cells) ..	A. No agglutination	No agglutination
	B. No agglutination	No agglutination
	C. No agglutination	No agglutination
Group II (Cells) ..	Group II (Serum)	Group III (Serum)
	D. No agglutination	Strong agglutination
	E. No agglutination	Strong agglutination
Group III (Cells) ..	F. No agglutination	Strong agglutination
	Group II (Serum)	Group III (Serum)
	G. Strong agglutination	No agglutination
	H. Strong agglutination	No agglutination
	I. Strong agglutination	No agglutination

* Jansky classification is used. Group IV (Jansky) or Group I (Moss) is not available. The technic is essentially that of Lee and Minot as described in "The Principles of Immunology" by Karsner and Ecker. Letters A, B, C, etc., refer to men of each group.

found to belong to Group III. The error was detected by a direct match with a recipient's blood which was a definite Group I (Jansky) or Group IV (Moss). When this occurred, inquiry always revealed that the Group III subject had been on a diet insufficient in greens. This led to a more or less empiric policy of placing men of Group III on a diet rich in greens before using their serums for grouping purposes. The results obtained were excellent.

In order to arrive at a definite conclusion, three men each of Groups I, II and III (Jansky) or IV, II and III (Moss) were grouped and placed on a green-free diet for a period of four days, and again grouped. These men were then placed on a diet consisting of a large quantity of greens for four days, and grouped again. The results are shown in the accompanying tables.

It will readily be seen that the grouping of the Group III Cells was influenced to a remarkable degree. It cannot be said, however, that there was a definite change of group.

SUMMARY

1. Diet apparently influences the serum of Group III blood.
2. Diet apparently interferes with the proper agglutination of cells of Group III blood in Group II serum.
3. As we were handicapped by lack of sufficient material, the series is, as we realize, entirely too small to form a basis for definite conclusions. However, we consider these findings sufficiently practicable to stimulate investigation along this line.

THE DIAGNOSIS OF EPIDEMIC
ENCEPHALITIS *

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Epidemic (lethargic) encephalitis is one of three important inflammatory, nonsuppurative diseases of the central nervous system which have certain features in common. Each regularly, at least in the more severe cases, affects to some degree the whole cerebrospinal axis and its meningeal coverings; but there is nearly always a maximal involvement of some particular portion which determines the principal symptomatology in any given case. The other two diseases to which I refer are acute poliomyelitis and syphilis. To distinguish the three diseases from one another and from other diseases, it will be helpful to bear in mind a few of their salient pathologic features.

POLIOMYELITIS AND ENCEPHALITIS

Poliomyelitis and encephalitis both affect the interior of the nervous organs more than the meninges, and gray matter more than white matter. In both we have collars of inflammatory cells about blood vessels; but neither tends to cause occlusion of the blood vessels. The meningeal inflammation is slight and only microscopic in both, but more marked in poliomyelitis; hence the greater tendency to meningitic signs, such as Kernig's sign and stiff neck and back, in poliomyelitis. Each disease really is poli-encephalomyelitis, but one principally affects the brain and the other principally the cord. Hence there is little difficulty in distinguishing between the two if we have a pure brain picture or a pure cord picture.

There is a place, however, where brain and cord meet, namely, the medulla oblongata; and with an acute inflammation there, producing an acute bulbar paralysis, we are often at a loss when it comes to making a diagnosis. If the patient should live long enough, the appearance of atrophy, reaction of degeneration, and residual flaccid paralysis of the affected muscles would almost settle the diagnosis in favor of poliomyelitic infection, as the less severe inflammation caused by the encephalitic virus rarely causes sufficient destruction of ganglion cells to produce these sequels.¹ But the patient usually dies in a few days, and as there are no

* Read before the Englewood Branch of the Chicago Medical Society, Nov. 7, 1922.

1. However, localized degenerative muscular atrophy in an unusual case of epidemic encephalitis also complicated by optic neuritis has been described by Julius Grinker: *J. Nerv. & Ment. Dis.* 52: 323 (Oct.) 1920.