TABLE 1

OBSERVED INDIGENOUS NATIVE POPULATION OF ALGERIA AND FITTED LOGISTIC CURVE

Year	Observed populations	Calculated populations from logistic curve	Percentage deviations of calculated from observed values
1881	2,842,497	2,962,000	+ 4.05
1886	3,287,217	3,224,000	— 1.95
1891	3,577,063	3,529,000	— 1.36
1896	3,781,098	3,859,000	+ 2.02
1901	4,098,355	4,184,000	+ 2.06
1906	4,477,788	4,478,000	± 0
1911	4,740,526	4,723,000	— 0.38
1921	4,924,938	5,060,000	+ 2.67

nous native population in that year was in total, 5,192,426.

The logistic curve shown in equation (i) was calculated on the basis of the data up to and including 1921. Extrapolating that curve it gives for the expected or probable magnitude of the indigenous native population in 1926 the value 5,162,000. This underestimates the population actually observed in 1926 by 30,426. This is a percentage error of only −0.59 per cent. To miss by just over one half a man in each hundred counted is certainly not a serious discrepancy. Probably few demographic experts would care to assert that the error made in counting a population, however highly civilized, is less than six tenths of one per cent.

The chief point of general significance in this result is that it makes still more valid the case of the native population of Algeria as an example of a human population following the logistic curve in its growth.

In closing I should like again to emphasize, as has been done repeatedly in what I have written on population growth, that the data in hand permit no prediction as to whether the native population of Algeria (or any other population) will in the future continue to follow its past logistic curve in its growth. All that the logistic theory of population growth is capable of saying on the point is that this result is to be expected only if the same forces, economic, social, geographical and possibly other, which have influenced the birth and death rates during the past history of the population continue to operate unaltered in the future, but not otherwise. If any or all of these factors undergo any considerable alteration in the future the course of population growth may be expected to depart from the particular logistic curve which it has followed hitherto.

RAYMOND PEARL

SCIENTIFIC BOOKS

Recent Advances in Haematology. A. PINEY, M.D., P. Blakiston's Son & Co., Philadelphia, Pa, 1927.

THE author of this welcome and timely book, dealing largely with clinical hematology, very properly bases his interpretations of blood pathology on the data of normal blood development and those of the normal histology of the several hemal formative organs, yolk sac, liver, spleen, lymph nodes and bone marrow. This is an approach to an understanding of the leucemias and primary anemias that seems to offer the most promising results. In future a still more adequate attack will be made by way of the evolutionary history of blood and the hemopoietic tissues. Information already at hand emphasizes the vastly greater genetic significance of the lymphocyte than is now generally accorded this hemal outcast. For a work of this sort, properly a fairly inclusive digest, the discussions and interpretations seem dominated unduly by a personal working hypothesis, namely, the assumption of the exclusively entodermal origin of the earliest embryonic red blood cells and the occasional anomalous persistence of this entodermal blood primordium in postnatal life.

The book includes 249 pages of text, subdivided into 14 chapters. Chapter I gives an account of the reticulo-endothelial system. In Chapter II the blood forming function of this system is discussed. Chapter III on "Leukaemia," and Chapter IV on pernicious anaemia, constitute in our opinion the most interesting portions of the book. Chapters V and VI deal with leukaemoid blood pictures and reactions, Chapter VII with reticulo-endotheliosis, Chapter XI with Gaucher's disease and Chapter XII with Banti's disease. There is an important appendix of 11 pages on hematological technique and a selected bibliography of 73 titles, with author's comments. In addition the book contains four plates of very excellent colored illustrations, 23 in number, and 18 text figures.

As regards the colored illustrations the reviewer, while admitting their artistic beauty and detailed accuracy, feels compelled to point out certain disconcerting omissions and seemingly forced interpretations. In the first place, one feels greatly handicapped in estimating the value of these illustrations as supporting the text by reason of the lack of any designation of either absolute or relative magnification. This is all the more serious in view of the great significance ascribed by Piney to his so-called megaloblast. In the next place one is not convinced on the basis of the illustrations that the so-called promonocyte is more closely related to the myeloblast

than to the lymphoblast. Then, too, the lack of illustrations of transition stages between the large lymphoblast and the small lymphocyte, and especially the absence of any illustrations of a large lymphocyte, seems unfortunate. Furthermore, the eleven figures grouped under the name "megaloblast" are so very different in nuclear configuration and cytoplasmic staining reaction, that they can not with any propriety or accuracy be designated by a common term. If the relatively larger size of these pro-erythroblasts is to be emphasized, surely Figures 9 and 10 are more properly called macro-normoblasts, a term improperly applied to Figures 12 and 14. These illustrations by no means support the claim that the alleged entodermal pro-erythroblasts differ specifically from the mesodermal pro-erythroblasts by the absence of a "cart-wheel" arrangement of the nuclear chromatin (Compare Figures 7 and 15).

In Chapter III, Piney stresses the importance of regarding leucemias as diseases of the hemopoietic tissues, rather than as "blood diseases." He accepts Orth's suggestion to designate all conditions of hemopoietic excess, usually accompanied by alterations in the circulating blood, as *haemoblastosis,* in essence a neoplastic condition. Such hyperplasias may be restricted to the leucocytopoietic tissues or to the erythrocytopoietic tissues. The former condition may be designated *leucosis* ("leukemia"), the latter *erythrosis.* When the leucosis affects only the myeloid tissue, the condition becomes a *myelosis;* restricted to the lymphoid tissue, it constitutes a *lymphadenosis.* Either condition may be acute or chronic, leucemic or aleucemic. The excessive number of red corpuscles in the circulation in the condition of erythrosis is termed *erythraemia* (polycythemia). A concomitant hyperplasia of both the erythrocytopoietic and leucocytopoietic tissue, Piney terms *erythro-leucosis,* or synonymously as *panmyelosis.* Hyperplasias of the ancestral reticuloendothelium may also occur as variations of hemoblastosis. Such conditions are designated *reticuloendotheliosis,* the leucemic variety possibly constituting monocytic leucemia, the aleucemic possibly Hodgkin's disease.

The central and guiding concept in Piney's interpretation of hemoblastosis relates to the so-called "megaloblasts." In current hematological literature the term "megaloblast" is definitely restricted to designate the earliest transition stage between the ancestral hemoblast and the hemoglobiniferous normoblast. This cell is characterized specifically by the presence of a minute amount of hemoglobin in its otherwise basophilic cytoplasm, imposing a slight acidophilic staining reaction in polychrome stains, *e.g.,* Giemsa. In addition, it has a slightly larger size than the later generations of red cells (normoblasts and erythroblasts) and its nucleus generally lacks the nucleoli of the hemoblasts, and the basichromatin approaches the relatively coarse reticular condition of the normoblast. Piney, quite arbitrarily it would seem, applies the term "megaloblast" to a large cell of the hemoglobiniferous series whatever its stage of development. He uses the term in its strict etymological sense to denote a large maturing red cell, ancestor of an anucleate "megalocyte." Very much more important, however, is his conception of the *origin* of the "megaloblast"; he derives it exclusively from the *entoderm.* The megaloblast of Piney is therefore an entodermal element, and occurs normally only in the embryo. He says, "The embryo is thus supplied, for a short time, with two varieties of blood; one derived from the entoderm, and composed purely of hemoglobiniferous cells; and the other of mesenchymal origin, and consisting of both red and white cells"(p. 19). This supplies the basis for Piney's interpretation of various leucoses in which megaloblasts appear in the blood. It constitutes his chief contribution to the question of the etiology of pernicious anemia. The appearance of megaloblasts in postnatal life he regards as always indicative of the persistence of some remnant of entodermal blood primordium. He assumes further, that if this hemic primordium atrophies only incompletely in postembryonic life, there occurs a concomitant defective development of the mesodermal myeloid tissues. Pernicious anemia is interpreted as a condition in which, following a hereditary defect inherent in a hepatic persistence of entodermal hemocytopoietic tissue, there occurs correlatively a poor quality of bone marrow. Naegeli had already pointed out that the presence of megaloblasts (mesenchymal ancestors of hemoglobiniferous megalocytes) in the blood is pathognomonic of pernicious anemia. But Piney interprets this datum in a unique sense; he ascribes to these cells the significance of specific entodermal derivatives.

Investigations on the origin of the initial red blood cells in the yolk sac of mammals and other vertebrates during the past twenty years give no support to the idea of an entodermal origin of these cells. From beginning to end, blood arises only from mesenchyme, the reticulo-endothelial system of various myeloid and lymphoid organs. An alleged exception is described in the papers by Havet[1] and Aron,[2] who claim that

[1] Havet, J., 1926. L'origine des cellules du sang. *Jour. Anat.* Vol. 60, pp. 231–258.

[2] Aron, M. Quelques observations novelles a' propos de l'origine du sang dans le foie embryonnaire des mammiferes. Arch. d'anat. d'Hist. et d'Embryol. T. 4, pp. 1–26.

in young mammalian embryos (rabbit, sheep, pig, guinea pig, and human) erythrocytes develop directly from hepatic parenchymal cells. These recently recorded results, however, lack confirmation, and the supporting illustrations admit of an alternative interpretation.

It is well known that in pernicious anemia generally the red corpuscles are of larger average size, thus determining a color index above unity, and that megaloblasts (initial stages in red cell formation) occur; but the relatively larger size of both may be quite as well, and much more likely, the result of an absence of normal proliferative activity of these proerythroblasts, as of persistent entodermal ancestors. The familial incidence of pernicious anemia may equally plausibly be explainable on the basis of a hereditary susceptibility to factors that affect the bone marrow, and other possible erythrocytopoietic foci, in such a way as to produce a condition characterized by relatively low proliferative activity among the ancestral cells, resulting in the appearance of many relatively large red cells, both mature and immature.

Another interpretative conclusion, in conflict with practically all recent work, relates to the restricted ancestry of the monocytes. According to Piney, monocytes are derived from the reticulo-endothelial system exclusively through the myeloblasts, a monocyte being "as much a myeloid cell as is any other granular leukocyte."

Though emphasizing the alleged distinctive character and apparent independence of the lymphatic and myeloid tissues in many respects, Piney is forced to admit certain obvious interrelationships as indicated in certain infections where lymphocytosis coexists with a neutrophilia, following a leucopenia during the height of the infective process. A probable explanation seems to be at hand in the repeated demonstrations that lymphocytes may develop into granulocytes. At the height of the demand for neutrophils the lymphocytes may be very rapidly converted, effecting a resulting lymphocytopenia, which may be later overcorrected through an attempt at compensatory readjustment of normal numerical relationships. Similarly the anemia almost invariably accompanying leucemia might be interpreted in terms of the limited availability of the common ancestor (the lymphoid hemoblast) for erythrocytes, granulocytes and monocytes. When the specific stimuli are such as to demand an excess of one of the derivatives, the other possible differentiation products may be reciprocally, at least temporarily, reduced in numerical proportion. This differs from Piney's explanation that since (as he claims) erythrocytes arise only intravascularly, and leucocytes extravascularly, specific morbid stimuli may reach one surface of the common blood cell primordium without affecting the opposite surface.

H. E. JORDAN

UNIVERSITY OF VIRGINIA

DOES THE AMOUNT OF FOOD CONSUMED INFLUENCE THE GROWTH OF AN ANIMAL?

THE question to which this discussion is addressed might well be considered an impertinent one to raise in a scientific journal were it not for the fact that a number of research laboratories in this and other countries appear to be attacking many important problems in nutrition by methods involving the tacit assumption that *the composition of the diet alone* determines the changes in body weight secured in experimental animals. When stated in such plain terms as these, the situation, if it actually exists, would appear to be sufficiently serious to justify general consideration and discussion, to the purpose that the time and energy being devoted to nutritional problems should yield the greatest return in unequivocal experimental evidence.

Obviously, the point of first importance is to determine whether the situation characterized above actually exists. This characterization may be illustrated in an abstract way as follows. It is desired to determine whether a given food material is deficient in a certain dietary essential A. Accordingly, two rations are made up, including the food material in question supplemental with adequate percentages of all known dietary essentials except A. In one ration, A is included in place of an equal percentage of a dietary diluent such as starch, while in the other ration the food itself serves as the sole source of A. Now, it is the general plan to feed ration No. 1 to one group of animals, and ration No. 2 to another, the food in each case being offered *ad libitum*. The changes in weight of the animals are then followed by periodical weighings. In some cases, records of food consumed may be kept and, in fewer cases, such records may be reported, but in the great preponderance of cases, no *control* of food intake is imposed, and the weight curves are interpreted with reference only to the composition of the rations.

Is not this type of interpretation tantamount to assuming that the amount of food consumed has no influence on growth? The assumption itself is obviously untenable, and the interpretation can be defended only on the very tenuous supposition that the