varies from 10,000 to 25,000, and is continuous for some time after birth.

Leucocytosis due to thermal and mechanical means.—After hot and cold baths, massage, muscular exercise not resulting in fatigue, a transient increase in the number of white blood cells is produced. I merely mention this as a factor to be guarded against in determining the significance of a leucocyte count.

Pathological Leucocytosis (Post-hemorrhagic leucocytosis).—Immediately after an acute hemorrhage a slight diminution in number is found to be followed in a few minutes by a well-marked polynuclear leucocytosis; this is to be found in the pulmonary hemorrhage of phthisis, hemorrhage from cancer of the uterus, ulcer of stomach, hemorrhage from the bowels. In a few isolated cases no leucocytosis is obtained, but in such cases a differential count shows a lymphocytosis to be always present. In general the leucocytosis following hemorrhage is in proportion to the extent and rapidity of the loss of blood (Ewing).

Cachectic Leucocytosis.—In cachectic conditions we often get a more or less marked increase, but in the majority of these cases this can usually be traced to some of the other casual factors of leucocytosis, namely inflammation and hemorrhage.

Drug Leucocytosis.—The administrations of drugs produces yet another point to be noted in the interpretation of leucocytosis; such drugs as quinine and atropine produce a slight diminution in number, whereas antipyrin, antifebrin, pilocarpine and probably morphia, produce a considerable increase. Again, irritants, such as free acids and alkalies, produce moderate leucocytosis, whereas vesicants, as copper sulphate, silver nitrate and mercurials, produce a fairly marked increase. Some have attempted to further increase toxic and infectious leucocytosis by the injections of drugs for increasing the number of the polynuclear white blood cells, expecting thereby to increase the resistance of the patient, but their efforts along such lines have not been very satisfactory.

Toxic Leucocytosis.—Examples of this class of leucocytosis are poisoning by ptomaines and coal gases, together with chloroform and ether narcosis, convulsions, acute delirium, and probably uremia may be put in this class. Metabolic products are, doubtless, the active toxic agency in this class of leucocytosis. In acute gas poisoning we get a marked leucocytosis of the polynuclear type. This increase is not due to peripheral stasis, as we were able to show in the study of this condition in the guinea-pig. In chronic gas poisoning a fairly well-marked increase