4. They have what is known as a chemotactic function; so that the leukocytes thus play a considerable rôle in the morphologic contexture, as well as the chemistry of the body.

The French supporters of the germ-theory of disease, believe that when microbes have penetrated the organism, the leukocytes increase; that the conditions that bring them to the blood carry them to the point of excitement, and that it is a chemical property that draws them there. This, they claim, is true of all high-grade inflammations, as pneumonia, but not of the low grade, as malarial fevers.

In pneumonia there is an increase of leukocytes during the height of the disease, but as the fever abates the number of leukocytes diminishes. It is, therefore, held that the real termination of the disease, whether by crisis or lysis, occurs at the time when the leukocytes begin to diminish in the blood.

In acute inflammation there are three stages of development: First, vascular dilatation; second, activity and proliferation of endothelium; third, exudation, with diapedesis of leukocytes. As a consequence of these three stages it is claimed that a great afflux of phagocytes takes place toward the point of attack, both in purulent as well as in catarrhal inflammations; it is less seen in serous varieties, and, perhaps, not at all in infectious processes, the reason for this being that the infectious matters destroy the phagocytic function of the leukocytes, and hence the body has no protecting element against the enemy, and becomes the prey of the microbe. Supuration is considered no exaggeration of inflammation, but is primarily due to the action of streptococci or staphylococci, and although great numbers of phagocytes may be found, yet their defensive action is much harassed by their deadly enemy, the pus-germs.

From the foregoing, if we are to accept this doctrine, we may conclude that inflammation is a physiologic process to develop phagocytes for the purpose of antagonizing microbes.

Anatomically the pleura is a great lymphatic sac, contiguous with the arterio-pulmonary system, and naturally derives its serosity from that source.

According to the eminent French teacher, M. Guerin, "pleurisy is nothing else than lymphangitis." "If one injected the pulmonary artery," says Guerin, "with colored liquid, it would be found that the liquid would appear in the polygonal ramifications of the lymphatics of the pleura."

Moreover, he has practised on the cadaver the injection of bullock's blood in the same manner, and finds that he obtains a serosity from the pleura which, if much force is used in the process of injecting, becomes bloody, or red in color. This is on the healthy lung and pleura. If, on the other hand, he injects a colored liquid in a subject that has suffered and died from pleurisy, there will be found no coloring-matter in the meshes of the lymphatics except those that have not been affected by the inflammatory process. If this be true, we can readily see that when from some cause the lymphatics of the pleura become congested and swollen, and the natural channels for the lymph impeded, an edematous condition of the subserous connective tissue will arise, due to forced diapedesis of the serous exudate, and cause the fluid to ooze from all parts into the cavity.

If, then, the lymphatic stoppage is complete enough, and the force behind is strong enough, there will also be more or less diapedesis of red blood-corpuscles, and hence the hæmorrhagic pleurisy. If we find leukocytes in the lymphatic exudation, does that not best explain the formation of pus? It has been stated that the serous exudate of acute pleurisy does not differ materially from the plasma of the blood. (The reason for this appears plain if we consider the contiguity of the arterial capillaries with the lymphatics.) If drawn off it will coagulate, often spontaneously; if beaten or whipped, it will show a deposit of fibrin; and the constituents are the same as the plasma of the blood, except in relative proportion.

It often happens, when one is practising puncture to draw off the effusion of acute pleurisy, that the needle, if small, becomes clogged with fibrinous flocculi. From this fact, it is argued by M. Lancereaux, that these flocculi, and more especially the finer ones, become obstructed in the openings of the lymphatics and produce a mechanical hindrance to the exit of the effused fluid by the thrombus thus formed. When this has occurred to such an extent that it impedes the absorption of liquid beyond the natural time required for the evolution of the disease, he avers, "one must wait for the disintegration and absorption of the clot before any diminishing of the quantity of the fluid by nature takes place."

Secondary pleurisies, or those acute attacks from some pre-existing disease which often occur, doubtless present in many cases the pathologic lesions in the pleura upon which such disease depends; but I can see no reason for the opinion of some authors, that every pleurisy is dependent on lesions of pre-existing maladies, and especially tuberculosis.

Etiology.—In considering the causation of acute pleurisy, one must of necessity admit that there are two general classifications from an etiologic point of view:

a. Those that are not due to any pre-existing disease.

b. Those that are so due.

At the present day there are many and diverse opinions held by eminent medical men as to the factors that produce primary idiopathic pleurisy. There are, indeed, those who would go so far as