to say that all pleurisies are tuberculous in origin, and, hence, secondary.

Undoubtedly, many pleurisies deemed primary are in reality dependent on some complicating or preëxisting disease; but to say that a person must be weakened by constitutional maladies in order to become privileged to have pleurisy, seems as if we were carrying the causative factors beyond the point warranted by our pathologic research. I doubt not, if the chests of many persons who to-day are in good health and have never been cognizant of pleurisy were opened, we would find various traces of old adhesions and other ancient lesions of former pleurisies.

It must, therefore, be admitted, even at the present day of biologic research, that the etiology of acute pleurisy is often obscure: the microbe will not account for all the cases; neither will any other one causative factor. While it is difficult to state with certainty that pleurisy originates in perfectly healthy persons, because latent pathologic lesions cannot be appreciated, yet we know that it does occur in persons, who to all appearances, to themselves and others, are in good health. M. Sée maintains that the etiology of acute pleurisy is always microbic; in fact, that it is a bacterial disease; cold is simply a stimulus to the activity and development of the microörganisms.

M. Jaccoud is of the opinion that in the human body many kinds and many thousands of microorganisms live in peace and harmony together so long as the functions are normal, but let a disturbing element arise, such as taking cold, and their physiologic relations become altered, so that they soon are hostile to each other and cause disease. Netter claims that all forms of pleurisy are of microbic origin, but that the microbes producing them are of many different kinds.

M. Bechamy says: "Microbes do not have so much importance in acute pleurisy as some would have them." He is certain that pleurisy may exist independently of tuberculosis, from the fact that at the age of thirty he was seized with acute pleurisy, and after the usual bleeding, blistering, and purging, he now, at the age of seventy-six, is still alive, and has not developed tuberculosis. In support of this, both he and Dieulafoy state that, in many cases, persons live ten, fifteen, and more years after the operation of thoracentesis, and do not develop tuberculosis.

That exposure to cold has a tendency to excite inflammations, and with them acute pleurisy, is no doubt true. Whether its influence is exerted through the nerve-centers, so as to directly cause pleurisy, or whether it acts simply as a stimulus to organized germs through whose activity the disease originates, is still a much-mooted question. M. Tresbot does not doubt that acute pleurisy in horses is the direct result of an exposure to cold, especially when, after a long, hard drive the animal is allowed to stand unprotected and exposed to a chilling wind. He says that "ordinarily there is nothing in common between sero-fibrinous pleurisy in the horse and tuberculosis"; and also that "it is impossible to class sero-fibrinous pleurisy in the horse with an erruptive fever, or, indeed, with any periodic disease." On the other hand, M. Lancereaux asserts: "Pleurisy should be rightly classed among the infectious maladies, and exposure to cold is nothing but an occasional exciting cause, while the action of the infecting agent still escapes us."

If we should accept the pathologic views of M. Guerin, we might easily explain the causation of the congestion of the blood-vessels by peripheral irritation, and reflex action of the vasomotor nerves from exposure to cold. This, however, does not explain why such excitement should be directed particularly to the pleura.

In a paper on "The Cause of Syncope in Pleurisy," M. La Borde has illustrated by experiment on animals that it is possible to produce a sero-fibrinous pleurisy in a few hours by the action of cantharidin injected into the blood. This leads me to ask the question, Is the causation of acute primary pleurisy ever, in a measure, governed by the ingestion of certain articles of food, taken just previously to an exposure to cold, *i. e.*, is the combination of the two forces sufficient to direct the action of inflamation toward the pleura?

The cause of acute purulent pleurisy is probably of microbic nature. Purulent pleurisies either begin as such, or are secondary to other diseases. Age and debility have much to do with the formation of pus, the young and aged being more susceptible to empyema. It is doubtful if simple sero-fibrinous pleurisies are ever transformed into the purulent form without the aid of outside interference.

Secondary pleurisies occur from a variety of causes, mostly from diseases microbic in origin.

While it may be rare to have pneumonia occur without some localized extension of inflammation to the pleura, it is doubtful if a general acute pleurisy, secondary to pneumonia, can be found with numerous pneumonia-cocci. In the same manner, the causation of pleurisy during an attack of typhoid fever, influenza, malarial fever, rheumatism, and kindred diseases, is undoubtedly due to the same influences that govern the coëxisting disease.

It is well known that pleurisy of an acute type may be secondary to tuberculosis; but there is considerable difference of opinion as to the proportion of cases arising from this source. M. Sée claims that 68 per cent. of all pleurisies are due to this cause. This seems high, or else persons radically recover from tuberculous pleurisy more often than from any other form of tuberculosis. Dr. G. G. Sears reports four hundred and fifty cases of