a matter of no account; so long as breathing was continued through the nose the particles of the air were retained in the nose and nasopharynx, and the lungs remained free. Nor is this to be expected otherwise. Imagine for the sake of simplicity, the air current passing in a straight direction toward the retropharyngeal cavity. Here it turns at an acute angle, and it would be more than peculiar if the bacteria would not be held there by the moist mucosa. On the contrary, one must suppose that the retropharynx keeps back the bacteria to a greater extent than the nose itself. But also deeper down the air current is deviated at the entrance into the larynx by the various prominences, such as the epiglottis, arytenoids, etc., and again, and more especially at the bifurcation of the trachea."

There is still another factor that makes the retropharynx the locus minima resistentia. The different kinds of bacteria, especially the cocci, according to the experiments of Babes, are prone to prepare the soil for the tubercle bacilli. Thus, this factor also facilitates the entrance of the tubercle bacillus into the mucosa of Once having penetrated the lymph tissue, it wanders with the lymph stream until it gets into larger lymph vessels, and finally into the thoracic duct. On its way thither the bacillus is quite frequently hindered or detained by stoppage of the vasa efferentia, etc., and the latent tuberculosis, so commonly met with, originates. On the other hand, the thoracic duct becomes the main source for the further spreading of the germs. From here the infection of the apices of the lungs, the loci minoris resistentia, proceeds.* It is evident that the organism frequently react against the invasion of the tubercle bacillus, and the glandular swellings, so common in children, appear. These lymphomata, often nothing else than a consequence of an existing retropharyngeal affection, bear the same relation to this condition as the inguinal bubo does to the primary affection in the genitals.

After bringing many more proofs in confirmation of my theory I came to the conclusion that the hypertrophy of the lymphoid tissue at the vault of the pharynx is nothing more than the expression of this reactionary inflammation. At that time I had overlooked an article by Trautmann, of Berlin, in which he says: "Although by careful examination of the hyperplastic tissue at the vault of the pharynx, of the follicles and of the secretion, neither giant cells nor tubercle bacilli were found, I nevertheless consider tuberculosis the cause of the hyperplasia. . . . A communication made to me verbally by Robert Koch also speaks in favor of tuberculosis being the cause of the hypertrophy. After injection of tuberculin he found at first a rise of temperature, then more swelling and hyperplasia of the lymphoid tissue, and after further injections both disappeared and the hyperplasia was cured, but not until treatment extended over several months."

I cannot mention here all the facts that have been elicited in .

^{*}W. Freudenthal: "Kleinere Beitrage zur Ætiologie der Lungen-tuberkulose," Archiv fur Laryngol., Bd. V., 1896, and Annals of Otology, February, 1897.