

this question from a more favorable standpoint, and I soon found that this serum contained a proteid body possessing a well-marked alkaline reaction and a power of destroying anthrax bacilli. Further, when injected into mice, along with fully virulent anthrax spores, it would prevent the development of the disease. On the other hand, defensive proteids of animals susceptible to anthrax can exert no such protective power, and consequently these experiments indicate a difference in the mode of action of defensive proteids from immune and susceptible animals respectively. Further, the amount of defensive proteid present in a rat can be diminished by those causes which are known to be capable of lowering its power of resisting anthrax. For instance, Feser states that rats become susceptible to anthrax when fed on a vegetarian diet. I have obtained similar results with wild rats. The ordinary white rat, however, I have found to be generally refractory to anthrax on any diet, and always the defensive proteid can be obtained from its spleen and blood serum. With the wild rat this is not the case. In one experiment eight wild rats were used; of these, four were fed on bread and meat, the others on plain bread, for about six weeks. Then one rat of each lot was inoculated with anthrax; of these, the one that had been subjected to a bread diet succumbed. The remaining rats were killed, and it was found that while the spleens of the flesh-fed rats contained abundance of the defensive proteid, only traces of this substance could be obtained from the spleens of the rats that had been fed on bread alone. A similar result was obtained in other experiments.

These facts appear to me to prove that the defensive proteid of the rat deserves its name, in that it tends to preserve it from the attack of the anthrax microbe; in other words, that this substance is, at any rate, a part cause of its immunity against anthrax.

Since the publication of my work on defensive proteids, Buchner has abandoned his view that the bacteria-killing power of blood serum is due to a remnant of vitality, and in a paper recently published he admits the importance of defensive proteids, and suggests for them the name "alexine." Certainly, if it were necessary to re-christen "defensive proteids," this name would be very appropriate. It would, however, be convenient to form names for the different classes of defensive proteids, and I do not think it would be premature to do so now. Defensive proteids appear to be ferment-like, albuminous bodies, and it is extremely unlikely that we shall for a considerable time be able to classify them by any other than physiological tests. From this point of view it is possible to divide them into two classes: first, those occurring naturally in normal animals, and, secondly, those occurring in animals that have

artificially been made immune. For these two classes I propose the names of "sozins" and "phylaxins." A "sozin" is a defensive proteid that occurs naturally in a normal animal. They have been found in all animals yet examined, and appear to act on numerous kinds of microbes or on their products. A "phylaxin" is a defensive proteid which is only found in an animal that has been artificially made immune against a disease, and which, so far as is yet known, only acts on one kind of microbe or on its products. Each of these classes of defensive proteids can obviously be further sub-divided into those that act on the microbe itself and those that act on the poisons it generates. The sub-classes I propose to denote by adding the prefixes "myco-" and "toxo-" to the class name. Thus, myco-sozins are defensive proteids occurring in the normal animal, which have the power of acting on various species of microbes. Toxo-sozins are defensive proteids also occurring in the normal animal, having the power of destroying the poisons produced by various microbes. Myco-phylaxins and toxo-phylaxins similarly will denote the two sub-classes of the phylaxin group. —E. H. Hanken, B.A., in *Lancet*.

ERRORS IN DIAGNOSIS.

I ask permission to say that I stand here now as a conscript rather than as a volunteer. I have no unique or striking cases that I desire to communicate, and I am not conscious of possessing any new development or theory of disease struggling to find utterance. When, therefore, I consented to read a paper to-day, I thought that, next to endeavoring to advance the general stock of knowledge, the best course to adopt might be to try to add to the security of that which we have by pointing our errors into which I and others have fallen. Having yielded in this matter, chiefly to my friend Mr. James Worthington's pressure, and having mentioned the subject I should probably handle, he replied to me in this pleasant way: "By the way, do you remember a particular case in which you made a great mistake?" This made me pause, and I reflected that, if many of my friends here to-day should, as they probably can, round on me in this prompt fashion, I should have a bad quarter of an hour, and should repent of my rashness. However, I resolved to persist in my intention. I am willing to offer myself as an oblation to the advancement of science, and I even challenge Mr. Worthington, when I have done, to describe this case in which I was so mistaken, because it is clinically interesting, as it led to an error in treatment which he had the sharpness to detect and the skill to rectify.

Confession of error, however, need not imply blame. Mistakes in diagnosis may be due to various causes—to inherent obscurity of signs and