verted into a swollen infiltrated tissue undergoing necrosis; in this tissue the typical diphtheria bacilli could be seen in large and small nests and groups in the superficial layers, and extending from them into the deeper portions of the necrotic membrane. Also in cat 4 cover-glass specimens of the tracheal and bronchial exudation showed the presence of the typical diphtheria bacilli. Now, the above epidemic started with the disease of the two cats, 1 and 2, about the end of March, and the question arises: how did the disease originate in these two animals? No cats had been ill in this shed, and the two cats were normal when some weeks previously they were received at the Brown Institution. But during the latter half of March I had two milch cows in the stables of the Brown Institution ill with diphtheria induced by inoculation with the bacillus from human diphtheria, in fact the two cows described on a former page. They were inoculated on March 17, and, as was mentioned on a former page, showed the peculiar eruption on the udder between the 5th and 11th day; in one animal on the 5th day, *i.e.*, March 21, the diphtheria bacillus was demonstrated in the milk drawn from the udder. As soon as the eruption on the udder and the pulmonary affection in the cows were noticed strict orders were given to the attendant that the milk of both cows was to be thrown away. This order was not obeyed, since part of the milk was given to the two cats above mentioned, and these two animals became affected during the last week of March. I ought to mention, however, that, though the time at which these cats became affected is in perfect harmony with the suggestion that the consumption of the above milk of the affected cow had been the cause of their illness, the man who attended to the cows was also attending to the cats. But in view of the fact that this person was free from diphtheria, the possibility of having conveyed the disease from the cow to the cat is out of the question, particularly if we remember that milk containing the diphtheria bacilli had been actually given to the cats.

II. "The Chemical Products of the Growth of *Bacillus anthracis* and their Physiological Action." By Sidney Martin, M.D., Pathologist to the Middlesex Hospital. Communicated by Dr. Klein, F.R.S. Received May 7, 1890.

The work here recorded was done for the Medical Officer of the Local Government Board, whose permission I have for publishing this abstract of it.

The research was commenced in May, 1889. The bacilli were grown in a solution of pure alkali-albumin (made from serum-

proteids) and of mineral salts of the composition of the salts of the serum.

The cultivation of the bacilli was continued for 10—15 days, and the organisms removed by filtering through Chamberland's filter. The filtrate contained the products of the bacterial growth, viz.:—

1. *Proto-albumose* and *deutero-albumose*, and a *trace of peptone:* all with the same chemical reactions as the similar bodies formed in peptic digestion.
2. *An alkaloid.*
3. Small quantities of *leucin* and *tyrosin.*

The chief characteristic of the anthrax proto- and deutero-albumose is their strong alkalinity in solution—an alkalinity not removed by absolute alcohol, by benzene, chloroform, or ether, nor by prolonged dialysis. Acid-alcohol dissolves from the alkaline albumoses a trace of a poisonous body, but this is not in proportion to the toxicity of the albumoses. The albumoses are precipitated in an alkaline condition by saturation with NaCl (proto-albumose) or $(NH_4)_2SO_4$. The alkaloid is soluble in absolute alcohol, amyl alcohol, and in water; insoluble in benzene, chloroform, and ether. It is strongly alkaline in solution, and a powerful base, readily forming salts with acids. The sulphate crystallises in small needles or prisms; the oxalate in long, branching needles or flat plates. From the salts the alkaloid is easily regained. In solution, the alkaloid is precipitated by phospho-tungstic, phospho-molybdic, and phospho-antimonic acids and platinic chloride, but not by potassio-mercuric iodide.* It is slightly volatile, and, when kept exposed to the air, it becomes acid, and loses, to a great extent, its poisonous properties.

Physiological Action.

1. The mixture of anthrax proto- and deutero-albumose is poisonous. In small doses it produces in mice a local subcutaneous œdema, with some sluggishness, ending in recovery. Larger doses produce a greater œdema with more signs of illness, sluggishness leading to prolonged stupor, coma, and death in twenty-four hours or longer. A fatal dose for a mouse of 22 grams weight is 0·3 gram (subcutaneously injected). In some cases the spleen is enlarged: no organisms being present, as shown by gelatine tube cultivations. Boiling for a short time diminishes the toxicity of the proteid, but does not completely destroy it, and death may result from the boiled albumoses.

2. The *anthrax alkaloid* produces symptoms and lesions similar to

* With Millon's reagent, a precipitate is formed which becomes red on heating. This is the same reaction as that given by most proteids, and shows that the base is probably an amido-compound.—May 17, 1890.

the albumoses, but much more rapidly and severely. The animal becomes ill directly after the injection, gradually becomes more and more sluggish, and dies in coma, or, if a non-lethal dose be given, it recovers from the state of stupor gradually. After death enormous local subcutaneous œdema is found, with congestion and sometimes thrombosis of the small veins. Peritoneal effusion is occasionally present, and the spleen is usually enlarged, dark, and congested, or simply congested without being greatly enlarged. The fatal dose for a mouse weighing 22 grams is between 0·1 and 0·15 gram, death occurring in two to three hours.

The anthrax bacillus in digesting the alkali-albumin forms (1) proto-albumose, (2) deutero-albumose, (3) an alkaloid. The alkalinity of the albumoses may explain their toxic properties, being due to the fact that the alkaloid is in a "nascent" condition in the albumose molecule. The bacillus forms the alkaloid from the albumose, and it is possible that the living tissues have a similar action when the albumose is introduced into a living animal.

III. "On the Development of the Atrial Chamber of *Amphioxus*." By Arthur Willey, Student of University College, London. Communicated by Professor Ray Lankester, F.R.S. Received May 5, 1890.

Preface.

Last year, through the kindness of Professor Lankester, I had the opportunity of spending several months—May to August—in Sicily, collecting the embryos and larvæ of *Amphioxus*.

Since then I have been working continuously on the material I obtained in the laboratory of University College, under the direction of Professor Lankester. The period of the development, to which Professor Lankester determined first of all to give attention, was that before which Hatschek's well-known work stops short. He proposed that I should cut sections, so as to ascertain the mode in which the atrial chamber takes its origin and the subsequent history of the gill-slits, viz., as to how the slits on the left side of the pharynx originate. The relation of the larval to the adult mouth and the details of the curious process of movement of the mouth from a unilateral to a median position were included in the scope of our enquiries.

Professor Lankester received a grant from the Government Grant Committee in aid of the present investigation, and it is therefore necessary to state that he has constantly supervised my work, and allows