respect similar to that in the first rabbit, but nothing would happen to the guinea-pig. (2) Alteration of virulence of streptococcus erysipelatis under varied conditions: If a culture of streptococcus erysipelatis (too attenuated to produce the disease in a normal rabbit) were injected into one into which a simultaneous intraperitoneal injection of a sterilised culture of bacillus prodigiosus had been made, typical erysipelas would appear at the point of inoculation.

In this case a rabbit which was immune to an erysipelas of a low virulence was made susceptible, and inoculation from it to a normal one would produce the typical disease in the second; and, by continuing the process through successive rabbits, a very virulent form of erysipelas could be produced in which a highly infective disease occurred with generalised symptoms, though no reaction occurred at the point of inoculation. Here undoubtedly the local cellular infiltration seemed to have played some part in the localisation of the disease. If a rabbit vaccinated against the bacillus pyocyaneus were inoculated with streptococcus erysipelatis, a general fatal infection occurred without local reaction at the seat of inoculation, but blood from this rabbit could only produce the local disease in a normal rabbit. Association of attenuated erysipelas with a common mould increased the virulence of the streptococcus both generally and locally.

ABSTRACT OF A LECTURE ON ERGOT.

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AFTER giving an account of the botanical character and life-history of the fungus which causes ergot, the lecturer went on to say that beside rye, many other plants were subject to ergotisation. The ergot of wheat is by no means uncommon, and has been found to possess similar properties. It has been recommended for use in medicine by Carbonneaux le Perdriel as a substitute for rye ergot, on the ground that it contains less oil and more extractives. He gives cases in which wheat ergot acted well by increasing labour pains and by arresting hæmorrhage. The ergot of diss (Ampelesmus tenax) a reed growing in the South of Europe and Algeria was found by Lallemand to act well in the same kind of cases, one gramme given in two doses with an interval of twenty minutes producing active pains. The ergot of Bromus secalinus caused an epidemic of gangrenous ergotism in Oberhesse in 1855 and 1856. That of Elymus virginicus caused an epidemic of the same disease amongst cattle in the United States in 1884. The ergots of Molinea cœrulea, Lolium perenne and the common reed were found by Diez[1] to possess the same active properties, and to prove fatal to birds (ravens and pigeons) in doses of from ½ to 1½ drachm. These facts are of importance as showing that ergot, upon whatever plant it occurs is poisonous and therefore dangerous to animals when consumed by them. Epidemic ergotism evinces itself under two forms either by producing gangrene or nervous symptoms, spasms of the muscles, cramps, and convulsions. Both forms are preceded by vomiting, diarrhœa, straining, headache, giddiness, and a peculiarly hard and slow pulse, formication, especially of the extremities, soon is experienced. Amongst the most common *post-mortem* signs are numerous small subcutaneous extravasations; similar hæmorrhages occur in the lungs, stomach, and under the peritoneum. Amongst the less common effects of ergotism are œdema of the skin, cataract, gangrene of the lung. Changes in spinal cord have been described by Tuczek,[2] consisting of a hyperplasia and fibrillar metamorphosis of the neuroglia with degeneration of the nerve tubules in the vicinity of Burdock's and Goll's columns most marked in the dorsal region, but extending from the lumbar up to the medulla—a posterior sclerosis in fact. With regard to the effect of ergot on animals, dogs have been made the subject of experiment by Shrine in the middle of the eighteenth century, who found they became convulsed and died, and by Orfila in 1823, who succeeded in producing gangrene of the feet, ears, and tail, so that dogs, like men, suffer from both forms of ergotism. Pigs are specially liable to gangrene of the ears, as was first noticed by Salerne (1748). Randall (1842), in North America, saw gangrene of feet with casting of the hoof in cows; and Colles observed the same thing in Ireland with horses in 1847. Birds are very susceptible to gangrene of the comb and wattles, as well as of the hard and soft palate, tongue, and epiglottis, as well as of the balls of the feet. Professor Kobert, of Dorpat, who has studied the subject of ergot in all its aspects, considers there is evidence that the Athenian plague, B.C. 430, was probably due to ergotism. Some remarks of Hippocrates (III, 4) where he speaks "of great falling of the flesh, tendons, and bones," and of the "forearm and arm dropping off," seem to give support to this view.

Thucydides, II, 49, gives, amongst the symptoms of this plague, "thirst, vomiting, retching, accompanied by spasms, redness, lividity, and sores on the skin, intense diarrhœa," and those that "survived its most fatal consequences lost their fingers and toes, whilst some lost their eyes," which is even stronger evidence in support of this view. In the Middle Ages ergotism is frequently mentioned under the names of "ignis sacer" and "ignis S. Antonii." Flodart von Reims records such a one in 945, which was attributed to eating bad bread, as does Sigebert de Gremblour (1089), in which year the bread was "dark red." Robert Dumont, in 1125, likewise refers to one caused by eating bad bread of a violet colour. The symptoms were discoloration of the skin on the limbs, chest, and abdomen, at first pale-bluish, then mulberry coloured, then quite black, after which great pieces necrosed, and the flesh fell from the bones, so that persons lost their arms and legs, and instances were encountered in which by the loss of all four limbs the unfortunate sufferers were reduced to mere trunks. In many cases the fingers and toes separated painlessly and were found in the gloves or stockings. The disease was very common in the eighteenth century—so much so that a commission was appointed by the Medical Society of Paris to inquire into it. The Commission included Jussieu, Paulet, and Tessier. As an instance of the poisonous character of ergot, the following case given by Vérillart[3] may be quoted: A poor man asked permission from a farmer to have some ergotised grain which had been rejected by the latter on this account. He was warned of its poisonous nature, but being hungry himself and having a starving family at home, took it home, ground it, and mixed it with meal, and used it for the family's consumption. In one month this man, his wife, and his two eldest children were dead, and the third child—an infant at the breast—who had been fed with a small quantity of sop made from this meal, was the only survivor; but this child not only became deaf and dumb, but lost both its legs.

According to Hirsch, in his *Handbook of Geographical and Historical Pathology*,[4] 130 epidemics have occurred in Europe between 591 and 1879. The curious fact comes out that the convulsive and gangrenous forms have different geographical distributions, the former being more frequent in Germany and Russia, the latter in France and Spain. The ecbolic action of ergot has, according to Dorvault,[5] been known to the Chinese from remote antiquity. Diez says that its properties were well known to the midwives of Poland, Germany, and Holland—so much so, indeed, that in 1778 its use by them was forbidden by law in Hanover.

Kobert[6] finds the active constituents of ergot to be three: ergotinic and sphacelinic acids and cornutine. The first-named is the principal constituent of Dragendorff's sclerotinic acid, and is also contained in Bonjean's ergotine, and therefore in our officinal liquid extract. When injected subcutaneously, it reduces the blood pressure, and gives rise to certain nervous derangements such as inco-ordinate movements, loss of the reflexes, paralysis, and causes death from failure of the respiratory process. It is without action on the uterus; when taken by the mouth, it becomes split up into inert bye-products, so as to be without action on the animal organism.

Sphacelinic acid, on the other hand, is a very energetic poison; it is the gangrene-producing constituent of ergot,

[1] Diez, W., *Versüche über die Wirkungen des Mutterkorns*, 1831, p. 142.
[2] Franz Tuczek, *Arch. für Psych. und Nervenkrank.*, xi, p 158 and p 366, 1881 and xii, p. 99, 1882.
[3] *Mémoire sur une Espèce de Poison connu sur le nom d'Ergot.* Tours. 1770.
[4] Vol. ii, translated by the New Sydenham Society. 1885.
[5] Dorvault, *Officine*, 5e edit., Paris, 1858, p. 558.
[6] For a full account of the literature of ergot, see the article on *Mutterkorn* by Kobert in von Geissler and Moeller's *Realencyclopädie der Pharmacie.*