(a) the injection of a tenfold fatal dose in the leg muscles of guineapigs was followed by the finding of the toxine in the blood and in the sciatic nerve of the same side; occasionally, if they waited for the development of general tetanus, in the other sciatic also. The muscle and fat from the neighbourhood of the injection contained no toxine. (b) *The sucking up of the toxine by the nerve is bound up with the integrity of the axis cylinders.* Thus, if in animals the sciatic was cut high up, it was found that after six days, when degeneration of the axis cylinders was well developed, the nerve would take up no toxine. Therefore the nerve sheath, or the lymphatics of the nerve sheath, do not materially share in the absorption of the toxine. (c) Again, if the main nerve be cut before the injection of toxine into the muscles of the foot, one can find the toxine later only in the distal portion, not in the proximal; therefore *the toxine gets into the nerve, not by the neural capillaries, but by the bare axis-cylinder endings in the muscle.*. Even when all injury of nerves is avoided, as in injection into the *corpus vitreum* of the eye, the toxine can be found later in the sciatics and the brachials. The more concentrated the poison is at any spot the more of it is taken up by nearby nerve-endings. (d) *The poison travels only centripetally.* Thus a nerve already containing toxine from a previous injection is cut and left *in situ*. It is found that the toxine soon disappears from the proximal end, which is protected by the division against the further inwandering of the toxine from the periphery; or, if the lumbar cord be injected, one finds toxine in the lumbar and dorsal cords, but not in the peripheral nerves. These were the experiments of Marie and Morax; the work of Meyer and Ransom not only confirmed these results, but brought our knowledge of the subject considerably further ahead, and gave it a strong practical application.

II. *The spinal centres can be protected from the toxine by blocking the afferent nerves with antitoxine.* (a) In local poisoning. Thus, a rabbit received the toxine in the right and left legs over the tibia subcutaneously; at the same time antitoxine was injected into the sciatic of the right leg. The latter remained perfectly free of tetanic stiffness, while the left became tetanic. On the third day the fore legs also became stiff and death occurred on the fifth day. It might be thought that the toxine had escaped outside the nerve, and being absorbed into the general circulation had caused death in that way. This difficulty was overcome in later experiments by sealing the injected nerve at the point of injection with collodion and embedding it in paraffin. The development of tetanus was still prevented, although in ten days the antitoxine disappeared from the nerve, after which the toxine, still being produced, was able to travel up and ultimately cause a light grade of tetanus. (b)