10231 P

Asphyxia of the Spinal Cord of the Cat.

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The effect of the onset of asphyxia of the spinal cord has been investigated frequently. Usually a period of increased reflex excitability during the development of asphyxia has been described. In a series of experiments we studied the recovery of reflex activity after various periods of asphyxia. The cord was asphyxiated by bringing it under a pressure higher than that of the blood, thus excluding the blood supply. The spinal cord was ligated in the lower thoracic region 5 days previous to the planned experiment, without opening the dura. A cannula was brought in the subdural cavity of the lower part of the cord, which was separated from the rest of the central nervous system by the ligature. Deoxygenated physiological solution was then forced into the cavity under a pressure of 23-25 cm of mercury. The cord was kept under pressure for periods varying between 25 and 75 minutes. The animals were observed up to 3 weeks afterwards.

After 75 minutes of asphyxia no reflex activity returned; after 25 minutes of asphyxia all the reflexes of the hind limb and the tail returned within 2 hours, and these reflexes resembled those in the normal animal except that their excitability was increased. As the time of asphyxia was lengthened in 10 minute increments more and more reflexes did not return. First the skin reflexes were lost, then the tendon reflexes, and finally tone. The excitability of the tendon reflexes when their return was observed was usually much higher than normal. The tone in the extensor muscles of the legs was greatly enhanced in many cases, even in some instances to such a degree that it resembled rigor mortis. This tone could continue for days or even weeks; it was proven to be reflex in origin because transection of the dorsal roots of the lumbo-sacral cord caused its disappearance. After the longer periods of asphyxia (55 and 65 minutes) the return of tone and tendon reflexes was only temporary, disappearing again after 48 hours.

Histological preparations, made 3 weeks later, of the cord of animals subjected to pressure for the longer times revealed the presence of only about 5% of the normal number of perikarya. Therefore it seems probable that the temporary reappearance of re-

flex activity must be ascribed to the temporary recovery of conduction in cells which are damaged so severely that they will die shortly.

Since the high tone could continue for weeks following asphyxia, it is concluded that it is a release phenomenon. By this it is meant that a system normally inhibiting the tone is damaged to a greater extent than the excitatory component of the tone reflex. Normal tone would be an equilibrium between an excitatory and an inhibitory component of this reflex. The increased excitability of the tendon reflexes is to be explained in a similar manner.

Since the inhibitory systems seem to be less resistant against asphyxia and would therefore be abolished first during the development of asphyxia, the increased reflex excitability which has been described by various authors must be considered a release phenomenon also.

10232

Effects of Nicotinic Acid on Specific Antibody Production.

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In a previous paper[1] it was shown that ascorbic acid mixed with or injected simultaneously with horse serum hastens and augments specific-precipitin production in rabbits. We have extended this work to include other "activators" of tissue-enzymes, the present paper summarizing the "co-antigenic" effects of sodium salts of nicotinic acid.

In our initial tests with this activator six 2000 g rabbits were injected intravenously with 0.5 cc horse serum. Three of these animals were then given intravenously a fresh mixture of 50 mg nicotinic acid plus 22 mg Na_2CO_3 in 1 cc NaCl solution. The rabbits were bled at frequent intervals, and the average precipitin-titer (ring test) was determined for each group. Data thus obtained are recorded in Table I.

The results in this preliminary series are similar to those pre-

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1 Madison, R. R., and Manwaring, W. H., PROC. SOC. EXP. BIOL. AND MED., 1937, **37**, 402.