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.

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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.

TABLE I.
Co-antigenic Action of Nicotinic Acid.

Avg titer per group	7th day	11th day	14th day	18th day	26th day
Nicotinic-acid group	1:1850	1:5500	1:7700	1:8300	1:10000
Control group	1:700	1:2100	1:1800	1:2300	1:2700

viously obtained with Vitamin C, except that the percentile increase in antibody-production is less than with ascorbic acid.

In order to determine the optimal antibody-stimulating dose of nicotinic acid, 21 rabbits were divided into 7 groups of 3 each. Each animal was given a single intravenous injection of 0.5 cc horse serum, mixed with varying amounts of freshly neutralized nicotinic acid. The average titers for 4 of the 7 groups are recorded in Fig. 1.

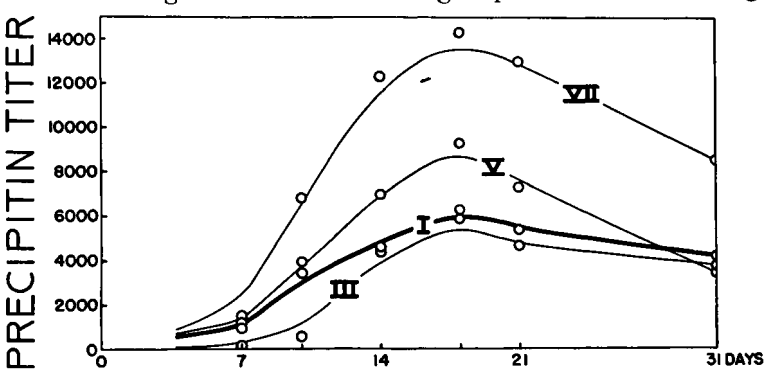

FIG. 1.

Effects of Varying Amounts of Nicotinic Acid on Antibody-production.
Group I—Control group, no nicotinic acid.
Group III—25 mg nicotinic acid.
Group V—100 mg nicotinic acid.
Group VII—800 mg nicotinic acid.
Intermediate groups II (12.5 mg), IV (50 mg), and VI (300 mg) gave data intermediate between those recorded in the figure.

Fig. 1 shows that under the conditions of the test, relatively small doses of sodium nicotinate inhibit specific-precipitin production. Medium doses may be without demonstrable effect, while massive doses augment antibody-production. Even with massive doses, however, antibody stimulation is less pronounced than that previously reported with ascorbic acid.

No theory is suggested to account for this dual effect. The phenomenon, however, is reminiscent of dual effects reported by Walbum, *et al.*, in their studies of the antibody-stimulating or inhibiting effects of metallic salts.

Since such massive doses of nicotinic acid are not clinically feasible, study of nicotinic acid as a possible adjuvant in vaccine-therapy was discontinued at this point.