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Abstract : The excretion of ascorbic acid by albino rats maintained as previously described (Abst. 3015, Vol. 9) was greatly accelerated by a series of compounds having in common the characteristic of functioning *in vivo* as depressants of the nervous system. Of the barbituric acid derivatives, the most active were sodium phenobarbital and calcium ipral, 20 mg. of each daily raising the average daily

acid excretion from 0.2 to 10 mg. within a few days. Other hypnotics had a paraldehyde and chloretone in doses of 20 mg. daily having the greatest increase in the ascorbic acid excretion, which amounted to 11 and 18 mg. daily, respectively. Among the antipyretics the pyrazolone derivatives, amino-pyrene and antipyrine were the most effective, causing in doses of 20 mg. daily, a daily excretion of 7 mg. a day within 5 to 6 days. Phenols, salicylates, sulphanilamide and sulphapyridine were slightly active; a dose of 20 mg. narcotine or nicotinic acid increased the excretion of ascorbic acid to 4 and 5 mg. daily, respectively. A number of alkaloids cause increased excretion.

Biological tests with guineapigs for the presence of vitamin C [ascorbic acid] in rats receiving narcotine and sodium pheno barbital confirmed the positive results obtained by titration with 2: 6-dichlorophenol indophenol. The excretion of ascorbic acid in rats after daily administration of 20 mg. of sodium phenobarbital or chloretone was observed for two months, during which period the animals excreted 20 to 30 mg. of ascorbic acid daily. It is assumed that synthesis of endogenous origin takes place in the liver. The percentage recovered from rats' urine of ascorbic acid orally administered was found to be rapidly as the amounts administered increased. If, however, the ascorbic acid was administered intraperitoneally, the excretion rate was greater, amounting to 60 per cent. It was found that the ascorbic acid was conjugated with any of the toxic substances administered but its endogenous production appeared to be related to the detoxication of the animals. E. Kodicek.

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