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Effect of Vitamin K on Hypoprothrombinemia of Experimental Liver Injury.*

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It has been shown that the plasma prothrombin level declines when the liver is injured[1, 2, 3] or when it is excised, either in part[4] or *in toto*.[5, 6] Hypoprothrombinemia develops also with vitamin K deficiency. In man, the two factors are often combined and the response to vitamin K is commonly incomplete. In such patients, the extent to which vitamin K compensates for the liver injury, if at all, is difficult to determine. To study this question further, we have performed experiments to determine the influence of vitamin K in excess on the hypoprothrombinemia which develops following liver injury alone.

Liver injury was produced in dogs (10-15 kg) by repeated administration of small doses of chloroform, as described previously.[2] In each experiment, 2 dogs of the same weight were given identical diets (mixed table scraps) and identical doses of chloroform. In addition, one of the animals of each pair received a daily vitamin K supplement consisting of the petroleum ether extract of 200 g alfalfa meal, emulsified in 30 cc of 2% solution of Wilson's bile salt. Plasma prothrombin determinations were made by the method of Warner, Brinkhous and Smith.[1,2]

Almost identical results were obtained in each of the 4 experiments performed. Chart 1 shows a typical experiment. It is seen that the administration of vitamin K failed to modify in any way, either the fall in prothrombin with chloroform administration or the rise in prothrombin during the recovery period.

In another experiment the daily vitamin supplement was started

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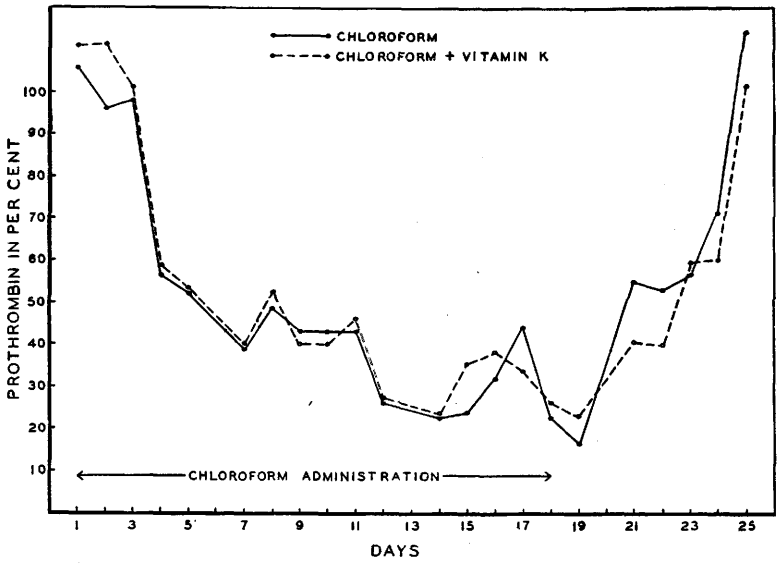

CHART 1.

The consecutive daily doses of chloroform given during the 18-day period of chloroform administration were 2, 2, 3, 2, 3, 2, 3, 3½, 3½, 4½, 5, 5, 4, 5, 6, 6, 7 and 6 cc.

one week prior to the beginning of chloroform administration. This procedure likewise had no demonstrable effect on the fall in plasma prothrombin.

It is suggested that when vitamin K deficiency and liver injury are both present, as in many patients, administration of the vitamin may correct the former, but one cannot expect that an excess of the vitamin will compensate for the element of liver injury.

Summary. The hypoprothrombinemia, which develops following liver injury (chronic chloroform intoxication), is not influenced by vitamin K administration.