These results show that the vitamin C probably acts on the red cells and the action is not irreversible, although it is not an easily reversible one. In order to see if this supposed protective action might be aspecific, I examined hæmolysis due to saponin.

A first set of five tubes were each filled with saline, saponin and sheep cells (red) in the following manner :

Tube	1	2	3	4	5
Saline	0·2 c.c.	0·3 c.c.	0·4 c.c.	0·5 c.c.	0·6 c.c.
Saponin (0·05%)	0·5	0·4	0·3	0·2	0·1
Sheep red cells (3%)	1	1	1	1	1

These tubes were used as controls.

A second set of five tubes were each filled with vitamin C and sheep red cells and were allowed to remain in contact, at room temperature, for 15 min. Then saponin was added. The quantities are listed below.

Tube	1	2	3	4	5
Vitamin C	0·2 c.c.	0·2 c.c.	0·2 c.c.	0·2 c.c.	0·2 c.c.
Sheep red cells (3%)	1	1	1	1	1
Saline	0	0·1	0·2	0·3	0·4
Saponin (0·05%)	0·5	0·4	0·3	0·2	0·1

All of the tubes of both series were left at room temperature, and after two hours the following observations were made.

	Tube 1	Tube 2	Tube 3	Tube 4	Tube 5
Hæmolysis in the tubes without ascorbic acid	+++	++	++	+	∓
Hæmolysis in the tubes with vitamin C	±	−	−	−	−

The same experiments were repeated three times with the same results.

These results show also in this case a clear anti-hæmolytic action of the vitamin C.

Thus the anti-hæmolytic effect *in vitro* of vitamin C on hæmolysis due to the complement and the saponin has been shown. How this anti-hæmolytic action takes place, it is now difficult to say ; it is my personal feeling that the strong reducing power of the vitamin C might account for it.

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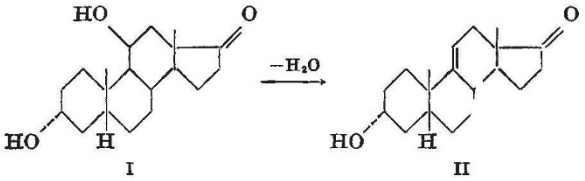
Isolation of 11-Hydroxy-etiocholanol-3(α)-one-17 from the Urine of Male Patients with Adrenal Cancer

A SUBSTANCE that proved to be Δ9-etiocholenol-3(α)-one-17 (II) was isolated by Lieberman *et al.*[1] from the urine of certain patients with cancer of the breast or prostate, lymphatic leukæmia, essential hypertension or Cushing's syndrome. They suggested that Δ9-etiocholenol-3(α)-one-17 was a derivative of 11-hydroxy-etiocholanol-3(α)-one-17 (I) and was formed from the latter compound by dehydration during the acid hydrolysis of the urine. Later, they were able to identify (I) in urinary extracts by infra-red spectroscopy[2].

We have now been able to isolate 11-hydroxy-etiocholanol-3(α)-one-17 from the urine of two male patients with adrenal cancer.



From the first patient, aged twenty-two years, who developed a typical Cushing's syndrome, about twenty litres of urine were collected. The urine was hydrolysed and extracted by the method of Dingemanse and Laqueur[3]. The extract was fractionated on chromatographic columns by the method of Dingemanse *et al.*[4].

A compound giving Zimmermann's reaction for 17-ketosteroids was eluted with benzene containing 0·5 per cent of ethanol. After repeated crystallization from aqueous ethanol, a substance with m.p. 237–238° C. was obtained. Further recrystallization did not change this melting point. (The melting point of 11-hydroxy-etiocholanol-3(α)-one-17, according to Sarett[5], is 238·5–240·5° C.)

As only a few milligrams of the substance could be isolated in a pure state, no analysis could be made. An acetate with m.p. 215–222° C. was prepared with acetic anhydride in pyridine solution. The amount of substance isolated was too small to allow further recrystallization. (The melting point of 11-hydroxy-etiocholanol-3(α)-one-17-acetate, according to Sarett[5], is 237–238° C.)

Eight litres of urine were then collected from a second patient, a male aged eighteen years, who was suffering from an adrenal cancer and developed an atypical Cushing's syndrome. About 8 mgm. of a 17-ketosteroid with m.p. 237–238° C. was obtained. An acetate with m.p. 238–238·5° C. was prepared ($C_{21}H_{32}O_4$ requires C, 72·35 ; H, 9·27 ; found, C, 71·78 ; H, 9·08).

According to these data the substance isolated from the urinary extracts was 11-hydroxy-etiocholanol-3(α)-one-17.

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Fate of N-Methylnicotinamide in Man

FOLLOWING the discovery that N-methylnicotinamide is a metabolite of nicotinic acid[1], the fate of this compound in the animal body has been studied by several workers. Perlzweig and Huff[2] found that only 10–20 per cent of a dose of N-methylnicotinamide administered orally to human beings could be recovered in the urine, and concluded that N-methylnicotinamide is not the final metabolite of nicotinic acid, but is further metabolized into unidentified products. Ellinger[3] studied the fæcal excretion of N-methylnicotinamide by the rat and discovered that