from this cause has not been serious in our own urine extracts. But if greater specificity in analysis is needed then recourse must still be had to the biological methods of assay.

It seems likely that similar considerations will apply to the comparable method for the estimation of compound F in blood published by Nelson and Samuels (1952), whilst our own work was in progress, and that their method will be similarly capable of giving a reasonable approximation to the true compound F content of the blood extracts used.

Summary

The yield of cortisone-like hormone recoverable from human urine has been estimated after enzymatic hydrolysis with β -glucuronidase and chloroform extraction. It has been compared with the amount recovered after the more usual acid hydrolysis at room temperature.

The enzyme treatment increases the yield of cortisone-like hormone by from 8 to 315%, the mean increase being 99%.

The Porter-Silber colour reaction can detect quantitatively cortisone added to urine extracts, but often gives quite erroneous values for the cortisone-like hormone content of the extracts themselves.

Much of the interfering chromogen can be removed by paper chromatography.

On extracts purified by paper chromatography the Porter-Silber reaction can give estimates of cortisone-like hormone content which agree with the results of bio-assay of the same urine extracts.

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In a paper which he read to the New York Health Conference last year Dr. I. Galdston discussed some of the public health problems in that city. He mentioned that there was a corp of police in the New York subway to hand out summonses for carrying lighted cigarettes or cigars, though the staff was insufficient to provide adequate or decent toilet facilities. For that matter, he added, there were inadequate public toilet facilities through the whole city of New York, despite the ageing population. Again, in a city which stretches for more than 10 miles, there were "few facilities whereby the over-taxed heart, the aching bones, or the fatigued muscles might be given respite on a public bench or park seat."

17-KETOSTEROID EXCRETION IN GOUT

BY

W. R. BUTT, B.Sc., A.R.I.C.

AND

F. GRAHAM W. MARSON, M.D., M.R.C.P.

(From the Department of Clinical Endocrinology, United Birmingham Hospitals, and the Department of Therapeutics, University of Birmingham)

Wolfson *et al.* (1949) reported that urinary 17-ketosteroid (17-K.S.) excretion was low in gout. In a group of 11 gouty patients, representing all stages of the disease, the average excretion was 3 mg. per 24 hours, with a range of 0.67–6.6 mg. per 24 hours. They stated that biological evidence of androgen activity was normal in 9 of the 10 men in this group, and explained this seemingly anomalous finding by postulating the presence of an abnormal androgen in gout which, when metabolized, does not materially contribute to the urinary 17-K.S.

Later, Robinson *et al.* (1949) reported findings in which 26 determinations of 17-K.S. in 10 gouty patients gave values consistently lower than normal, both during attacks and in interval periods.

Material and Methods

Our findings were obtained from a study of 33 gouty patients (see Table).

Basic Data on 33 Gouty Patients, Showing Serum Urate and 17-ketosteroid Levels

Case	Sex	Age (Years)	Average Serum Urate*	17-K.S. (mg./24 hours)	Acute Gout Witnessed	Satisfactory Colchicine Response
Recurring Acute Gouty Arthritis						
1	M	43	9.8 (5)	4.4		
2	M	53	6.6 (23)	7.4	+	+
3	F	68	6.7 (15)	5.4	+	+
4	M	55	5.6 (8)	10.9		
5	M	37	9.2 (21)	9.5	+	+
6	M	42	8.3 (18)	11.9	+	+
7	M	48	8.2 (6)	9.8	+	+
8	M	49	6.3 (5)	18.0		
9	M	57	5.1 (12)	8.3	+	+
10	M	36	7.8 (5)	16.4		
11	M	52	7.2 (7)	5.0		
12	M	69	7.5 (5)	6.7		
Chronic Gouty Arthritis						
13	M	53	5.0 (22)	7.0	+	+
14	M	65	5.5 (19)	10.8	+	+
15	M	56	4.8 (17)	6.8	+	+
16	M	59	7.2 (8)	14.0	+	+
17	M	60	10.8 (16)	11.0	+	+
18	M	40	8.7 (11)	8.6		
19	M	70	6.8 (9)	10.0		+
20	M	64	9.9 (7)	11.9		
21	M	56	8.4 (13)	13.1	+	+
22	M	59	9.2 (3)	9.3	+	+
23	M	54	9.2 (12)	13.9	+	+
24	F	61	7.4 (11)	6.0	+	+
25	M	45	7.2 (4)	11.2	+	+
26	F	67	5.0 (9)	7.3	+	+
27	M	57	5.8 (9)	11.7		
28	M	62	9.1 (3)	12.3		
29	M	45	8.5 (3)	12.4	+	+
30	M	58	8.6 (3)	5.7	+	+
31	F	68	7.4 (20)	1.4	+	+
32	M	49	8.6 (14)	10.7	+	+
33	M	37	12.7 (13)	5.0	+	+
Average all patients		54.4	7.7	9.5		

* The numbers of serum urate estimations are given in parentheses.

The neutral 17-K.S. values were obtained by a polarographic method (Barnett *et al.*, 1946a; Butt, 1950). Normal ranges for males are 10–20 mg. per 24 hours (age group 20–38 years), and for females 6–18 mg. per 24 hours (age

group 18-37 years) (Barnett *et al.*, 1946b; Butt *et al.*, 1948). Wolfson *et al.* (1949) use a colorimetric method in which the normal ranges are stated to be 9-25 mg. per 24 hours for males and 7-12 mg. per 24 hours for females. These figures agree well with those of Hamburger (1948), who studied the variation of 17-K.S. excretion with age and sex in normal individuals. The urate estimations were performed colorimetrically by Brown's (1945) method.

Results

The excretion of 17-K.S. was measured in 29 male and 4 female patients suffering from gout. The average age of the subjects was 54.4 years, and the range of 17-K.S. was 1.4-18 mg. per 24 hours, with an average of 9.51 mg. per 24 hours.

The Graph shows the normal variation of 17-K.S. output (for males) with age (Hamburger, 1948). Normal outputs by our method all lie within this range: male cases in this series are plotted in the Graph. The outputs in the four female cases are within the corresponding female range.

17-K.S. in gout (29 males). Broken lines enclose area within which 97.1% of normal values lie (males) Hamburger (1948).

Discussion

Polarographic and colorimetric methods for estimation of 17-K.S. have been discussed by Barnett *et al.* (1946b) and Butt *et al.* (1951). Results obtained by polarography are comparable with those obtained by colorimetry if a colour correction is made. In some urine extracts, however, chromogenic substances are present which are not recognized by polarography, and in these cases the colorimetric method gives values considerably in excess of those found polarographically.

Wolfson *et al.* state that the non-ketonic chromogens are apparently disproportionately high in the urine of gouty patients, and that separation of the neutral ketonic material should be done with Girard reagent T. The polarographic method is particularly suitable here, as the estimation is done on the Girard derivative, and no colour correction is needed.

The 17-K.S. outputs obtained in this series are appreciably higher than those of Wolfson *et al.*, and fail to show any significant deviation from the normal range.

Summary

Urinary 17-K.S. outputs have been estimated in 33 gouty patients. The results show no significant deviation from normal values.

We have been unable to confirm that gout is characterized by a low 17-K.S. excretion, a finding first reported by Wolfson *et al.* (1949) and later by Robinson *et al.* (1949). Therefore we see no grounds for postulating the presence of an abnormal androgen in gout.

Our thanks are due to Professor A. P. Thomson, who was in charge of the clinical cases, and to the nursing staff, whose full co-operation is so essential in the collection of 24-hour specimens of urine.

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ACUTE ACETONE POISONING CAUSED BY SETTING FLUID FOR IMMOBILIZING CASTS

BY

L. C. HARRIS, M.D., M.R.C.P.

AND

R. H. JACKSON, M.C., B.M., B.Ch.

(From the Children's Department, Royal Victoria Infirmary, Newcastle-upon-Tyne)

In recent years lightweight casts have been used for immobilization of limbs. These consist of glass and textile bandages which are set by means of a fluid consisting chiefly of acetone. They have the advantage of being easily penetrable by x rays, and it is therefore unnecessary to remove them when x-raying the immobilized limb. Because of their light weight they are often used for children.

It is not generally recognized that the setting fluid is a potential cause of serious poisoning. Indeed, this is not surprising, since we have found no adequate appraisal of the more severe toxic effects of acetone in the literature. Hamilton and Johnstone (1945), for example, write: "Acetone has an innocent history as far as its industrial use is concerned. Kobert said that it was almost inconceivable that poisoning could follow its use." We have had under our care a case of acetone poisoning following the application of a lightweight cast, and we feel that it is important to draw attention to the potential danger of this method of immobilization.

Case History

The patient, a 10-year-old boy, was being treated in a sanatorium for tuberculous arthritis of the right hip. His condition had improved while he had been immobilized on a plaster bed, and the orthopaedic surgeon in charge of the case decided to have a lightweight hip spica applied.

The cast was applied on January 11, 1952, by a male nurse in an extremely poorly ventilated theatre measuring 21 by 24 by 12 feet (6.4 by 7.3 by 3.7 metres). The room was very warm, being heated by means of metal plates in the walls.