TABLE III.
Effect of Metrazol Shock on Experimental Poliomyelitis. Complete paralysis in 5 days.

Monkey No.	Dose of metrazol Iv.	Type of treatment	Dose of virus (5% suspension) Ic.
20	0.3-0.6 cc	before infection	0.1 cc
21	"	after infection	1 "
22	—	—	0.1 "
23	—	—	1 "

group with no perceptible difference. Upon autopsy all monkeys showed severe lesions in the cord.

Conclusions. Neither the administration of narcotic doses of luminal nor the production of systemic shock by means of insulin or metrazol were capable of influencing the course of experimental poliomyelitis. Moreover, the extent and severity of the lesions in the spinal cord showed no significant difference between treated monkeys and untreated control animals. Even though essentially negative, the above results are considered important in demonstrating that propagation of the virus of poliomyelitis in the central nervous system is not affected by profound cytological and metabolic changes in the nerve tissue, as were produced by the methods employed in this work.

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On the Detoxication of Phenylacetic Acid by Glucuronic Acid in Humans.

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Using reduction methods, Quick[1] working with dogs, concluded that phenylacetic acid is excreted in the form of its glucuronide to the extent of 34%; and a considerable portion of the remainder appeared in conjugation with glycine. Working with human subjects, and using similar methods of determination, Ambrose, Power, and Sherwin[2] claimed that by far the largest quantity eliminated is in combination with glutamine, whereas only 5% appears to be conjugated with glucuronic acid.

[1] Quick, A. J., *J. Biol. Chem.*, 1928, **77**, 581.

[2] Ambrose, A. A., Power, F. W., and Sherwin, C. P., *J. Biol. Chem.*, 1933, **101**, 669.

In estimating the quantity of glucuronides in the urine, Quick, Sherwin, etc., used reduction methods[3] and assumed that the increase in reducing substances in the urine, produced as a result of the ingestion of phenylacetic acid, was due solely to an increase in urinary glucuronides.

Realizing the inadequacy of such reduction methods, Sherwin[4] wrote: "Unfortunately, many of the studies in glucuronic acid have been confined to a qualitative or 'quantitative' test for reducing substances in the urine, and attempts to check many of these results have not proved satisfactory." For this reason, and because of the importance of glucuronic acid in processes dealing with detoxication—the ingestion of phenylacetic acid has been suggested as a liver functional test—we were interested in studying the detoxication of phenylacetic acid by glucuronic acid, using a *direct* method for the determination of glucuronides. Fortunately, this was made possible by the work of Maughan, Evelyn, and Browne,[5] who devised a photo-

TABLE I.
Amounts of Glucuronic Acid Eliminated.

Subject	Mg glucuronic acid per 24-hr sample Days								
	1	2	3	4	5*	6	7	8	9
A	496	534	341	437	965	539	414	440	423
B	604	568	627	643	1115	382	476	651	465
C	418	401	530	496	992	617	520	425	421
D	502	551	615	537	1078	369	333	378	448
E	289	471	398	377	951	405	442	486	535
F	528	545	499	480	1154	477	549	549	583
G	470	503	565	658	1067	664	369	516	423
H	623	554	708	717	996	588	574	602	

*Ingestion of 5 g of phenylacetic acid at the beginning of the 5th day.

TABLE II.
Increase in Glucuronic Acid in the Urine as a Result of Feeding Phenylacetic Acid.

Subject	Avg mg glucuronic acid per 24 hr in normal urines	Increase in mg glucuronic acid on ingestion of 5 g of phenylacetic acid
A	453	512
B	540	575
C	479	513
D	467	611
E	425	526
F	526	628
G	521	546
H	624	372

[3] Somogyi, M., *J. Biol. Chem.*, 1926, **70**, 599.

[4] Harrow, B., and Sherwin, C. P., *A Text-Book of Biochemistry*, 1935, p. 380.

[5] Maughan, G. B., Evelyn, K. A., and Browne, J. S. L., *J. Biol. Chem.*, 1938, **126**, 567.

electric method for the quantitative estimation of glucuronic acid and conjugated glucuronides in human urine. They made use of the color developed by the reaction of the glucuronide with naphthoresorcinol on being heated with hydrochloric acid.

The experimental procedure was as follows: 24-hour urine samples of normal male subjects were collected for 4 days. On the fifth day, 5 g of phenylacetic acid—which is as much as can be conveniently tolerated—previously neutralized with 0.2 molar NaOH, were ingested. The urine was collected for the next 5 days. The amount of glucuronide was determined in each 24-hour sample according to the method previously referred to.[5] The analyses were carried out in a photoelectric colorimeter of the type described by Withrow, Shrewsbury, and Kraybill.[6] The results with 8 subjects are shown in Tables I and II.

Conclusion. Dealing first with normal (control) urines, the amount of glucuronic acid in 24-hour samples varied from 350-650 mg, with an occasional lower or higher figure. These results agree well with those obtained by Maughan, Evelyn, and Browne[5] and by Roe and Hall.[7] Next, with regard to the effect of ingesting 5 g of phenylacetic acid, we find that this results in an average increase of 535 mg, an amount which corresponds to a detoxication by glucuronic acid of 7.5% of the phenylacetic acid ingested.

11235 P

Metabolic Studies in Dermatomyositis, with a Note on the Effect of Wheat Germ.*

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Patients with dermatomyositis frequently have extensive changes in the skin, muscles and bones. In addition, many patients have symptoms of Raynaud's Disease. The muscular disability in dermatomyositis can be as marked as that occurring in advanced pro-

[6] Withrow, R. B., Shrewsbury, C. L., and Kraybill, H. R., *Ind. Eng. Chem.*, Analyt. Edition, 1936, **8**, 214.

[7] Roe, J. H., and Hall, J. M., *J. Biol. Chem.*, 1939, **128**, 329.

* Aided in part by a grant from the National Foundation for Infantile Paralysis, Inc.