

FAMILY PERIODIC PARALYSIS

REPORT OF A TYPICAL CASE, WITH METABOLISM STUDY *

THEODORE DILLER, M.D., AND JACOB ROSENBLOOM, M.D., PH.D.
PITTSBURGH

This is a curious and rare disease of which we have as yet only a few reports in the literature, most of which have been from this country. The first report appears to have been that of Westphal¹ in 1885. Cousot reports five cases, Goldflam² nineteen and Taylor eleven cases. Taylor,³ in his exhaustive paper published in 1898, collected at that time fifty-three cases. Since then a few other cases have been reported, three by J. K. Mitchell, Flexner and Edsall.⁴ Last year Gardner⁵ reported a single isolated case which he studied in an interesting way. Dr. L. Pierce Clark⁶ last year reported a new subtype of this palsy. The most important and extensive observations, however, have been made by Dr. G. E. Holtzapple,⁷ a general practitioner of York, Pa., and despite the subsequent reports, Holtzapple's paper still constitutes the most important contribution to the subject. His paper is based on observations running over a period of twenty-two years made on one family in four generations. In this family he has observed seventeen cases of family periodic paralysis, eighteen cases of sick headache and five cases of paralysis and headache. Fourteen members of this family have had paralysis only and thirteen have had attacks of headache only. The total number afflicted with

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* From the Wards of the Allegheny General Hospital and the Laboratory of Dr. J. P. McKelvy, Pittsburgh.

1. Westphal, C.: Ueber einen merkwürdigen Fall von periodischer Lähmung aller vier Extremitäten mit gleichzeitigen Reloschen der elektrischen Erregbarkeit während der Lähmung, *Berl. klin. Wchnschr.*, 1885, xxii, 489, 509.

2. Goldflam, S.: Ueber eine eigenthümliche Form von periodischer, familiärer wahrscheinlich autointoxicatorische Paralyse, *Ztschr. f. klin. Med.*, 1891, xix, Supplement, 240; Weitere Mittheilung über die paroxysmale, familiäre Lähmung, *Deutsch. Ztschr. f. Nervenhe.*, 1896, vii, 1; *ibid.*, 1897, xi, 242.

3. Taylor, E. W.: Family Periodic Paralysis, *Jour. Nerv. Ment. Dis.*, 1898, xxv, 637 and 719.

4. Mitchell, Flexner and Edsall: *Brain*, 1902, xxv, 109.

5. Gardner, H. W.: A Case of Periodic Paralysis, *Brain*, 1913, xxxv, 243.

6. Clark, L. Pierce: Remarks upon the Irregular and Unusual Types of Familial Periodic Paralysis and Conditions Simulating the Same with a Preliminary Report upon a New Subtype of this Palsy, *Review of Neurology and Psychiatry*, 1913.

7. Holtzapple, G. E.: Periodic Paralysis, *Jour. Am. Med. Assn.*, 1905, xlv, 1224.

either paralysis or headache was thirty-two. Of the seventeen members who have suffered from attacks of periodic paralysis, ten were still living at the time of Holtzapple's report. Six of the seven who died succumbed in an attack of paralysis, one of them in Holtzapple's presence. Of this paralytic group, fourteen were under his personal observation. The disease was transmitted through the father in six instances and through the mother in four instances; through the father having had attacks of sick headache only in four instances and through the mother having had attacks of sick headache only in three instances. In nine cases the disease was transmitted through the father, and eight through the mother. Holtzapple describes the attacks as follows:

This affection in its typical form is characterized by periodic flaccid motor paralysis, involving all of the voluntary muscles, except those of the face, eyes, tongue, organs of speech, of deglutition and the sphincters of rectum and bladder. The attacks may be distinctly periodic for a time and at short intervals, especially in young adults, but frequently later in life at much longer and irregular intervals. The paralysis may be partial or complete, localized or general; the upper extremities alone or only the lower may be involved. It may be confined to the neck, or one-half of the body may be completely paralyzed, while partial paralysis affects the other half. The paralysis may be partial in the morning and become complete during the day.

During an attack of paralysis there may be one or more periods of marked improvement, usually of a few hours' duration, followed by complete helplessness. In a typical attack of complete general paralysis the patient is utterly helpless, cannot move a finger or toe, can neither lift nor turn the head on the pillow, and if unsupported, the head either drops on the sternum or backward between the scapulae. In some instances breathing becomes distinctly labored, and deep breathing, coughing and vomiting impossible. Speech and deglutition are in rare instances affected. The order of involvement is not always the same. It most frequently begins in the muscles of the lower extremities, but may only involve those of the upper or the muscles of the neck. There are no sensory symptoms, except in some instances formication and numbness in the paralyzed parts, also a sense of heaviness and a tired feeling, which at times become very annoying to the patient, necessitating frequent change of posture, to the great relief of the patient.

Holtzapple states that "the duration of an attack may be a few hours, one, two or in rare instances, three full days." Recovery may be rapid, or it may require three or four hours or even half a day. "It is nothing uncommon for one of these patients to be as helpless as a log at 7 a. m. and at 11 a. m. be engaged in doing a hard day's work, not feeling the slightest inconvenience from having been completely paralyzed a few hours before."

The frequency of attacks varies greatly in different members of the family and also in the same individual at different periods. Attacks are most frequent between puberty and the middle period of life; later they become less frequent and more irregular. During an attack the mind remains perfectly clear. Neither cranial nerves nor special senses are involved and the patient retains full control of bladder and rectum.

During severe attacks the pulse is weak and irregular and there is evident cardiac dilatation. During the intervals between attacks the members of the family seem perfectly well.

The hereditary nature of the attacks seems thoroughly established inasmuch as every one of those paralyzed had either a father or mother who was subject to either similar paralytic attacks or sick headache or both. The headache attacks are very similar to those of ordinary hemicrania.

Holtzapple studied 139 specimens of urine, seventy-three specimens from six members of the paralytic group, thirty-six specimens from three individuals who have characteristic headache and thirty specimens from persons who were the picture of health. He found that the members of the paralytic group were not excreting normal quantities of metabolic products. In all of Holtzapple's cases an attack was brought on by overeating.

The case which Gardner reported appears to be a perfectly typical one, although there is no history of heredity in the family. He believes that the disease is due to a congenital defect of metabolism. Dr. Head offered the suggestion to Dr. Gardner that the disease might be an expression of anaphylactic reaction to some form of food such as pork. And although it was known that attacks have occasionally followed the eating of pork sausage, Gardner did not feel justified in making this experiment in his case, as, if the theory were true, the experiment would not be without danger.

Gardner summed up his reasons for believing the attacks due to presence of toxins because of some defect in metabolism as follows:

1. The many points of resemblance to other conditions which are undoubtedly due to auto-intoxication.
2. The sudden onset and the rapid recovery.
3. The invariable occurrence of the phenomena after sleep, when waste and toxic products may be assumed to accumulate, just at the time that intestinal digestion is taking place.
4. The fact that the condition might occasionally be walked off.
5. The symmetrical distribution of the paralysis.
6. The occurrence of headache, drowsiness, thirst, anorexia, aching of the limbs and sweating during the attacks. These toxic symptoms closely resemble those which obtain in other transitory toxemias.
7. The fact that previous attacks of headache, in all probability toxicemic, had been replaced by the attacks of paralysis.
8. The high specific gravity of the urine passed during the attacks, and the presence in it of small quantities of indican.

9. The presence of high pulse tension with accentuated aortic second sound and slow pulse in the initial stages and the cardiac dilatation which so rapidly followed.

And acting on this theory he prescribed for his patient as follows:

1. The patient was to give up drinking beer altogether, and to avoid all rich food; and he was to drink large quantities of water.

2. To take magnesium sulphate, 1 dram, in half a pint of hot water on rising every morning.

3. To take compound pill of mercurous chlorid, 2 grains, once a fortnight at bedtime, followed by Epsom salts in the morning.

4. To take tincture of digitalis, 4 minims; potassium acetate, 20 grains; syrup, $\frac{1}{2}$ dram, and peppermint water, 1 dram, three times a day.

5. To take caffen citrate, $7\frac{1}{2}$ grains; potassium bromid, 10 grains; diluted hydrobromic acid, 15 minims; syrup $1\frac{1}{2}$ dram, and chloroform water, $\frac{1}{2}$ dram, in a tumbler of water whenever any warning symptoms of an attack made their appearance.

He writes that the attacks which had been getting more and more frequent and threatening to incapacitate the patient at once ceased after the institution of this treatment.

The record of our own case may be inserted at this point:

History.—The patient is a healthy-looking man aged 21, single, habits good. He does not drink or use tobacco. He denies specific disease of any kind. Occupation, foreman in rubber works; he formerly worked at blacksmith's trade. He works ten hours a day. He drinks a glass of wine occasionally.

Patient's great grandmother and grandfather on his mother's side suffered from periodic paralysis, the latter dying of it. A second cousin on his mother's side (a man in middle life) suffered with this disease for many years and suffers from it now. In this cousin the disease was never as severe as in the patient and of late years has grown a good deal less severe.

The patient for the last seven years has been subject to periodic attacks of paralysis. These attacks are of irregular occurrence lasting from a half hour up to three or four days. Of the severe and more prolonged attacks he has had about three or four in a year. He experiences some kind of an attack two or three times every week. They always come on in the night. He awakens in the morning to find himself paralyzed in some part of his body; or he may waken in the night to find himself paralyzed. In severe attacks he is paralyzed in all parts of the body below the head (including the neck). The muscles of the face, tongue, eyes and throat have never been affected. He may recover within half an hour or an hour completely, even from a severe attack. During a severe attack he has only slight motion in the various members of his body. In lighter attacks he awakens in the morning to find some part of his body paralyzed. It may be one leg, or both legs, an arm or both arms, or an arm and leg on the same side or an arm on one side and a leg on the other. Neck muscles alone may be paralyzed. The legs are rather more commonly paralyzed than other parts of the body. In lesser attacks, when he awakens paralyzed, he goes about his work and gradually recovers—so gradually that he is not able to state just when improvement began; and usually about 8 or 9 o'clock in the morning he has entirely

recovered. Recalling some of the more severe attacks, he states that on the evenings preceding them he has always had a hearty meal and he attributes these attacks to the meals. He has never had pain or sensory symptoms of any kind. He has never seemed feverish or delirious, or in any way mentally upset.

The patient was admitted to the Allegheny General Hospital September 30, paralyzed all over his body, with only feeble movements in arms and legs. He was unable to raise his arms above his head, to stand or even turn in bed. This attack, which was a major one, had begun about sixty hours previously.

Examination.—Oct. 1, 1914, 2 p. m.: There is almost complete paralysis of the left arm. The right arm is paralyzed to a little less degree. There is but slight movement in legs. The patient is able to flex the feet, but not to extend them. The neck muscles are very weak. He is unable to raise the head from the bed and has only feeble power in throwing the head backward. The muscles of respiration are weak. The diaphragm is weak and he can protrude the abdomen only feebly. The knee-jerks are much reduced—quite feeble. Sensation as to temperature, pain and contact seem everywhere normal. The movements of the head and face all seem normal, also those of tongue and eyes. There is no defect of articulation, or in swallowing. There is no muscular atrophy. The patient's mind seems perfectly clear. The pulse is about 75; the blood-pressure seems somewhat reduced.

October 2, 2 p. m.: The nurse records that at 12 o'clock last night the patient seemed even weaker than in the afternoon and unable to turn himself in bed. Pulse about 50. When she observed him at 2 in the morning he seemed to have fully regained his strength.

The patient seems possessed of his normal strength of all parts of his body. He gives a firm grasp of the hand, and the legs are quite strong. There is no wasting of any of the muscles. The knee-jerks, which yesterday were very much reduced, are quite active to-day, forming a marked contrast to those of yesterday. Dr. Rosenbloom saw him, gave directions for food and diet and for examination of urine and feces. An attempted lumbar puncture was unsuccessful.

Observation at the Hospital.—Shortly after the patient was put to bed his pulse went down to 50 and continued low during the week he was in the hospital, ranging from 50 to 60. A day or two before leaving I joked with him, saying, "Why don't you get up an attack of paralysis, which you say you can do so readily?" His reply was, "You just give me a good, hearty meal to-night and I will guarantee to be paralyzed to-morrow morning."

It would seem that we need look for no further explanation of his freedom from attacks in the hospital than the diet which he took while there.

During his stay of one week in the hospital the patient seemed to be, after the first day, normal in every way. The blood-pressure was:

October 3: Systolic, 68; diastolic, 60.

October 4: Systolic, 68; diastolic, 55.

October 5: Systolic, 90; diastolic, 65.

October 7: Systolic, 92; diastolic, 72.

October 9: Systolic, 95; diastolic, 75.

From consideration of the review of the literature it will be noted that our own case is a highly typical one, and conforms in the main to the criteria laid down by Holtzapfle. The attacks seem to be per-

fectly typical and much more frequent than in most of the cases reported; heredity is plain and clear. It is also like other cases in other respects as follows: The attacks always come on at night; they are often, if not always, preceded by a heavy meal; paralysis comes on during sleep; paralysis wears off by exercise; attacks last from a few hours to and never exceeding three days; there is absence of mental symptoms; there is absence of sensory symptoms; the paralysis is of flaccid character with absence of knee-jerks; the health of the patient between attacks is perfect. Our case also lends strong support to the views expressed by Gardner regarding the nature of the disease, namely, that it is probably due to some congenital defect of metabolism.

TABLE 1.—THE NITROGEN METABOLISM—

Day	Urine †								
	Volume c.c.	Nitrogen gm.	Urea-Nitrogen		Ammonia-Nitrogen		Creatinin ‡		
			Gm.	Per Cent. of Total Nitrogen	Gm.	Per Cent. of Total Nitrogen	Gm.	Nitrogen	Percentage of Total Nitrogen
1	1,300	14.9	12.6	84.6	0.51	3.4	0.75	0.275	1.8
2	2,125	15.2	12.9	84.5	0.55	3.6	0.86	0.314	2.06
3	1,200	15.4	13.1	85.1	0.62	4.0	0.97	0.354	2.3
4	1,550	15.5	13.0	83.9	0.64	4.1	0.70	0.256	1.7
5	1,420	15.8	13.3	84.1	0.75	4.8	0.86	0.314	2.0
6	1,400	15.1	12.9	85.4	0.66	4.6	0.67	0.244	1.6
7	1,240	15.2	13.3	87.5	0.72	4.7	0.78	0.285	1.9

*Creatin was absent from all specimen of urine.

† At no time did the urine contain acetone, diacetic acid, beta-oxybutyric acid, excess of

‡ Average creatinin coefficient = 4.8 mg. creatinin-nitrogen per kilogram of body weight.

Supporting this view are the facts that undoubtedly attacks have been produced (if not always) by hearty meals; and supported further by the fact that while in the hospital and on a limited diet the patient was free from attacks.

ABSTRACT OF METABOLISM STUDY BY DR. ROSENBLOOM

We have studied in this patient, in a seven-day metabolism experiment, the nitrogen metabolism and urinary nitrogen partition, the sulphur metabolism and urinary sulphur partition, the calcium, magnesium, phosphorus and fat metabolism. We have found a very low creatinin excretion and a very high "undetermined nitrogen" excretion; these findings may indicate the type of metabolic perversion which accounts for the paralytic seizure.

This condition has so many characteristics of a toxemia that it was thought that a complete metabolic study of this patient might throw some light on the disease. Some work has been carried out in the field already. Holtzapfel found a marked diminution in the average quantity of urea eliminated by his paralytic group. As no attention was paid to diet and as the hypobromite method for the urea estimations was used, little reliance can be placed on this observation. Goldflam found that during the attacks the toxicity of the urine was increased as determined by Bouchard's method. This method has so many pitfalls that this result is of doubtful value. Singer and Goodbody⁸ estimated the amounts of nitrogen, urea, uric acid, chlorids, total sulphates,

—AND URINARY NITROGEN PARTITION *

Urine †					Feces		Intake	Balance
Uric Acid			Undetermined Nitrogen		Nitrogen		Nitrogen	Nitrogen
Gm.	Nitrogen	Percentage of Total Nitrogen	Gm.	Pct. of Total Nitrogen	Gm.	Pct. of Nitrogen Intake	gm.	gm.
0.42	0.14	0.94	1.37	9.2	1.90	12.1	15.6	—1.2
0.30	0.10	0.66	1.34	8.8	1.90	10.1	18.8	+1.7
0.36	0.12	0.78	1.21	7.9	1.90	11.8	16.2	—1.1
0.39	0.13	0.84	1.47	9.5	1.90	11.6	16.4	—1.0
0.36	0.12	0.76	1.32	8.4	1.57	9.4	16.7	—0.67
0.33	0.13	0.86	1.17	7.7	1.57	9.3	16.9	+0.23
0.33	0.13	0.85	0.76	5.0	1.57	9.6	16.4	—0.37

indican or any other abnormal constituent.

alkaline sulphates, ethereal sulphates and phosphates, in the urine of the case studied and found no marked changes in the amounts of these substances excreted. They did find a lessened amount of urine excreted during the attacks.

Crafts and Irwin⁹ asserted that they had extracted from feces passed immediately after an attack, a substance which produced temporary paralysis when injected into guinea-pigs and rabbits. They also found that the amount of urea excreted during an attack appeared to be diminished. They also estimated in the urine the amounts of nitrogen, uric acid, chlorids, purin bases, alkaline and earthy phos-

8. Singer, H. D., and Goodbody, F. W.: A Case of Family Periodic Paralysis, with a Critical Digest of the Literature, *Brain*, 1901, xxiv, 257.

9. Crafts and Irwin: *Am. Jour. Med. Sc.*, 1900, cxix, 651.

phates and preformed and ethereal sulphates without finding any marked changes. Their results as regards the isolation of a toxic substance from the feces could not be confirmed by Mitchell, Flexner and Edsall. The last-mentioned observers studied the amounts of ammonia and creatinin in the urine and seemed to think that there existed some relation between the attacks and the amount of creatinin excreted. They thought that the creatinin was an index of certain alterations in metabolism which leads up to a seizure and concluded that the attacks are due to some metabolic disturbance situated in the muscles.

TABLE 2.—THE SULPHUR METABOLISM—

Day	Sulphur gm.	Urine			
		Total Sulphate Sulphur		Inorganic Sulphate Sulphur	
		Gm.	Percentage of Total Sulphur	Gm.	Percentage of Total Sulphur
1	1.12	1.05	93.8	0.89	79.5
2	1.29	1.19	92.3	1.08	83.8
3	1.38	1.27	92.0	0.96	69.5
4	1.30	1.18	90.8	0.88	67.8
5	1.24	1.15	92.8	1.03	83.1
6	1.00	0.85	85.0	0.74	74.0
7	1.22	1.10	90.2	1.00	82.0

METABOLIC DATA

The patient was placed on Folin's¹⁰ diet, consisting of:

Whole milk	500 c.c.
Cream	300 c.c.
Horlick's Malted Milk.....	200 gm.
Sugar	20 gm.
Salt	6 gm.
Eggs	450 gm.
Water	2,000 c.c.

This diet contains approximately 119 gm. of protein, 148 gm. of fat and 225 gm. of carbohydrate, yielding 2,787 calories, or about 46 calories per kilogram body weight of the patient.

Two ounces of the foregoing diet was taken out daily and the following constituents estimated: total nitrogen, phosphorus, sulphur, calcium and magnesium. The fat was estimated in a seven-day sample, 5 c.c. of each day's mixture being preserved by means of 2 drops of liquor formaldehydi.

10. Folin: *Am. Jour. Physiol.*, 1905, xiii, 45.

A. *Method Used in Urine Analysis.*—The nitrogen was estimated according to Kjeldahl, the total sulphur by Benedict's¹¹ method. Total and ethereal sulphates by Folin's¹² method; the inorganic sulphates were computed by subtracting the ethereal sulphates from the total sulphates, and the neutral sulphur by subtracting the total sulphate sulphur from the total sulphur. Urea was estimated by Benedict's¹³ method; ammonia by Folin's¹⁴ method; total phosphorus by the Neumann¹⁵ method, weighing the phosphorus as magnesium pyrophosphate; creatinin and creatin by Folin's¹⁶ method; uric acid by Folin's¹⁷

—AND URINARY SULPHUR PARTITION

				Feces	Sulphur	
Ethereal Sulphate Sulphur		Neutral Sulphur		Sulphur	Intake	Balance
Gm.	Percentage of Total Sulphur	Gm.	Percentage of Total Sulphur	gm.	gm.	gm.
0.16	14.3	0.07	6.2	0.32	1.60	+ 0.16
0.11	8.5	0.10	7.7	0.32	1.70	+ 0.09
0.31	22.5	0.11	7.9	0.34	1.80	+ 0.08
0.30	23.0	0.12	9.2	0.34	1.50	— 0.14
0.12	9.7	0.09	7.3	0.30	1.40	— 0.14
0.11	11.0	0.15	15.0	0.30	1.30	0.0
0.10	8.2	0.12	9.9	0.30	1.32	— 0.22

method; calcium and magnesium by McCrudden's¹⁸ method, and total acidity by Folin's¹⁹ method.

B. *Methods Used in Analysis of Food.*—The nitrogen was estimated by the Kjeldahl method; total sulphur by the Wolf and Osterberg²⁰ modification of Benedict's method; calcium and magnesium by McCrudden's method after ashing and extracting the ash with hydro-

11. Benedict: Jour. Biol. Chem., 1909, vi, 363.
12. Folin: Am. Jour. Physiol., 1905, xiii, 51; Jour. Biol. Chem., 1906, 1, 131.
13. Benedict: Jour. Biol. Chem., 1911, viii, 405.
14. Folin: Ztschr. f. physiol. Chem., 1902, xxxvii, 161; Am. Jour. Physiol., 1903, viii, 330.
15. Neumann: Ztschr. f. physiol. Chem., 1902-1903, xxxvii, 129; 1904-1905, xliii, 35.
16. Folin: Am. Jour. Physiol., 1905, xiii, 45.
17. Folin: Ztschr. f. physiol. Chem., 1901, xxxii, 552.
18. McCrudden: Jour. Biol. Chem., 1911, x, 187.
19. Folin: Am. Jour. Physiol., 1903, p. 265.
20. Wolf and Osterberg: Biochem. Ztschr., 1910, xxix, 429.

chloric acid, and phosphorus by Neumann's method. Fat was estimated in a five-day period by Soxhlet extraction.

C. Methods Used in Analysis of Feces.—The feces were marked off by means of carmin into periods of four and three days, respectively. Nitrogen was estimated by the Kjeldahl method; sulphur by oxidizing with fuming nitric acid followed by Benedict's method; phosphorus by the Neumann method; calcium and magnesium by the same method as used for food, and fat by Soxhlet extraction.²¹

The accompanying tables contain the results obtained in this study.

COMMENT ON TABLE 1

In the seven-day metabolism period there was a loss of 5.81 gm. of nitrogen. The absorption of nitrogen is normal. The amount of nitrogen excreted as urea and uric acid is also normal. The creatinin nitrogen in gram and creatinin nitrogen in per cent. of the total nitro-

TABLE 3.—THE CALCIUM, MAGNESIUM—

Day	Urine			Feces		
	Phosphorus gm.	Calcium Oxid gm.	Magnesium Oxid gm.	Phosphorus gm.	Calcium Oxid gm.	Magnesium Oxid gm.
1	1.30	0.82	0.32	1.02	1.46	0.12
2	1.32	0.79	0.28	1.02	1.46	0.12
3	1.41	0.81	0.26	1.02	1.46	0.12
4	1.46	0.84	0.24	1.02	1.46	0.12
5	1.41	0.76	0.30	1.16	1.50	0.11
6	1.52	0.78	0.32	1.16	1.50	0.11
7	1.54	0.72	0.30	1.16	1.50	0.11

gen is markedly decreased as is also the creatinin coefficient. This may indicate a poor muscular efficiency of the individual, as it will be recalled that Shaffer finds the normal creatinin coefficient to be from 7 to 11 mg. of creatinin nitrogen per kilogram of body weight and thinks that it is an index of some special process of normal metabolism, taking place largely, if not wholly, in the muscles. On the other hand, it may indicate, as Mitchell, Flexner and Edsall thought, an index of certain alterations in the metabolism which lead up to a seizure, and this disturbance is situated in the muscles.

21. In addition to the references already given, the following will be found of interest:

Buzzard, E. Farquhar: Three Cases of Family Periodic Paralysis, *Lancet*, London, 1901, ii, 1564.

Atwood: C. E.: *New York State Med. Jour.*, October, 1912.

The amount of undetermined nitrogen excreted in the urine is also increased. Our knowledge of this group of nitrogenous products is very meager. We do know that the so-called neutral sulphur and colloidal nitrogen are represented in this fraction. It would be of great interest to follow the amount of undetermined nitrogen excreted in the urine during an attack. It is possible that individuals affected with periodic paralysis may be unable to metabolize those products sufficiently and therefore an increased amount is excreted in the urine, or that they produce an excessive amount of these substances and that the attack is due to retention of these products.

COMMENT ON TABLE 2

In the seven-day metabolism period there was a loss of 0.17 gm. of sulphur. The urinary sulphur partition (total sulphate sulphur, inorganic sulphate sulphur, ethereal sulphate sulphur and neutral sul-

—AND PHOSPHORUS METABOLISM

Intake			Balance		
Phosphorus gm.	Calcium Oxid, gm.	Magnesium Oxid, gm.	Phosphorus gm.	Calcium Oxid, gm.	Magnesium Oxid, gm.
2.4	2.6	0.42	+ 0.08	+ 0.32	— 0.02
2.4	2.6	0.42	+ 0.06	+ 0.35	+ 0.02
2.4	2.6	0.42	— 0.03	+ 0.33	+ 0.04
2.4	2.6	0.47	— 0.08	+ 0.30	+ 0.11
2.2	2.1	0.42	— 0.37	— 0.16	+ 0.01
2.2	2.3	0.44	— 0.48	+ 0.05	+ 0.01
2.2	2.2	0.43	— 0.50	— 0.02	+ 0.02

phur) is normal in character. The fact that the neutral sulphur is not increased shows that this type of substance is not increased in the partition of the urine known as undetermined nitrogen. There were marked increased amounts of ethereal sulphates excreted on the third and fourth day coinciding with a period of constipation which we often find in patients placed on this diet.

COMMENTS ON TABLES 3 AND 4

In the seven days there was a loss of 1.32 gm. of phosphorus, a retention of 1.17 gm. of calcium oxid and 0.19 gm. of magnesium oxid. The fat metabolism is normal with an absorption of 92.8 per cent. of the ingested fat, and the percentage of neutral fat, fatty acids and soaps in the feces fat is normal.

SUMMARY

1. The clinical history and a metabolism study of seven days' duration on the Folin diet is described in a case of family periodic paralysis.

2. We have studied the nitrogen metabolism and urinary nitrogen partition (total nitrogen, urea nitrogen, ammonia nitrogen, uric acid nitrogen, creatinin nitrogen and undetermined nitrogen), the sulphur metabolism and urinary sulphur partition (total sulphates, inorganic sulphates, ethereal sulphates and neutral sulphur). We have also studied the calcium, magnesium, phosphorus and fat metabolism.

TABLE 4.—THE FAT METABOLISM

Period Days	Fat Intake gm.	Feces					Balance gm.
		Fat gm.	Percentage of Fat Absorbed	Percentage of Neutral Fat in Feces Fat	Percentage of Fatty Acid in Feces Fat	Percentage of Soaps in Feces Fat	
7	978	70	92.8	38	24	38	908

3. The only decided metabolic changes found was a marked decrease in the amount of creatinin and creatinin nitrogen excreted in the urine and a marked increase in the amount of undetermined nitrogen of the urine.

4. It would be of great value, using the same methods described in this paper, to repeat this study on a patient suffering from family periodic paralysis during an attack, so as to correlate the findings with those described in this paper.

Westinghouse Building—5737 Forbes Street.