Open access • Journal Article • DOI:10.1001/ARCHINTE.1916.00080100009002

Hypoglycemia and progressive muscular dystrophy — Source link

R. H. McCRUDDEN, C. S. Sargent

Published on: 01 Apr 1916 - JAMA Internal Medicine (American Medical Association)

Topics: Muscular dystrophy

HYPOGLYCEMIA AND PROGRESSIVE MUSCULAR DYSTROPHY *

R. H. McCRUDDEN, M.D., AND C. S. SARGENT, S.B.
BOSTON

We recently made urine and blood examinations in a typical case of progressive muscular dystrophy which was being studied clinically by Drs. Goldthwait and Spear of Boston. The clinical findings are to be made the subject of a separate report, but the chemical findings seem well worth recording, even though they represent the data from but one case, since, if confirmed in other cases, they constitute a distinct contribution to our knowledge of carbohydrate metabolism. They are, therefore, published in the hope that similar examinations will be made by any other investigators who may observe a case of this uncommon disease.

THE OBSERVATIONS

The patient was a man 33 years of age who showed no abnormalities except progressive muscular weakness. He was put on a constant diet; the urine was saved and examined quantitatively for calcium, magnesium, nitrogen, creatinin, creatin, uric acid, and ammonia; and the blood for glucose, creatinin, creatin, uric acid, cholesterin, and nonprotein nitrogen.

DIET: The patient was kept on a constant diet throughout the period during which specimens were taken. The diet was constant in calories, and in its nitrogen, fat, and carbohydrate content; and practically identical foodstuffs were given from day to day. In spite of this physiologic constancy, the food was served in a variety of forms to the patient. This constancy in composition combined with an appearance of variety constitutes a characteristic and very useful feature of our studies at this hospital. The results are obtained by cooperation of physician and dietitian; and we believe that an adoption of the principles applied will serve to clear up many dietetic difficulties. The principle to be recognized is that the physician must prescribe the diet, for he alone knows what is needed; but the physician rarely possesses the technical knowledge necessary for putting his diet into palatable form and for giving it variety; for this he must call on the dietitian. The relation of physician and dietitian in this respect resembles somewhat that of the physician and pharmacist with respect to medicines. For convenience of explanation, the complete carrying out of the process may be divided into four steps:

1. After deciding on the approximate diet to be given, preliminary observations of a day or two are made to find out the amount of food the patient can take and his dietetic idiosyncrasies.

2. A list of foodstuffs is then given to the dietitian from which a dietary of several days is to be made out. In this case the following list was prescribed for each twenty-four hours:

4 eggs (which may be replaced in part by cheese in the proportion of 1 ounce of cheese to 1 egg)
4 glasses of milk
600 gm. of potato (which may be replaced in part by bread, spaghetti or rice in the proportion of 600 gm. of potato = 240 gm. bread = 150 gm. rice)
60 gm. butter.

* Submitted for publication Dec. 18, 1915.
* Laboratories of the Robert Breck Brigham Hospital.

All these foodstuffs are fairly constant in composition and in caloric value, so that composition and caloric value can easily be calculated. In addition, once a day, a small amount of lettuce, cucumber, celery, and tomato, and a small amount of fruit, either raw or cooked, were allowed; these latter foodstuffs have very little caloric value, and contain but little protein, fat, and carbohydrate. Such a diet has the composition shown in Table 1.

TABLE 1.—COMPOSITION OF EXPERIMENTAL DIET

	Grams	Protein	Fat	Carbohydrate	Calories
Four eggs	200	25	25	...	335
Four glasses milk	800	32	32	32	560
Potato	600	..	..	120	492
Butter	60	..	48	...	446
Total		57	105	152	1,833

3. The third step—a conference of physician and dietitian for examination and alteration of the first tentative dietary submitted by the dietitian—comes after the dietitian has studied the diet list. It sometimes happens that the diet prescribed can be improved in palatability or variety by slight alterations or additions which may or may not be permissible, depending on the exact purpose of the diet. In this particular case the diet was correct as first submitted.

4. The fourth step consists in the preparation of a dietary. For this patient we give three days' diet as examples in Tables 2, 3 and 4.

TABLE 2.—SAMPLE DIET

Date			Eggs	Milk, c.c.	Potato, gm.	Bread, gm.	Butter, gm.
	Breakfast	Baked potato	.....	.....	100		
		Toast, butter, milk	.....	200	.....	48	14
		Soft cooked egg	1				
	Dinner	Bread, butter, milk	.....	225	.....	48	14
		Escalloped spaghetti with egg	1	55	.....	.....	12
		Lettuce (dressed)	.....	.....	.....	.....	2 (oil)
19		Ice cream	1	175			
	Supper	Soup	.....	200			
		Baked stuffed potato	¼	20	200	.....	14
		Bread, butter, milk	.....	225	.....	56	14
		Baked custard	¾	100			
		Grape fruit					
	Total for the day		4	1,200	300	152	70

Besides vegetables and fruits and small amounts of sugar, flour, condiments, etc.

TABLE 3.—SAMPLE DIET

Date			Eggs	Milk, c.c.	Potato, gm.	Bread, gm.	Butter, gm.
20	Breakfast	Toast, butter, milk	.....	200	.....	48	12
		Cream toast	.....	125	.....	24	7
		Omelette	1				
	Dinner	Asparagus soup	.....	200	.....	.....	7
		Potato salad	.....	.....	200	.....	2 (oil)
		Baked stuffed egg	1	75	.....	.....	7
		Bread, butter, milk	.....	200	.....	48	12
		Lemon soufflé	¾				
		Foam sauce	¼				
	Supper	Cream of potato soup	.....	200	100	.....	9
		Croutons	.....	.....	.....	32	
		Toast, butter, milk	.....	200	.....	24	14
		Dropped egg on toast	1	.....	.....	24	
		Cut fruit					
	Total for the day		4	1,200	300	200	70

Besides vegetables and fruits and small amounts of sugar, flour, condiments, etc.

TABLE 4.—SAMPLE DIET

Date			Eggs	Milk, c.c.	Potato, gm.	Bread, gm.	Butter, gm.
21	Breakfast	Toast, butter, milk	.....	200	.....	48	14
		Grape fruit					
		Scrambled eggs	1	20	.....	.....	7
	Dinner	Soup, toast	.....	200	.....	24	
		Baked potato	.....	.....	200		
		Egg with tomato salad	1	.....	.....	.....	2 (oil)
		Bread, butter, milk	.....	200	.....	24	12
		Ice cream	1	160			
	Supper	Soup	.....	200	.....	.....	7
		Potato soufflé	½	20	200	.....	7
		Bread, butter, milk	.....	200	.....	64	14
		Orange fluff	½				
	Total for the day		4	1,200	400	160	70

Besides vegetables and fruits and small amounts of sugar, flour, condiments, etc.

We intend to make this question of dietetics as carried out at the Robert B. Brigham Hospital the subject of a separate publication and will not, therefore, go into any more details here.

URINE: The greatest care was observed in collecting the twenty-four-hour quantities of urine. This is a difficult task. The number and kind of accidents and errors that can occur in quantitative urine collections can be appreciated only by one who has made a special study of this subject. It is our belief that the results of urine analysis offered in the literature without accompanying creatinin determinations as evidence of completeness of collection are of

little value. Belief in the good intentions and trustworthiness of the nurse cannot replace creatinin determinations. With the most expert assistance, and with the most rigid system, losses and unexplainable accidents will occur. Since we intend to make this whole question of urine collection the subject of a separate publication, we will not go into details here further than to say that three factors are essential:

1. Creatinin determinations.

2. The closest oversight possible, supplemented by complete detailed ward reports concerning all the happenings to the patient written and signed by the nurse in charge (such reports serve as the basis of an investigation and often give the clue to the cause of irregularities disclosed by creatinin determinations).

3. Hearty cooperation in instituting, modifying, and carrying out the whole régime, the facilities for which are unusually good at this hospital.

The patient was kept in a room about 20 feet from the laboratory. He was not allowed to leave the room and all visitors except the day and night nurse in charge excluded. The régime to be carried out was explained to the nurses both by myself and by the superintendent of nurses and minute typewritten directions given besides. Urine was collected from 7 a. m. to 7 a. m. in special bottles containing a small amount of chloroform and alcoholic thymol solution as preservative. The bottle was kept right beside the patient's bedside in a box that was not unsightly or conspicuous. The urine secreted during the night was voided at 6 a. m. and then the bladder completely emptied again at 7 a. m. In order to avoid losses at stool, the patient was instructed to void each time before emptying the bowels. He was asked each time by the nurse if he had remembered to void in advance and also if he had lost any urine while evacuating the bowels. Reports similar to the one in Table 5 were sent to the laboratory each day with the urine.

TABLE 5.—SAMPLE OF DAILY REPORT

Patient's Name, Mr. X. Date, Oct. 22, 7 a. m. to Oct. 23, 7 a. m.

Urine	Feces	Food	Extra	Nurse
		7:30 a. m., orange, egg on toast, butter and milk	No accident	
9 a. m.	No stool		No accident	
No urine	9:05 a. m.		No accident	
11:25	No stool		No accident	
12:10	No stool		No accident	
No urine	12:15		No accident	
		12:30 p. m., potato soup, cheese, salad, bread, butter, potato with rice with egg sauce		
3:45	No stool		No accident	
		5:30, celery soup, bread, butter, milk, tomato, macaroni		7:30 a. m. to 7:30 p. m. Miss A. E. B.
9:15 p. m.	No stool		No accident	7:30 p. m. to 7:30 a. m. Miss M. A. B.
3:15 a. m.	No stool		No accident	
6:15 a. m	No stool		No accident	
7:00 a. m.	No stool		No accident	

The methods of analysis used were the following:

URINE:

Creatinin: Folin's method, described in Journal of Biological Chemistry, 1914, xvii, 470.

Creatin: Folin's original method of heating the urine with HCl for three hours on the water bath and determining creatin plus creatinin.

Uric Acid: Folin and Denis's method described in Journal of Biological Chemistry, 1913, xiv, 97.

Total Nitrogen: Kjeldahl method.

Calcium Magnesium: McCrudden's method described in Journal of Biological Chemistry, 1911, x, 187.

BLOOD:

Nonprotein Nitrogen: Folin and Denis's method described in Journal of Biological Chemistry, 1912, xi, 529.

Creatinin and Creatin: Folin and Denis's method described in Journal of Biological Chemistry, 1913, xiii, 469.

Glucose: Lewis and Benedict's method described in Journal of Biological Chemistry, 1915, xx, 61, as modified by Myers and Fine in their pamphlet on Chemical Composition of the Blood in Health and Disease, New York, 1915.

Uric Acid: Folin and Denis's method, described in Journal of Biological Chemistry, 1913, xiii, 469.

Cholesterin: Authenrieth and Funk's method, described in München. med. Wchnschr., 1913, lx, 1243.

The urine and blood examinations were all made in duplicate, except the sugar determinations, which were made in triplicate.

THE FINDINGS

Urine Examinations.—The twenty-four-hour quantity of urine varied in amount from 920 to 1,450 c.c.; it was slightly acid to litmus paper. It was made up each day to 1,500 c.c., examined qualitatively for sugar, acetone, diacetic acid and albumin (these compounds were always absent); and quantitatively for creatinin, creatin, uric acid, ammonia, calcium, magnesium, and nitrogen. The results will be found in Table 6.

TABLE 6.—URINE EXAMINATIONS

Date	Volume, c.c.	Specific Gravity	Creatinin	Creatin	Uric Acid	Total Nitrogen	Ammonia Nitrogen	Calcium	Magnesium
1	1,230	1.020	1.568	0.050	0.388	12.72	0.284	0.383	0.092
2	1,020	1.025	1.483	0.283	0.447	13.64	0.240	0.404	0.100
3	920	1.029	1.503	0.316	0.447	14.03	0.270	0.411	0.099
4	1,200	1.023	1.481	0.599	0.417	15.30	.....	0.477	0.091
5	1,130	1.022	1.493	0.624	0.385	14.34			
6	1,230	1.023	1.486	0.578	0.445	13.89			
7	1,450	1.021	1.481	0.409	0.405	14.36			
8	1,100	1.027	1.474	0.448	0.451	13.05			

Creatinin: The creatinin in the urine averages 22.6 mg. per kilo body weight, which is normal.[1] It is very constant from day to day, showing how completely and carefully the urine was saved. The variations from day to day were unusually slight; they are ordinarily

1. In a case of pseudohypertrophic muscular dystrophy Spriggs (The Excretion of Creatinin in a Case of Pseudohypertrophic Muscular Dystrophy, Biochem. Ztschr., 1907, ii, 206) found low creatinin excretion.

greater than found in this case. But we should like to point out—as a result of abundant experience—that when the twenty-four-hour quantities of urine are completely collected and the diet is constant and free from creatinin-producing substances, the excretion of creatinin is very constant, the variations being not more than 0.02 or 0.03 gm. per day from day to day.

Creatin: Creatin is present in large quantities. This is abnormal. By this method of creatin determination, which, we are convinced, is more reliable than the newer methods that appear to show traces of creatin even in normal urine, normal urine shows no creatin whatever.

Ammonia: The ammonia is normal in amount. This is significant. Other conditions in which creatin has been found present are associated with high ammonia excretion. In this case the low ammonia and absence of acetone and diacetic acid shows that we are dealing with a case of creatin-urea not accompanied by acidosis.

The uric acid, total nitrogen, calcium, and magnesium show no marked abnormalities, though the ratio of calcium to magnesium is rather high.

Blood Examinations.—Blood examination showed the results set forth in Table 7.

TABLE 7.—BLOOD EXAMINATIONS

	Mg. Per 100 Gm.
Creatinin	1.43
Creatin	3.86
Nonpretein nitrogen	28.9
Uric acid	2.30
Glucose	0.073 per cent.
Cholesterin	0.050 to 0.144 per cent.

Glucose: The glucose in the blood is low. Methods for the accurate quantitative determination of glucose in small amounts of blood have been available within only a very short time. Examination by these methods shows that the normal amount of glucose in the blood varies within very narrow limits and Allen[2] has pointed out that approximately the normal percentage is stubbornly maintained throughout prolonged starvation, almost up to death. By the method we used the variations are from 0.09 to 0.11 per cent. according to Lewis and Benedict;[3] from 0.09 to 0.12 per cent. according to Myers

2. Allen: Glycosuria and Diabetes, 1913.
3. Lewis and Benedict: Jour. Biol. Chem., 1915, xx, 61.

and Fine.[4] Our figures show an average of 0.073 per cent. (0.0731, 0.0717, and 0.0747 per cent. in three determinations).

Cholesterin: The cholesterin is very low. Of twenty-five determinations of cholesterin which we made in various other diseases all but four showed between 0.19 and 0.26 per cent. The four exceptions showed 0.17, 0.17, 0.14, and 0.15 per cent., respectively.

The nonprotein nitrogen, uric acid, creatinin, and creatin content of the blood show no abnormalities.

DISCUSSION

The striking abnormalities were, then, a low glucose and cholesterin content of the blood, and the presence of creatin in the urine.

The *creatin* in the urine suggests some abnormality of the glucose metabolism. It is found in the urine in conditions like diabetes and starvation when sugar is not being properly oxidized, and in such cases is usually associated with acidosis. In the present instance the normal ammonia excretion and absence of acetone and diacetic acid from the urine show that we are dealing with creatinuria without acidosis.

The low *cholesterin* content of the blood has no definite significance in the present state of our knowledge of the physiology of this substance. The most that can be said is that it makes us think of the possibility of involvement of the adrenals, a possibility that is still further indicated by the hypoglycemia.

The association of muscular weakness with *hypoglycemia* is striking, for we know that the muscle gets its power through oxidation of sugar; and a low sugar content of the blood might be looked on as sufficient cause for muscular weakness. Many investigators have demonstrated the power of sugar to make exhausted muscles capable of more work; the subject, with literature, is discussed by Fürth and Schwarz.[5] A direct relationship between muscular weakness and hypoglycemia has been noted by different investigators. Weiland,[6] using a Gärtner ergostat, had several of his colleagues do severe muscular work, almost to the point of exhaustion, and determined the glucose content of the blood before and after the work. In every case there was a decrease in the glucose content of the blood, the average decrease being 27 per cent. In three cases of Addison's disease — a condition

4. Myers and Fine: Chemical Composition of the Blood in Health and Disease, New York, 1915. The results by older methods are less to be relied on.

5. Fürth and Schwarz: Ueber die Steigerung der Leistiengsfähigkeit des Warmblütermuskels durch gerinnungsbefördernde Muskelgifte, Pfluger's Arch. f. d. ges. Physiol., 1909, cxxix, 525.

6. Weiland: Ueber den Einfluss ermüdender Muskelarbeit auf den Blutzuckergehalt, Arch. f. exper. Path. u. Pharmakol., 1907-08, xcii, 223.

characterized by intense muscular weakness—quantitative determinations of the glucose content of the blood by Porges[7] showed 0.052 per cent., 0.033 per cent., and 0.067 per cent.— all very low results. Porges followed this point further in experiments on animals. He removed the adrenals from several dogs and compared the glucose content before and a few hours after the operation. His figures are as shown in Table 8. In every case there was a decrease in the glucose content of

TABLE 8.—GLUCOSE CONTENT BEFORE AND AFTER REMOVAL OF ADRENALS

	Per Cent. Before	Per Cent. After
Dog 1	0.258 0.103	0.0058 0.0057
Dog 2	0.120	0.033
Dog 3	0.084	0.066
Dog 4	0.092	0.044

the blood. Mayer[8] had previously reported a fall in the blood sugar content in cats after removal of the adrenals. This association of fall in blood sugar content with muscular asthenia and decrease in adrenalin is quite in harmony with the fact previously observed by Batteli and Boatti[9] and by Schur and Wiesel[10] that the epinephrin content of the blood of dogs decreases when these animals are made to undergo exhausting work on a treadmill. An association between muscular asthenia and hypoglycemia has been noted in a case of dyspituitarism and in atrophied babies. In Cushing's[11] book on the hypophysis there is a case history of a man suffering from dyspituitarism, showing marked muscular asthenia and low blood sugar (0.039 and 0.053 per cent. in two determinations). Frank[12] observed in three atrophied babies, respectively, 0.046 per cent., 0.040 per cent., and 0.050 per cent. blood sugar (0.10 to 0.11 per cent. is the average figure for normal babies).

Muscular asthenia alone, secondary to some other condition, is not necessarily accompanied by hypoglycemia. Through the kindness of

7. Porges: Ueber Hypoglykämie bei Morbus Addison sowie bei nebennierenlosen Hunden, Ztschr. f. klin. Med., 1909-10, lxix, 341.

8. Mayer: Ablation des surrénales et diabéte pancréatique, Compt. rend. Soc. de biol., 1908, lxiv, 219.

9. Battelli and Boatti: Influence de la fatique sur la quantité d'adrénaline existant dans les capsules surrénales, Compt. rend. Soc. de biol., 1902, liv, 1203.

10. Schur and Wiesel: Beiträge zur Physiologie und Pathologie des chromaffinen Gewebes, Wien. klin. Wchnschr., 1907, xx (2), 1202.

11. Cushing: The Pituitary Body and Its Disorders. Phila., 1910, p. 130.

12. Frank: Ueber einige Grundtatsachen aus der Physiologie des Blutzucker nebst methodisher Vorbemerkungen, Ztschr. f. physiol. Chem., 1910-11, lxx, 129.

Dr. Goldthwait we had an opportunity to examine the blood in two very rapidly progressing cases of chronic arthritis of the infectious type, one of them of less than twelve months' duration, the other of less than six months' duration. In both cases there was a very marked wasting of the muscles. Examination showed 0.119 per cent. and 0.120 per cent. blood sugar, respectively, in the two cases. The significant feature in these cases was not muscular weakness alone, but rather muscular wasting.

As to the cause of the low sugar content of the blood, two possibilities may be imagined:

1. Hypoglycemia can be brought about by a lowered threshold value for excretion of glucose through the kidneys — renal diabetes. Thus the sugar content of the blood can be lowered by the administration of phloridzin, a drug which increases the permeability of the kidney for glucose and causes glucosuria. With the patient we had under observation the fact that glucose was absent from the urine excludes this as a possible cause of the hypoglycemia.

2. Glucose is constantly passing from the blood into the muscles to undergo oxidation; this loss is made up by the introduction of glucose into the blood from the glycogen store in the liver; and hypoglycemia can be brought about as the result of loss of balance between the rate at which glucose is introduced into the blood and the rate at which it disappears from the blood into the muscles. This regulation of the rate of formation of glucose from glycogen is under the control of the adrenals, the pituitary body and the thyroid gland;[13] administration of these substances or stimulation which increases the amount of adrenal principle in the blood leads to hyperglycemia.[13] That the cause of hypoglycemia in Addison's disease and after removal of the adrenals is associated with a decreased formation of glucose from glycogen is evident from the experiments of Porges,[14] who showed that after removal of the adrenals from dogs the liver becomes practically free from glycogen. Porges[15] followed the subject still further and noted that adrenal principle increases not only the rate of formation of glucose from glycogen, but also the rate of formation of glycogen from glucose; so that when the adrenals are diseased the liver loses its power to store glycogen. Frank and Isaac,[16] furthermore, have shown that the hypoglycemia resulting from phosphorus poisoning results from a loss of power of the liver to store glycogen.

13. The literature on this subject is voluminous.

14. Porges: Ueber Hypoglykämie bei Morbus Addison sowie bei nebennierenlosen Hunden, Ztschr. f. klin. Med., 1909-10, lxix, 341.

15. Porges: Zur Pathologie des Morbus Addison. II. Ueber Glykogenschwund nach doppelseitiger Nebennierenextirpation bei Hunden, Ztschr. f. klin. Med., 1910, lxx, 243.

16. Frank and Isaac: Ueber das Wesen des gestörten Stoffwechsels bei der Phosphorvergiftung, Arch. f. exper. Path. u. Pharmakol., 1910, lxiv, 274.

The possibility that the low sugar content of the blood in our case might be due to inability to store glycogen as effectively as normally, led us to determine the glucose content of the blood after a short period of starvation. Normally, as fast as the glucose of the blood is oxidized, a continuous new supply resulting from glycogenolysis maintains the glucose of the blood at its normal level; starvation and ingestion of food has but little effect on the glucose content of the blood. Any interference with glycogen storage might become apparent by a fall of the glucose content of the blood after a short period of starvation. It was not our purpose really to starve the patient, but merely, by omitting a meal, to exclude the effect of food on the glucose content of the blood; the patient was asked merely to omit his breakfast on one day. He went without food from 6 p. m. one evening until noon the next day, when blood was again taken for examination. The glucose content was 0.064 per cent. (triplicate determinations showed 0.0651, 0.0628 and 0.0637 per cent., respectively) at this time — decidedly lower than when previously taken. The decrease is at least suggestive, though not of course to be taken as definite indication of any disturbance in glycogenolysis.

Another fact of interest to be mentioned here is the relationship between loss of power to store glycogen and fatty transformation. In one of his experiments on dogs Porges[15] noted that during the operation of removing the adrenals the liver was normal; at necropsy, shortly afterward, it was observed that the liver had undergone fatty transformation; in other words, fat was stored instead of glycogen. A similar observation is recorded by Frank and Isaac;[16] these investigators noted that the loss of power of the liver to store glycogen which results from phosphorus poisoning was accompanied by a storage of fat instead. The fact noted by Cushing[11] that fatty transformation of the liver cells accompanies states of hypopituitarism — another condition associated with muscular weakness and hypoglycemia — may be mentioned in this connection. In view of these facts showing a relationship between hypoglycemia, fat storage and loss of power to store glycogen, the significance of the deposition of fat in the muscle cells in cases of pseudohypertrophic muscular dystrophy becomes apparent; the fat storage may well be due to an inability of the muscle to exercise its normal function of storing glycogen, fat instead being stored.

TREATMENT

In view of the facts connecting hypoglycemia, muscular asthenia and diminished activity of the adrenals and hypophysis, Dr. Spear suggested the use of epinephrin and pituitary extract for treatment. A detailed report of the treatment and its results will be made elsewhere. The point to be brought out here is the relationship between improvement in the physical condition and the increase in blood sugar.

Improvement in health, strength and weight was prompt and marked; and, with the improvement, the sugar content of the blood rose first to 0.080 per cent. then to 0.099 per cent., the latter a normal amount. An increase in the cholesterin content of the blood accompanied the increase in sugar content; with 0.080 per cent. sugar the cholesterin content was 0.177 per cent; with 0.099 per cent. sugar the cholesterin content was 0.211 per cent., the latter a normal amount.

As a preliminary to leaving our care entirely and returning to his home, the patient was allowed to leave the hospital and do much as he pleased in the city. At the end of a week his condition showed no change; the blood sugar showed a slight drop to 0.087 per cent., a figure which is only slightly below the normal average.[17]

It may be wise here to warn for the present against any attempts to treat such conditions by intravenous administration of glucose, a form of treatment that might naturally suggest itself. Underhill[18] tried this form of treatment on three dogs which showed hypoglycemia and muscular asthenia as a result of hydrazin poisoning; the animals all died within a few hours.

SUMMARY

In a case of progressive muscular dystrophy, the creatinin excretion was normal; large amounts of creatin were found in the urine; the ammonia excretion was low; there was no evidence of acidosis; the amount of sugar in the blood was low; the cholesterin content of the blood was low. A relationship between sugar content of the blood and ingestion of food suggested the possibility of a decreased power to store glycogen. Treatment which increased the glucose and cholesterin content in the blood led to improvement in the clinical condition.

We intend to follow this subject further. But since this disease is not common, our present results are published in order to call attention to the desirability of determining blood sugar in other cases of a similar nature. If confirmed in other cases, the association of hypoglycemia and muscular asthenia in this disease is, of course, of great significance in the physiology of carbohydrate metabolism.

Robert Breck Brigham Hospital.

17. Two months after leaving us we received a letter from the patient, who is in a distant part of the country, stating that he had continued taking epinephrin and pituitary extract; that he had gained much in weight and strength, and that his muscles were larger and firmer.

18. Underhill: Studies in Carbohydrate Metabolism. 1. The Influence of Hydrazin upon the Organism with Special Reference to the Blood Sugar Content, Jour. Biol. Chem., 1911-12, x, 159.