THREE CASES
OF
PROGRESSIVE MUSCULAR DEGENERATION.

BY
I. N. KERLIN, M.D.,
Superintendent of the Pennsylvania Institution for Feeble-Minded Children,

AND
CHARLES K. MILLS, M.D.,
Neurologist to the Philadelphia Hospital.

EXTRACTED FROM THE
TRANSACTIONS OF THE AMERICAN MEDICAL ASSOCIATION.

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The three patients discussed in this paper are three brothers, who have been for several years inmates of the Pennsylvania Institution for Feeble-Minded Children at Media.

Case I.—J. M., the eldest of the brothers, was, when examined in January, 1880, twenty-eight years old. At the time of his birth, the age of his father was only twenty-one years, and that of the mother eighteen years. He showed signs of deficient animation at birth. He was the first-born of the family. When between four and five months old he had convulsions, but not afterwards. During infancy he was troubled with indigestion and diarrhoea. His parents noticed that his limbs seemed weaker than those of other babies of the same age. He could not walk until he was four years old. He was admitted to the Pennsylvania Institution for Feeble-Minded Children in 1873. Unfortunately, no facts of value in regard to the condition during childhood, of either this patient or his brothers, could be obtained.

His weight was 83½ pounds; height, 5 feet 4½ inches. He had almost the appearance of a living skeleton, except in the gluteal regions and the calves, which were better developed than other portions of the body. The muscles of the back, and of the chest and abdomen, were very much atrophied; those of the shoulders, arms, and forearms were also wasted, but not quite so markedly; in the lower extremities the wasting was most pronounced in the thighs. His mouth was full and firm. His hands were well shaped. The face, eyes, and limbs showed no
localized paralyses, and no deformities or contractures. The wasted muscles were, however, weak, the weakness varying in different muscular groups. Both grips were poor, the left better than the right. The extensor muscles of the thighs were extremely weak; lying in bed, he could flex the thighs on the pelvis, but not readily; and he could only extend them again by a wriggling, downward movement, performed with great effort.

The following measurements were made: right semi-circumference of the chest, 13½ inches; left, 13¾ inches; right semi-circumference of abdomen, 12¾ inches; left, 12½ inches; length of the right arm, 26 inches; of left, 27 inches; circumference of middle of right arm, 7½ inches; of left, 7½ inches; right forearm, 5½ inches; left, 7¼ inches; length of right leg, 33 inches; of left, 33½ inches; circumference of right thigh, 10¼ inches; of left, 10¾ inches; right calf, 10½ inches; left, 10¾ inches.

In standing, his feet were planted widely apart, one in advance of the other. The upper part of the trunk was thrown far backward, and the belly forwards, producing a characteristic lordosis, the scapula assuming a wing-like appearance. The upper part of the spinal column showed a slight lateral curvature to the left. Without support he could stand only for a few minutes. In walking he carried himself in the same position as when standing, lifting himself forwards with a twisting and swinging movement on the balls of his feet. He could only move along very slowly, assisting himself by taking hold of anything within reach.

Farado-contractility was retained in varying degree, being least in those muscles which were weakest and most wasted. It was diminished in all. Fibrillary tremors were not observed during any of the examinations. Good reflex responses could be brought out from the skin. The patellar reflexes, however, were abolished. His hands and feet, on slight exposure, rapidly became cold and blue, but this condition did not remain. Numerous yellowish patches were scattered over his chest and abdomen. Hair was scanty on his head and face, but comparatively abundant on his body. He complained sometimes of sensations of numbness, and frequently of feelings of fatigue and soreness in his limbs and body. Cutaneous sensibility—tested for touch, pain, pressure, heat, and cold—was found to be
normal. Electro-muscular sensibility was lowered. Hearing was diminished in acuteness about one-half in each ear. Accommodation was good, but acuteness of vision was considerably decreased. His eyes were large, gray, staring, and apt to be fixed in one direction. He was slow in concentrating his attention on objects of sight and hearing. His speech was slow, and slightly hesitating. Appetite and digestion were good, but he showed some tendency to constipation. He was of energetic disposition.

Physical examination of the lungs showed some dulness on percussion, both at apices and bases; it was greater on the right side. Vocal fremitus was increased, and the lungs did not properly expand. Inspiration was short, expiration prolonged. The abdomen was not thrown forward as in normal respiration. Respirations were 24 to the minute. Lately he had had frequent coughing spells with yellowish expectoration. The impulse of the heart was weak, but no murmurs were present.

During the last seven years, two or three times annually, he had had febrile attacks lasting several days, and accompanied by pains in the back and limbs. After these spells a notable decrease in strength was always observed.

Case II.—H. M. was twenty-two years of age at the time of examination. He never had convulsions, but he suffered during infancy from digestive and intestinal disorders, and early gave evidences of muscular weakness. The exact age at which he became able to walk could not be learned.

His weight was 80½ pounds; height 5 feet 1½ inch. The size, shape, and contours of trunk and limbs were better in this patient than in either of the other two. The muscles of the trunk, upper extremities (except the deltoids), and thighs all exhibited decided wasting. The calves of both legs were, however, enlarged, presenting a pseudo-hypertrophic appearance. He had slight left lateral curvature of the spine. He walked with his shoulders and arms thrust backwards, the latter being flexed and tense, and the fists clenched. His feet were without spring, and his mode of walking caused a sort of rocking movement of the pelvis. His method of rising from a sitting position on the floor was that described by Gowers and others as “climbing up the thighs.”

The gluteal muscles and deltoids were also somewhat enlarged.
Weakness, but without definite palsies or deformities, was the condition here as in the first case. He did not seem to have ready control of the movements of his eyes and face.

The following were the measurements: Right semicircumference of the chest, 14½ inches; left, 14¾ inches; right semicircumference of abdomen, 12⅛ inches; left, 12¾ inches; length of right arm, 27 inches; left, 27 inches; circumference of middle of right arm, 7¾ inches; of left, 8 inches; right forearm, 8 inches; left, 7⅛ inches; length of right leg, 38⅝ inches; of left, 38⅔ inches; circumference of right thigh, 11 inches; of left, 10½ inches; right calf, 12¾ inches; left, 12 inches.

He could stand alone with his heels together for a few minutes. He stood by preference with his feet well separated. The shoulders and upper part of the trunk bent backwards, but not as markedly as in the other cases.

In regard to farado-contractility, fibrillar tremor, superficial and deep reflexes, vaso-motor manifestations, sensibility, and special senses, attention, speech, and general state, the conditions in this case were practically the same as those shown by the elder brother. None of the yellowish patches were present on the skin.

Respiratory movement was imperfect. A slight aortic regurgitant murmur was present. The pulse was 80, and somewhat feeble and irregular.

For several years every two or three months he had had attacks of acute illness, the chief symptoms being nausea, vomiting, constipation, fever, cough, and muscular soreness.

Case III.—F. M. was seventeen years old at the date of examination. His history during infancy was the same as that of each of the other boys. He never, however, like the oldest, had convulsions. When admitted to the Institution, the disease was less marked than in either of the other cases, but since admission his downward course had been the most rapid of any.

His weight was 74 pounds; height, 5 feet, 2½ inches. In this, as in the other cases, the muscular structure of the trunk and thighs showed the greatest wasting. All the muscles of the trunk were remarkably atrophied. The glutei muscles and those of the calves were comparatively large; and the muscles of the upper extremities were not so much wasted as other parts of the body. Palsies and deformities were absent.
The measurements were as follows: Right semi-circumference of the chest, 14½ inches; left, 14½ inches; right semi-circumference of abdomen, 11½ inches; left, 12 inches; length of the right arm, 26½ inches; of left, 27½ inches; circumference of middle of right arm, 6½ inches; of left, 6¼ inches; right forearm, 7 inches; left, 7 inches; length of right leg, 38¼ inches; of left, 39½ inches; circumference of right thigh, 11 inches; of left, 11¾ inches; right calf, 12½ inches; left, 12 inches.

He could not stand without support for more than a minute. It will be unnecessary in this case to go into details in regard to standing, walking, sitting down and rising, farado-contrac-
tility, fibrillary tremor, reflexes, vaso-motor and sensory symp-
toms, state of lungs, etc. The conditions and manifestations were strikingly similar to those found in the other two cases, differing only in degree.

We are led, from a study of these cases, strongly to favor the idea of the essential identity of progressive muscular atrophy and pseudo-hypertrophy of the muscles, as advocated by Fried-
reich and Eulenburg, who have made a thorough investigation of both diseases. Friedreich, quoted by Eulenburg, says¹ of pseudo-hypertrophy, that "it is a form of progressive muscular atrophy modified by the peculiar strength of the diathesis and by certain special attributes of childhood." Eulenburg thor-
oughly discusses the question of the identity or independence of the two affections. His chief arguments in favor of identity are (1) Agreement in the fundamental histological changes of the muscle; (2) The extremely frequent coincidence of pseudo-
hypertrophy of single muscles with diminution in the size of others, near or remote, which are similarly impaired in their functions; (3) Essential agreement in respect to etiology and clinical course.

Gowers² devotes scant attention to this matter, but concludes that the pseudo-hypertrophic paralysis of early life is not a dis-
ease of the spinal cord, is not an infantile spinal myo-atrophy similar to the common forms of progressive muscular atrophy of adults.

It will be found, we believe, that Eulenburg's arguments are well borne out by a study of our cases.

By means of Duchenne's histological harpoon a small fragment was removed from the gastrocnemius muscle of the left leg of each patient. The specimens obtained were too minute for satisfactory sections to be made. Dr. G. M. Christine, however, prepared them by teasing, stained them with carmine, and temporarily mounted them in oil of cloves. They were examined microscopically by Dr. E. O. Shakespeare.

In the three specimens nearly the same condition was found. Some of the fibres presented normal appearances; in others, no fibrillation could be distinguished. In some the muscle corporcles were more or less increased in number. In a few the sarcolemma appeared to be crammed with an accumulation of embryonic cells. In some bundles the muscle fibres were not separated by more than a normal amount of interstitial connective tissue. In others, however, this tissue was both increased in quantity and was in a state of cellular hyperplasia. No fat-vesicles could be seen.

This investigation was, of necessity, unsatisfactory. As has been pointed out by Gowers and others, the use of the harpoon gives rise sometimes to mistakes, the very small specimens obtained not being sufficient to show the entire pathological condition present. We did not, however, feel justified in resorting either to numerous harpoonings or to excisions of muscle. The absence of fat-cells may have been accidental, owing to the method of obtaining and preparing the specimens. The examinations showed, at least, the existence of a chronic interstitial inflammatory process, and were, so far as they went, in agreement with the researches of Friedreich and Eulenburg, to which we will here briefly refer.

According to Friedreich's investigations, as given by Eulenburg, progressive muscular atrophy consists in an essentially inflammatory process, a "polymyositis chronica progressiva." The first changes begin in the perimysium internum as hyperplastic growth of the interstitial connective tissue, in its finest ramifications among the single primitive bundles. At the same time there occur phenomena of irritation in a greater or smaller number of primitive bundles, in the form of swelling and multiplication of the muscular corporcles, and especially proliferation of their nuclei, and sometimes parenchymatous granular cloudi-
ness of the cross-striped fibrillary substance. In a few cases, hypertrophied muscular fibres and a dichotomous or trichotomous division of the hypertrophied fibres (Friedreich) were seen. During this increase in interstitial tissue, the wasting of the muscular substance goes on in various ways, partly by simple atrophy and progressive dissolution, without loss of transverse striation (sometimes preceded by longitudinal, transverse, or elementary fission), and partly by waxy or fatty degeneration. The final result is a more or less complete fibrous degeneration (cirrhosis) of the muscle. An accessory process, by no means constant or essential, is the diffused lipomatosis of the muscle, which appears sometimes early, sometimes late in the course of the disease."

According to these authors again, in cases of pseudo-hypertrophy, such muscles as are attacked without being hypertrophied present the same appearances as are found in progressive muscular atrophy. The abundant development of fat, interstitial and even inter-fibrillary, is not the primary fact, but is preceded by a stage of hyperplastic development of connective tissue, so that the whole must be spoken of as an irritative, inflammatory process, as in progressive muscular atrophy.

The coincidence of pseudo-hypertrophy of some muscles with atrophy of others, was particularly shown in Case II. In this patient, the enlarged calves were almost typical of pseudo-hypertrophy, although they were by no means of the enormous dimensions sometimes attained in reported cases. In the youngest of the three, the glutei muscles and those of the calves were comparatively large. Gowers, speaking of the muscular enlargement, says, "that, while pathognomonic when present in conjunction with the other symptoms, its absence is of little importance, since muscles gravely diseased may be of normal size."

In regard to etiological conditions several characteristics, some of which are illustrated by these cases, are presented almost equally by pseudo-hypertrophy of the muscles and progressive muscular atrophy. Both diseases show a remarkable tendency to affect several members of the same family; in both males are much more frequently attacked than females; in both heredity has been proved to play a very important part.

Let us glance, for the sake of comparison, at some of the records of the two diseases as given by Hammond, Eulenberg, 1 Ibid., p. 133-134.
and Gowers. Meryon gives an account of four brothers who were attacked by progressive muscular atrophy; Eulenburg, senior, describes twin brothers affected by the same disease; Eulenburg, junior, relates the particulars of a family of seven, out of which two brothers and two sisters were attacked. Friedreich's statistics show that of 176 cases only 33 were females. In one remarkable instance eight boys were attacked while four sisters were not. All of Hammond's cases but one were males. The remarkable history of the Wetherbee family will be recalled by all familiar with Dr. Hammond's treatise; in four generations nine or ten members of this family were the victims of this disease. Dr. Hammond also refers to the instructive histories related by Nanny, who gives an account of a family in which six generations were subject to the disease.

Turning now to pseudo-hypertrophy of the muscles, we find that in four of the examples given by Gowers, the tendency to affect several members of the same family was noticeable. In one family four cases and in another six occurred. Lutz described six cases in one family. Barsichow gives an extraordinary series of twenty-four cases of an analogous malady in two connected families. Meryon mentions an instance in which eight brothers all died of this disease. Numerous cases of two children of the same parents are reported. Heller, Wagner, and Seidel report cases of three brothers; Lutz and Eulenburg of three sisters. Of 220 cases collected by Gowers, 102 were apparently isolated, and 118 were grouped in thirty-nine families. It is probable, however, as this author says, that these statistics underrate the frequency with which more than one member of a family is affected because, in many of the examples, other members of the family were young, and may have subsequently presented symptoms of the disease, and in some instances it is not evident that the point was investigated.

Of forty-four original cases described by Gowers, the relationship of the subjects of the disease was as follows: In eighteen families two brothers were affected; in five, three brothers; in three, four brothers; in one, eight brothers; in two families, a brother and sister; in two families, two brothers and a sister. In the remaining twelve families the members affected were: one boy and his uncle; three brothers and uncle; a son, aunt, and uncle; two brothers, uncle, aunt, and great-uncle; two brothers, two cousins, and two uncles; two brothers, sister, and
two cousins (also brothers); four brothers, aunt, and uncle; two sisters and brother; three sisters, uncle, aunt, and cousin. Poore gives an interesting analysis of cases with reference to the occurrence of the disease in numerous members of the same family. In regard to sex, we will simply refer to the statistics of Gowers as the latest and fullest: of 139 cases, 123 were males and 16 females.

The tendency to attack males and members of the same family is, of course, illustrated by our cases.

The following are our notes upon heredity with reference to these cases:

No history of either pseudo-hypertrophy of the muscles or of progressive muscular atrophy in the ancestry of the patients could be obtained, but we were unable to trace the family history further back than to the grandparents. Nothing could be learned in regard to collateral branches of the family. The paternal grandfather and grandmother died at 62 and 83 years respectively, the maternal grandparents at 60 and 80 years. The character of the diseases which carried them off could not be ascertained. The father died of what was pronounced to be intestinal tuberculosis. He was addicted to the use of alcohol. The mother died paralyzed when only thirty-three years old. She is said to have been deaf, scrofulous, and for several years an epileptic. She had two miscarriages and five living children—three boys who form the subjects of this paper, and two girls, both of whom are healthy. Neither parents or grandparents were related before marriage. Each of the children was born at full term, the labors being easy. The mother was not subjected during pregnancy to any continuous anxiety or hardship, or exposed to any shock, accident, or especially painful emotion.

If pseudo-hypertrophy of the muscles and progressive muscular atrophy are looked upon as independent affections, these cases, in spite of the great preponderance of atrophy at the date of their examination, would belong more properly to the pseudo-hypertrophic type. The disease in each case began in early childhood, and pseudo-hypertrophic muscular paralysis, as described by all authors, is a disease of infancy and childhood, while progressive muscular atrophy in its ordinary form usually

1 New York Medical Journal, June, 1875.
begins in adult life. Almost every point of supposed distinction can, however, be shown not to be absolute. We have ourselves seen one case of pseudo-hypertrophy beginning in adult life. Dr. A. Hughes Bennett \(^1\) reports an adult case. In eighty cases collected by Eulenburg five began after adult life.

A few more points in the symptomatology of these cases are deserving of attention.

When between four and five months old the oldest of the patients had convulsions. We think it doubtful, however, whether any causative or other relations existed between this attack and the development of the disease. Duchenne and Benedikt have reported cases in which epileptiform convulsions are said to have preceded the appearance of pseudo-hypertrophy of the muscles. In two of our three patients no spasmodic attacks of any kinds had ever occurred.

It is not without interest and possible importance that the three patients suffered during infancy from indigestion and diarrhea.

The methods of rising and of standing and walking were carefully studied in these cases. Gowers fully considers the subject of the attitude and gait of patients suffering from pseudo-hypertrophy. He also gives a series of illustrations showing the mechanism of the movements performed by these patients in rising from the floor or a chair. His remarks on the lumbar lordosis, which was so marked a manifestation in his and our cases, are very interesting.

Fibrillary tremors or contractures are looked upon by some as of pathognomonic importance in progressive muscular atrophy; and are said to be absent in pseudo-hypertrophy. These movements, in our three cases, did not appear spontaneously and were not provoked by external agencies during our examinations. These tremors are not, however, absolutely pathognomonic of progressive muscular atrophy. According to Duchenne they are present in only one out of five cases. We believe, however, that the average frequency of their presence is much greater than this. According to Wagner, Eulenburg, Rosenthal, and others, they are not infrequently to be observed in pseudo-hypertrophy. Eulenburg says that the twitchings in the pseudo-hypertrophied muscles are less striking to the eye, on account of the masses of

\(^1\) Brain, October, 1879.
fat. They were not, however, in our patients to be seen, either in the atrophied or hypertrophied muscles.

Good reflex responses could be elicited from the skin in the three cases, but in all the patellar reflexes were abolished. According to Gowers\(^1\) the patellar reflex is a true reflex action, depending on the integrity of the reflex loop at the level of the second and third lumbar nerves. It is impaired by disease of any part of this loop—(1) of the posterior nerve-roots, outside the cord, or in the posterior column; (2) of the gray matter; (3) of the anterior roots; (4) of the mixed nerve-trunks. It is also exceptionally absent in normal conditions. In our cases wasting of the quadriceps extensor was very marked. As different pathological investigations have revealed, in addition to muscular degeneration, atrophy of peripheral nerve-fibres, and changes in the anterior roots and in the ganglion cells of the anterior horns, we have a choice of explanations of the destruction of the integrity of the reflex loop on which the production of the knee phenomenon is dependent.

The three patients were very sensitive to cold, and the hands and feet of the two eldest became temporarily blue on exposure. Marked mottling of the skin has been observed in some of the reported cases of pseudo-hypertrophy. Painful swellings of the joints, large or small, had never been present. Fatigue-pains and sensations of numbness and crawling were among the sensory manifestations. Electro-muscular sensibility was diminished. This has been usually found to be the case in progressive atrophy; in pseudo-hypertrophy, according to Eulenburg, it is sometimes increased and sometimes decreased. Physical examination of the chest seemed to indicate that, besides the accessory muscles of breathing, the diaphragm, and perhaps even the heart itself, were involved in the process of degeneration.

These cases, it will be remembered, are inmates of an institution for the feeble-minded and idiotic. Although of energetic dispositions, they showed considerable intellectual deficiency. The condition of speech, and of the special senses also, indicated some cerebral involvement. We are told by Gowers that in most recorded cases the mind has been unaffected. \textquoteleft\textquoteleft In some, however,\textquoteright\textquoteright he says, \textquoteleft\textquoteleft there has been mental dulness, and even actual idiocy.\textquoteright\textquoteright

Several of such cases have been reported by Dr. Langdon Down. It appears, therefore, that mental defect is not part of the disease, but that the muscular affection is rather more common among children who have mental defect than it is among others. In a few cases on record there have been epileptic fits, probably, like the mental defect, the result of an associated, not of a related, cerebral disease.

It is evident that in all diseases in which muscular degeneration is prominent, and particularly in those in which it is the chief peculiarity, careful examinations of the urine may be of considerable value. Foster\(^1\) considers it a more or less probable view that the kreatin found, and presumably formed, is a more or less distinct antecedent of urea. Other things being equal; in diseases in which muscular wasting is excessive, we should expect an excess of urea. Kreatin itself is not a normal constituent of the urine. Kreatinin, however, which is simply a dehydrated form of kreatin, occurs normally as a constant constituent of urine and of muscle extract.

We have been unable to find any records of urinary analyses in cases of pseudo-hypertrophy of the muscles. Eulenburg makes the following observations in regard to progressive muscular atrophy. "Concerning the qualitative and quantitative changes in the composition of the urine, there are but few and very discordant statements. Freidberg and Fromman each observed in one case a deposit of lime in the urine; the latter found the urine pale, turbid, slightly alkaline, with abundant sediments of carbonate of lime, which he believed to originate from the atrophied muscles. Bamberger, however, found in one case urea and chloride of sodium in normal or increased quantity, great increase of sulphuric acid, and considerable diminution of the uric and phosphoric acids. An examination of the blood at the same time showed a considerable increase in albumen and blood-corpuscles. More important, but not yet confirmed by other observations, is the increase of kreatinin in the urine, observed by M. Rosenthal in three cases. Friedreich found the urine acid in three cases, of quite high specific gravity, containing more or less constant sediments of urates, and free from albumen."

\(^1\) Text-Book of Physiology.
\(^2\) Ibid., p. 128.
PROGRESSIVE MUSCULAR DEGENERATION.

Analysis of Urine, February 3, 1880.

<table>
<thead>
<tr>
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<th>Case I</th>
<th>Case II</th>
<th>Case III</th>
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<td>Quantity in 24 hours</td>
<td>976 c.c.</td>
<td>1500 c.c.</td>
<td>1869 c.c.</td>
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<tr>
<td>Specific gravity</td>
<td>1013</td>
<td>1010</td>
<td>1013</td>
</tr>
<tr>
<td>Urea</td>
<td>13 grammes</td>
<td>24.3</td>
<td>17.55</td>
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</table>

The color was in each case reddish-yellow; reaction acid; phosphates about one-half the normal; chlorides and sulphates were only examined for qualitatively, but were estimated as nearly normal; crystals of triple phosphates were present; no albumen or sugar.

The diet for the day was as follows: Breakfast, stewed potatoes, bread and butter, and coffee; dinner, cold tongue, potatoes, blanmange, milk, apples, and crackers; supper, bread and butter, and tea.

Analysis of Urine, May 4, 1880.

<table>
<thead>
<tr>
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<th>Case I</th>
<th>Case II</th>
<th>Case III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quantity in 24 hours</td>
<td>1230 c.c.</td>
<td>1980 c.c.</td>
<td>1000 c.c.</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1016</td>
<td>1018</td>
<td>1921</td>
</tr>
<tr>
<td>Urea</td>
<td>20 grammes</td>
<td>22.5</td>
<td>27</td>
</tr>
</tbody>
</table>

Examination for color, reaction, phosphates, etc., gave practically the same results as February 3d.

Diet: Breakfast, eggs, bread and butter; dinner, farina, bread and butter, corn-starch, milk; supper, arrowroot, and milk.

On February 3d and May 4th, 1880, an investigation of the urine in our cases was made by Dr. D. W. Jeffries, assistant superintendent to the Institution for Feeble-Minded Children. The patients, while under observation, were kept in a warm room, under the constant supervision of a watchful attendant, so that the whole quantity of urine passed in twenty-four hours was carefully collected.