most edge and all the coats of the intestine, one needle being passed through on one side of the mesentery and the other on the opposite side, the other thread is passed through the ring and intestine in a similar manner on each side of the convex surface of the intestine. The needles are next passed through the serous and muscular coats of the large intestine at each end of a longitudinal incision which has been made in the colon of sufficient length to allow of the passage of the small intestine, which is next pushed into the colon through it. The two pairs of threads are next firmly tied, not too tightly, and a couple of Lembert sutures passed on each side to unite the lateral portion of peritoneum of the colon with that of the small intestine. The results, as shown from a drawing from a specimen, are very perfect (Fig. 13).

Fig. 13.—Ileo-colostomy completed.

The same operation may be performed when the disease attacks any portion of the colon or upper part of the sigmoid flexure, as it is quite as easy to implant a portion of small intestine in the lower part of the sigmoid flexure as it is to implant it into the transverse colon.

PERNICIOUS ANÆMIA: RECOVERY.¹

By H. HANDFORD, M.D., M.R.C.P.,

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PERNICIOUS anæmia terminates fatally in such a large majority of instances that any other result is apt to excite a suspicion as to the correctness of the diagnosis. Nevertheless, a few cases of recovery have been recorded by good observers, and at present we know of no reason why recovery should be impossible. Possibly in the following case an attack of influenza started the process. But I think it may be of advantage to study, in connection with pernicious anæmia, such cases of purpura hæmorrhagica as the one of which I give a few details at the end of this paper, where a blood poison is evidently very actively at work.

The following case presented all the symptoms of pernicious anæmia to a most marked degree, and at one time the patient was in imminent danger of death. He has now been gaining his living as a hawker for the past eight months, and may fairly be said to have recovered. Whether he will relapse, as other cases have done, and die of the disease in the end remains for the future to determine.

J. H., aged 17, a farm labourer, was admitted into the Nottingham General Hospital on April 23rd, 1890, complaining of vomiting, extreme weakness, and frequent painful micturition. He had a light complexion with fair hair, and had always been pale. He had had no previous illness and had a good family history. His weight was 7 stone 5½ lbs. He lived in an elevated district, on a light sandy soil, where malaria is unknown. He dated his illness from what appeared to have been an attack of influenza in January, 1890. Since that time he had been frequently sick after food, but had never vomited blood. He had a fair appetite, no pain at the stomach, and had not lost much weight. For some weeks he had had pain over the bladder and frequency of micturition,

but this very soon ceased after he came into the hospital, and no cause for it was discovered except the irritating qualities of the urine.

On admission, he was extremely pale and very feeble. The skin was moist, unduly transparent, and lemon coloured. The sclerotics had a distinct yellow tinge. (His illness had been diagnosed as jaundice.) The spleen could be felt 2 inches below the ribs, and it afterwards became larger and harder, and the notch could be distinctly felt. The liver was enlarged, the dulness extending 4½ inches downwards from the fifth rib to just below the costal margin. This is of some significance taken in connection with the well known accumulation of iron in the liver in pernicious anæmia. The heart's impulse was feeble and diffused and extended to the left as far as the nipple line in the fourth space. There was a systolic murmur at the apex and in the pulmonary area. There was also a rough superficial systolic murmur audible over the sternum between the two second costal cartilages. It was thought to be pericardial. The urine was always very high coloured—like brown sherry—generally of low specific gravity (1012 to 1016), and contained neither albumen nor bile, but always deposited a large quantity of bright-coloured urates (? purpurine).

On examining the blood, even to the naked eye, a drop appeared pale and watery. The coloured corpuscles were pale, and some of them almost colourless; they varied greatly in size, a few reaching $\frac{2}{3000}$ inch, and many being below the normal average. In a considerable number of cells the hæmoglobin was collected into one or more masses, and in some was in the process of being extruded, producing a budding appearance. The liquor sanguinis was distinctly coloured. The white corpuscles were increased in number relatively, being about 1 to 20 coloured; but as the coloured were diminished down to about 4 per cent. of the normal, the actual number of leucocytes was only equal to the average. The enumeration of the coloured corpuscles on three occasions during the first ten days gave per cubic millimetre 164,000, 213,570, and 154,655 instead of 5,000,000, or an average of 4 per cent. nearly. The hæmoglobin was 10 per cent. of the normal. Thus the corpuscular richness was diminished much in excess of the loss of hæmoglobin, so that, as is stated to be the rule in pernicious anæmia, the hæmoglobin value of each corpuscle, according to the usual calculation, had increased two and a half to three times. I doubt if this supposed increase in the hæmoglobin value of each corpuscle really exists, but believe the true explanation to be that much hæmoglobin has become extravasated into the serum, partly no doubt from the existing corpuscles, but largely from those already destroyed, and that this hæmoglobin in the serum is included in the calculation. By May 14th the hæmoglobin had risen to 35 per cent. and the corpuscles to 12 per cent.; by May 30th to 40 per cent. and 14 per cent.; by July 14th to 60 per cent. and 23 per cent.; by August 4th to 55 per cent. and 26 per cent.; by September 28th to 50 per cent. and 46 per cent. At this period (September 28th) the hæmoglobin corresponded with the corpuscular richness. The lemon-coloured, jaundiced appearance of the skin had disappeared, and also, as seen under the microscope, the blood serum had lost its yellow tinge. The spleen had diminished in size so that it could no longer be felt; and, in fact, the case had become one of simple anæmia.

During his illness the patient had two periods of high temperature, each lasting rather more than a fortnight, and with just one week's apyretic interval between them. The course of the temperature was irregular, with an average of 101° F., and an upper extreme of 103.2°. After May 29th the temperature never rose above 100.5°. Three days after admission the retina was examined, and found pale but otherwise normal. A fortnight later there were numerous hæmorrhages; six were counted in the left eye and eight in the right. Three weeks later they had become absorbed, leaving little trace. Vomiting was a very troublesome and urgent symptom, which continued more or less for many weeks. For the first fortnight he vomited from one to seven times daily. The rejected matter consisted sometimes of food and sometimes of greenish watery fluid. He had no epigastric pain, and the vomiting had no definite relation in time to the taking of food. For rather more than the first fortnight he was generally delirious at night.

¹ Read before the Midland Branch of the British Medical Association.

The treatment was mainly by arsenic, in doses varying from 5 minims of the liquor arsenicalis three times a day up to (on May 10th) 15 minims four times a day; or 1 drachm in the twenty-four hours. On May 1st, a few grains of the ammonio-citrate of iron were added to each dose. The irritable condition of the stomach prevented arsenic being taken in as large doses as would otherwise have been desirable. Small doses of morphine were for a time combined with the arsenic to facilitate tolerance. The vomiting was most urgent before the arsenic was commenced, and could not, therefore, be due to it. During the course of the treatment, a patch of herpes developed below the left clavicle, and there was eventually well-marked arsenical pigmentation. When made an out-patient on September 29th, 1890, he had gained 9 lbs. in weight.

Forming a contrast by its rapid course, but in other respects presenting a strong resemblance, the following case of purpura hæmorrhagica is worthy of record: A boy, aged 10, who had always been rather delicate, was taken ill on March 13th with headache, feverishness, and vomiting. He was seen by Dr. Travell on March 16th, and found to be suffering from a severe attack of purpura. On the afternoon of March 17th, as his condition seemed grave, I was asked to see him in consultation. He was very pale, had a large hæmorrhage into the lower lip, and was very apathetic. On the legs and the lower part of the trunk were numerous purpuric spots. The temperature was 101.2°, pulse 140, and he suffered much pain at the epigastrium, worse after food. In the past twelve hours there had been very profuse hæmaturia, and he also had melæna. There was distinct optic neuritis in the right eye, and some old choroiditis with pigmentation in the left. Seven-grain doses of gallic acid every six hours stopped the hæmaturia within two days. Before the hæmaturia came on he had been treated by 5-minim doses of turpentine, but had only taken 20 to 30 minims in all. Afterwards liquor arsenicalis was given in doses of 5 minims with iron; but in spite of treatment he died on March 21st, the total duration of illness being under nine days.

A CASE OF INFANTILE SPASTIC DIPLEGIA, WITH TREMORS OF THE "DISSEMINATED SCLEROSIS" TYPE.¹

By T. C. RAILTON, M.D. LOND., M.R.C.P.,

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A. T., aged 9, was admitted into the Manchester Clinical Hospital in February, 1891. The parents are said to be healthy, although there is an account of five dead-born children out of a family of thirteen, the reason alleged being that the mother always bore big children, and was delivered with difficulty. There is, however, no appearance of congenital syphilis about the patient, nor about another of the children who was brought to the hospital for examination, nor does the early history of the patient point in that direction. She was born after a prolonged labour with breech presentation, but did not show any symptoms of asphyxia immediately afterwards. She was brought up at the breast, and cut her teeth early. It was soon remarked that she was unnaturally quiet, that she did not toss her limbs about like other infants at the same age, and she did not begin to "notice" until she was about 2 years old. It was also observed that her head seemed disproportionately large and that when she was lifted up it drooped as though too heavy for her. A medical man, consulted on the subject, said, however, that it was not "water on the brain." There has never been any attempt at walking; in fact, she can barely stand now, and only for a very short time when holding herself up with her hands clinging to the rail at the foot of her bed. When 2 years old she had a fit, and a year later two more attacks in the same day, but has had none since. A few months before the first fit her hands were noticed to shake when she used them.

At the present time her appearance is peculiar, and rather gives an impression of imbecility. She has an extremely

large head (56½ centimetres in circumference), globular in shape (antero-posterior diameter, from root of the nose to occiput, 19 centimetres; transverse diameter, 17 centimetres). Both size and shape are suggestive of hydrocephalus, but no other evidence of that disease is now perceptible. The forehead, though normal according to age, is relatively narrow. There is internal strabismus which has existed from birth, but neither paralysis nor spasm of any of the muscles of the face. The fundi of the eyes are normal. There are no tremors of the head, tongue, nor lips; no nystagmus, no scanning nor drawing speech, and no vertigo. In intelligence she is backward, and can neither read nor spell, though able to answer simple questions sensibly enough, after the fashion of a child much younger. Part of her mental deficiency may be due to want of education, which has been entirely neglected, and this also may perhaps account to some extent for the uncontrollable temper she occasionally exhibits.

All the superficial reflexes are present and active. Her chest and abdomen are free from disease, and her limbs are well nourished, firm, and symmetrical, the muscles reacting normally to electricity. There are no signs of her having suffered from rickets. She is able to sit up without support. In the arms there is no rigidity perceptible on passive movement. When she raises them voluntarily they show a fine tremor, which persists so long as they remain unsupported, and when she attempts any action requiring precision the movement changes to a coarse oscillation, which gradually becomes extreme as she approaches attainment, in exactly the same manner as the tremor of disseminated sclerosis. Thus, she cannot convey a glass to her mouth without spilling its contents, and cannot feed herself. Her difficulties are increased by the fact that, in addition to the clonic spasms, there is considerable awkwardness (inco-ordination) in the way she grasps objects. The movements cease entirely as soon as the arms are at rest.

As is usual in birth palsies, the brunt of the mischief has fallen upon the lower extremities. As she lies down, these remain in a condition of nearly complete extension, with the exception of the left leg, which is slightly contracted at the knee. When raised from the bed the feet have a fine tremor. On passive moment there is distinct resistance. The knee-jerks are exaggerated, and ankle clonus is readily obtainable. When supported under the arms the patient is able to bring one foot alternately in front of the other, with the toes dragging on the ground, and during this exercise she complains that her feet "shake." There is well-marked rigidity of the legs when in action, but little or no spasm of the adductors of the thighs. When she attempts to stand, holding on to some support, her legs are habitually bent at the knees.

She is extremely sensitive to touch; the slightest prick of a pin anywhere about the limbs or body produces a universal involuntary twitching, extending even to the muscles of the face.

This case is interesting from its similarity to one of sclerosis in patches. The tremors, paresis of legs with contracture at the left knee, the retention of normal electrical reactions, the exaggerated knee-jerks, the ankle clonus, and finally the mental deficiency, all combine to impart a superficial resemblance to that disease which might well prove misleading at first. The distinction between the two diseases, however, becomes comparatively easy upon closer investigation. In the first place the history of the case very clearly points to an injury to the cerebral cortex from meningeal hæmorrhage at the time of birth, rather than to the gradual development of a progressive disease, the patient, one might almost say, having remained *in statu quo* from birth. This conclusion is further supported by the absence of any peculiarity in the speech (a drawing, "die-away" voice being an almost invariable concomitant of disseminated sclerosis), by the total absence of tremors of the head or vertigo, and still more by the presence of the remarkable sensitiveness of the skin to stimuli, which is not observed, so far as I am aware, in connection with sclerosis in patches, but has been previously reported in certain cases of infantile spastic paralysis. As regards the presence or absence of nystagmus—absent in this case—as it is equally met with in both diseases; its presence here would not have been of assistance in forming a diagnosis.

It seems very possible that there may have been some hy-

¹ Read before the Manchester Medical Society.