mode of treatment in severe cases of this very troublesome complaint.

Although in its mild forms, and when of short standing, sciatica easily yields to constitutional and local treatment of various kinds, in its severer varieties it is one of the most intractable maladies with which we have to do, trying the patience alike of physician and patient. Every now and then a patient, most frequently a male, presents himself suffering from this most painful complaint which has incapacitated him from work for a longer or shorter period and for which he has generally tried many forms of treatment, both regular and irregular. If the patient has to earn his living he generally tries to go on as long as possible in spite of pain or with only partial relief from remedies. I believe it is this neglect in the early stages that makes the disease in certain cases so intractable and difficult of cure. So long as there is any pain the complaint is not cured and is liable to relapse from a mild into a severe form. It was whilst treating a severe and long-continued case in which many measures had been tried without benefit that I first employed Dr. Weir-Mitchell's plan of treatment and this was completely successful. Since then I have carried it out in all severe cases of sciatica that have come under my care. For mild cases and for those that come under treatment just after the onset it is hardly necessary, though in the latter if the pain is very acute it is effectual in relieving the symptoms and it does so quickly.

The details of the treatment will be found in a most instructive paper on sciatica in Dr. Weir-Mitchell's *Clinical Lessons on Nervous Disease*,¹ where he emphasises as the essential point that the splint must check motion at the hip and the knee. The leg is first carefully and evenly bandaged with a firm flannel bandage from the hip to the knee. A long splint is then applied, the knee being in slight flexion, and bandaged on with flannel bandages and secured round the trunk by two or three broad bands. The heel must, of course, be carefully supported. In my own practice the limb is left untouched for the first three days, or if it is uncomfortable the bandages are taken off and reapplied with as little disturbance as possible. In any case this is done once a day after five days, and after five or six days gentle passive movement of the joints is made at the same time. When the patient has been free from pain for some days, generally at the end of two or three weeks, or longer in a severe case of long standing, the splint is left off by day and reapplied at night. Then the splint is discontinued altogether, the leg being still kept bandaged, and after another week or less the patient is allowed to get up. I have followed Dr. Weir-Mitchell in the above stages of treatment and in taking the absence of pain as the indication to pass from one to the other, and also have followed his valuable caution that when a patient with sciatica first gets up he should stand or lie but not be allowed to sit. When the splint is first left off by day massage of the limb may generally be begun and as in these severe cases the muscles are flabby and wasted it is of great advantage in hastening the restoration of the use of the limb.

I may briefly mention, without going into details, the result of the last 12 consecutive cases of severe sciatica treated in hospital in this way. 11 of the patients were men, their average age being 49 years. The longest time the disease had lasted was 21 months and the shortest time was seven weeks. The average duration before coming into hospital was 23.6 weeks, the average duration of treatment in hospital was five and a half weeks, and the average time from admission until the splint was left off entirely (by night as well as by day) was 25 days. As showing that the cases were severe I may point to the duration, the facts that from the great pain caused by movement four of the patients were carried in unable to walk at all and a fifth could only hobble a few steps with help, that in ten the muscular wasting was marked, and in four there was defective sensation on the outer side of the leg.

Ten patients were cured. One had a severe relapse some weeks later but was cured by a second course of treatment. One was not relieved; he was a restless, impatient man and would not submit to keep the splint on. It will be noticed that the patients only stayed a short time in hospital after they were allowed to get up; a few of them attended for a short time afterwards as out-patients in order to go on with the massage. It seems to me that the result in these cases as to cure and as to the duration of stay in hospital—the latter

an important consideration both to the hospital and to working-men patients—is satisfactory.

In patients of the well-to-do classes I have had equally good results. A cure was effected in one man who had suffered from sciatica almost continuously for ten years and he has remained free from pain and able to walk about since the treatment four years ago. In his case the time under treatment was longer than in those given above.

The restraint of the splint is trying to nearly all the patients and extremely irksome to some and the pain may be considerable for the first 24 to 48 hours. With some patients considerable persuasion is required during the first two or three days to induce them to go on with the treatment. A hypodermic injection of morphia is often necessary on the first night and sometimes on the second, but not, in my experience, afterwards. By the third to the fifth day there is great relief and the patients begin to sleep well; indeed, in favourable cases the acute pain is not felt after the third day. If the pain persists it often does so behind the outer malleolus of the ankle or in the foot. This sometimes means that a readjustment of the support is required.

In the more neurotic patients who come into the hospital and in private patients one of the chief difficulties is at first flatulent dyspepsia. Many sufferers from sciatica have already been troubled with this before they come under treatment and it is then aggravated by the enforced rest. Very careful feeding and a light diet are therefore better in all cases at first and afterwards the latter can be gradually improved, for as liberal a diet as the patient can digest is generally advantageous. Lastly, care must be taken that the bed is flat and even and that there is a firm basis of support under a flat mattress. The limb must be absolutely at rest in its whole length and failure to secure this will prevent a successful result. A short course of massage, tonics, and cod-liver oil are advantageous to complete the cure.

I think I have given a fair account of my experience of this method of treatment, and if I have seemed unduly to emphasise small details it is because they are essential, as in many other matters, to success.

Clifton.

A CASE OF INFANTILE ACUTE ECZEMA; HÆMAT- EMESIS; DUODENAL ULCER; DEATH.

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I DESIRE to put this case on record on account of the unique character of the lesion and the fact that I have been unable to find in the literature of the subject a recorded case of duodenal ulcer following eczema, which I believe in the present case are related to each other and analogous to the cases of duodenal ulcer following severe burns.

The patient was a baby, aged eight months. At birth he was well nourished and fully developed. He was breast-fed till seven months old. When about two months old he began to suffer from eczema capitis which persisted more or less during the latter months of life. Vaccination was performed at the fifth month; this ran a normal course and had but little influence on the eczema. About the sixth month the face became eczematous with considerable itching. The left submaxillary gland became swollen and the skin became inflamed as if suppuration was imminent. The swelling, however, subsided by resolution. When seven months old the child was weaned, as the mother at that time was suffering from acute rheumatism. The artificial diet—Mellin's food and milk—on which he was put did not do so well as the mother's milk. On April 2nd, 1903, when eight months old there was an exacerbation of the eczema which took on a virulent form, affecting the head, the face, the neck, and to a slight extent the trunk. From this time onwards the child seemed to go back in every way; soothing applications failed to arrest the intensity of the affection. The patient, previously happy and contented, became peevish and fretful. On the 3rd I saw him and found that the state of the head and face was becoming rapidly worse, but the

¹ Lea Brothers and Co., Philadelphia and New York, 1897.

eruption over the trunk had by this time faded entirely. The temperature was subnormal; the pulse was small and 140 per minute; the respirations were 30; and the feet and hands were cold and pallid. The face and head for the first time now assumed a livid colour. The eyes could not be kept open owing to the oedematous swelling of the eyelids which had supervened. I found on cleaning away purulent exudation from the closed eyes that the skin was abraded with the gentlest swabbing. On the 5th I called in the morning and found the child asleep in his father's arms; the discharge from the head had exuded copiously on to the father's shirt-sleeves. When I removed the shawl which was adhering to the head of the little patient I found that the scalp was simply reeking with vapour. I observed further that the character of the eruption had undergone a change: the vesicles or pustules were now enlarged to the size of a split pea; these were purulent, raised, discrete, and impetiginoid. Besides the mass over the scalp and brow there was an extension of the pustules to the cheeks, the chin, and to a less degree to the neck, but there was still no eruption on the trunk. Towards evening the child rapidly became worse and at 3 A.M. on the next morning he vomited some blood and from this time the motions were tarry. He drew up his little legs continually and often passed his hands down on the abdomen as if in pain. There were considerable vomiting and retching. On the 6th the patient had a restless day and at midnight he seemed to be sick and vomited milk on two occasions. The bowels were moved just about the same time; the motions were again tarry. He then vomited "some dark-like material." The child's hands were clenched, he lay much distressed, and the feet were cold. On the 7th, at 1 A.M., he vomited some blood which was red in colour. In order to relieve the retching a poultice was placed over the stomach and sips of iced water were given by the mouth. He settled down and was quiet thereafter for several hours. During the day, however, retching of a severe nature returned. He became very restless; at 5.30 P.M. he was looking very ill; the head and face became bluish black. His strength was slowly slipping away and a gurgling sound was occasionally heard. A few sips of whisky and iced water had been given to him during the day. At 10 P.M. he vomited a single mouthful of bright-red blood. There was no special retching before this last and fatal vomit, but subsequently he spat a little and lay down quietly for some time apparently in a collapsed condition. When raised up at any time he cried as if in pain and at times placed his left hand over the left side of the abdomen. He was laid down on his right side about 1 A.M. and at 2.30 A.M. a movement was heard of something passing down the bowels. He gave a sharp cry of pain, after which he became quiet, but it was noted that the eyes rolled about and twitching of the mouth ensued. He died at 3 A.M. on the 8th.

I have to record my thanks to Dr. John Anderson, pathologist to the Victoria Infirmary, Glasgow, for making the post-mortem examination for me and I append a summary of his report. From external appearances the patient was a well-developed and well-nourished child. Rigor mortis was pronounced. The pupils were equal and of medium size. Eczema of the face and head was present. As to the thorax, the pericardium was healthy. The heart was of normal size and appearance, the chambers presenting healthy characters. A small quantity of clot was found in the right ventricle. All the valves presented normal and healthy appearances. There was a tiny patency of the foramen ovale. The heart muscle was of good colour and consistence. The lungs were non-adherent and apart from a slight degree of congestion of the bases presented remarkably healthy appearances. With regard to the abdomen a few ounces of blood-stained fluid were found in the general peritoneal cavity. No evidence of peritonitis could be found. In the lesser sac of the peritoneum was found a large-sized piece of clot filling practically the entire space. The posterior wall of the stomach and surface of the pancreas on which it rested showed presence of fibrinous exudation. A round ulcer situated on the posterior wall of the duodenum just beyond the pylorus was made out. The ulcer had perforated and the perforation was of about the size of a split pea with punched-out edges and directed towards the lesser sac of the peritoneum (see figure). The stomach contents were chiefly of the nature of altered blood. The stomach presented healthy appearances; the mucous coat was rugose and slightly congested. The intestine was examined throughout and apart from blood-staining of the contents showed healthy appearances. The

liver was of normal size and slightly cloudy in appearance. The spleen was healthy. The kidneys and adrenals were healthy in appearance.

Illustration showing the stomach and duodenum of the child. The place where the perforation occurred is indicated by the wooden probe *in situ*.

The skin of a child is relatively greater in area than that of an adult in its ratio to the body weight, hence a cutaneous lesion predisposes infants to constitutional disturbances of greater intensity and may be expected to tell more severely upon them. This may be due to impairment of the function of the skin, to nerve irritation, to destruction of red corpuscles or leucocytes, or to absorption of toxins from the disintegration of the blood corpuscles. I am disposed to regard the occurrence of the duodenal perforation in this case of eczema as due to the same contributory causes which we find to obtain in some cases of burns. In both there is a like destruction of the function of the skin and the soluble fibrin ferment in both cases readily gaining entrance to the circulation will conduce to the formation of emboli which results in a partial necrosis of portions of the duodenal mucous membrane.

Cases of duodenal ulcers have been recorded after burns as already mentioned, after frost-bite, and in erysipelas, pemphigus, and septicæmia. In those due to infective processes bacterial influences obviously play an important part.

The etiology of duodenal ulcer and its relationship to cutaneous burns.—As bearing upon the above case it is interesting to consider the remarkable coincidence of burns of the skin with ulcers of the stomach and duodenum in young subjects first observed by Curling¹ who was able to collect no less than ten cases; since then, according to Dreschfeld,² the subject has been investigated by several observers, especially in England, with the result that though the occasional appearance of an ulcer after an extensive burn has now been well established the occurrence is certainly very rare. In 125 cases of severe burns Holmes³ found the duodenum ulcerated in 16 and other portions of the intestine in two. The earliest appearance of the ulcer was from four to six days after the burn. Perry and Shaw⁴ noticed it five times in 149 necropsies of cases of burns. It is further established that these ulcerations occur chiefly in young subjects and more especially when there are extensive burns of the trunk; that the ulcer occurs in the second or inflammatory period of the burn; and that before ulceration occurs there is marked congestion followed by hæmorrhagic infiltration which affects

¹ Curling: On Acute Ulceration of the Duodenum, Transactions of the Royal Medical and Chirurgical Society, vol. xxv., p. 260.

² Dreschfeld: Allbutt's System of Medicine, vol. iii., p. 548.

³ Holmes: System of Surgery, vol. i., p. 733.

⁴ Perry and Shaw: Guy's Hospital Reports, p. 171, 1894.

other parts of the alimentary tract besides the duodenum. In several cases the lesion did not go beyond a hæmorrhagic infiltration; in other cases, an ulcer forms which may lead to a perforation or to profuse hæmatemesis. The formation of the hæmorrhagic infiltration is most rationally explained by a septic embolus and the change into an ulcer by the action of the gastric juice. The nature of the ulcer is similar in character to the gastric ulcer, round or oval, punched-out appearance, with little inflammation of the periphery, and microscopically shows the same condition—more of a necrosis than inflammatory round cell or new cell formation. The ulcer is situated in the first part of the duodenum and above the bile papilla and is therefore classifiable with the gastric ulcer. It falls, then, under the same group of causes. It may arise from mechanical, chemical, thermal, &c., agencies; bacterial necrosis has been said to play an important part, while in the case of burns the destruction and emboli formation of red corpuscles have been assigned an important place.

Micro-organisms have been brought forward as the cause of gastric ulcer. Letulle⁵ found numerous streptococci in the veins of the submucosa and of the uterus in a case of recent ulcer of the stomach which appeared during the course of puerperal septicæmia. Pure cultures of these injected into guinea-pigs also caused ulcerations in the stomachs of the animals and these threatened to perforate the walls of the distended organ. Letulle obtained the same result in four cases with staphylococcus pyogenes aureus cultivated from various abscesses and in one case with the microbes of dysentery. Experiments by Ponfick and others have shown that in animals extensive scalds will cause a destruction of red blood corpuscles similar to that seen on injecting toluyldiamin. Dr. W. Hunter succeeded in producing extensive ecchymoses and ulcers of the duodenum in dogs by injecting toluyldiamin and suggested, as a probable explanation of the duodenal ulcer in burns, the secretion, with the bile, of a poison which causes the ecchymoses and ulceration. Virchow's view that the process may result from plugging the nutrient artery of the part, either by an embolus or by a thrombus, and that the infarct so produced is destroyed by the gastric juice, has gained general acceptance. There is obviously a local disturbed nutrition over a circumscribed area. The corrosive action of normal gastric juice plays an essential rôle in attacking the spot deprived of its vascular supply just as under favourable conditions it leads to softening and digestion of the dead tissue of the stomach. If the gastric juice meanwhile has become hyperacid and cannot be neutralised till it meets with the pancreatic secretion, this will readily explain why round ulcers are so prone to occur in the duodenum. Ebstein,⁶ making use of a discovery of Schiff, produced gastric hæmorrhages and corroding ulcers, and even perforation, by injury to the anterior corpora quadrigemina and it has been assumed that the excessive production of acid secretion in these cases was due to cerebral irritation.

The relationship of the eczema to the lesion.—It was noticed in the last three days of the child's life that the eczema was characterised by a virulent exacerbation—that the septic condition of the skin had become intensified, enabling the flora of the skin to assume fresh activity from the increased pabulum. The lesion had merged into eczema pustulosum—eczema impetiginoides due to secondary inoculation of the inflamed skin by pus cocci; this is to be distinguished from impetigo contagiosa by the fact that the pustules in the latter disease occur on healthy skin and not on an already inflamed surface. When the eczema of the child had passed into the acute pustular form it is a reasonable theory to suppose that septic absorption supervened from bacterial toxins and that these, acting as a source of irritation on the central nervous system, produced greater acid secretion in the stomach and duodenum. The cutaneous lesion of this nature brings about an alteration in, and destruction of, the blood corpuscles through the superficial inflammation of the skin, followed by necrosis of the superficial layers of the epidermis and the prolonged secretion of serum and pus.

Treatment.—Surgical intervention was in this case beyond consideration after the diagnosis was assured. Had the case not presented such grave features surgical interference would

have been worthy of suggestion and adoption in view of the absolutely hopeless prognosis of ulcer of the duodenum after perforation, although it must be conceded that the results of such operations offer nothing to boast of.

Glasgow.

THREE CASES OF PERFORATED GASTRIC ULCER; RECOVERY AFTER OPERATION.

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CASE 1.—A single woman, aged 34 years, was admitted into the Metropolitan Hospital on Jan. 1st, 1902, at 9 P.M. The patient had been seized with sudden pain in the epigastrium at 9 A.M. on the same day and was much collapsed. She was seen by Mr. Reginald Brown who gave her morphia: she was also seen by a surgeon in the afternoon. Towards evening the pain returned, the abdomen became rigid, and the pulse rate increased. There was a past history of anæmia and indigestion for years but there had never been any hæmatemesis. When seen at the hospital she was not in great distress but was evidently well under the influence of morphia. The abdomen was not moving at all and on the upper and left side where the pain was complained of the muscles were very rigid. Resonance was unaltered all over; the liver dulness was normal. No dulness was present in the flanks.

Operation was performed at 11.30 P.M. on the day of admission. Chloroform was the anæsthetic used. An incision was made in the mid-line above the umbilicus. There was no sign of general peritonitis or free gas on first opening the abdomen but on reflecting the omentum to expose the posterior surface of the stomach there was a small escape of gas. A perforation was found in a small lymph-covered area on the posterior wall, near the cardiac end of the lesser curvature, about two millimetres in diameter. There was very little escape of stomach contents. The perforation was sewn up by invagination with Lembert's sutures. Any lymph and fluid were sponged off the neighbouring peritoneum and no fluid was used in the abdomen. The abdomen was closed and no drain was employed.

The patient made an uninterrupted recovery. She sat up in bed on the second day, was on the couch the middle of the third week, and left the hospital at the end of six weeks in better health than she had been for years. She was seen in September, 1902, and in July, 1903, when she was as well as could be wished. The following was the diet used while in hospital. Rectal feeding was begun on the morning after the operation with three and a half ounces of peptonised milk every four hours and one yolk of egg. Nothing was given by the mouth until the fifth day after the operation when small quantities of water were allowed. On the next day one drachm of whey was given every hour; occasionally the same amount of beef-tea was given instead. This quantity was gradually increased until at the end of three days four drachms were given every hour. Very gradually the amount given by the mouth was increased and the rectal feeding was discontinued. On the nineteenth day after operation baked custard was added to the diet and the enemata were discontinued entirely. On the twenty-third day a small amount of boiled sole was given. On the twenty-sixth day the note reads: "Diet as follows with slight variations daily: Breakfast, 6 A.M., milk, thin bread-and-butter; luncheon, 9 A.M., milk or beef-tea and jelly; dinner, 12 noon, boiled sole, bread-and-butter, custard with milk or water; tea, 3 P.M., milk, bread-and-butter, sponge cake; and supper, 7 P.M., beef-tea or milk and custard or jelly. Total, one pint of milk and half a pint of beef-tea in the 24 hours." On the twenty-ninth day two lightly boiled eggs were added and a week later minced chicken was given. On Feb. 14th the patient went home taking ordinary diet except meat.

CASE 2.—The patient, a woman, aged 21 years, was admitted to hospital on Jan. 24th, 1902. She had felt sudden pain in the epigastrium on Jan. 21st; this had been constant ever since but it recurred more violently on the morning of the day of admission. On this day she vomited three times.

⁵ Letulle: Origine infectieuse de certains Ulcères simples de l'Estomac ou du Duodenum, Comptes Rendus, tome cvi., No. 25.

⁶ Ebstein: Experimentelle Untersuchungen über das Zustandekommen der Blutextravasate in der Magenschleimhaut, Archiv für Experimentelle Pathologie, Band ii., S. 183.