feeling like malignant disease, but which proved to be the surface of the pancreas exposed by ulceration (see photograph). After passing the finger well through the pylorus, I decided to close the stomach wound. This was done by a fine silk Lembert's suture with a reinforcing row outside the first.

Convalescence was uncomplicated by any sickness, rise of temperature, or any other complication. The patient was fed for the first three days on nutrient enemata, then on peptonised food, and took solid food on the tenth day. She was a little sick (8 ounces) on the thirteenth day, but got up on the fifteenth day, and gradually improved. She was sick twice only in December, and was discharged on January 1st, her weight at that date being 6 st. 5 lbs., or a gain of 9 lbs. since the operation. She still suffered at times, however, from epigastric pain. After her discharge from the hospital she continued well for three or four months, though there was occasional and slight sickness, and she walked out of doors. She then, as she expressed it, began to suffer from the scanty supply of poor and indigestible food, and the pain and vomiting gradually returned, and she died from exhaustion after an attack of diarrhoea on September 20th, eleven months after operation.

Cavity of stomach laid open, showing the projecting ring of the pylorus, A, A, and the ulcerated surfaces. A, pylorus; B, ulcerated areas.

Post-mortem Examination.—This showed an absence of disease in any organ except the stomach. This organ was much dilated, and lay almost entirely to the left of the mid line; stretching from the cicatrix on the abdominal wall to the anterior surface of the stomach was a thin old adhesion. The pylorus was much thickened, and adherent to the under surface of the liver, and to the pancreas posteriorly. On opening the stomach, no sign of the incision in its walls could be seen, and, apart from the dilatation, the cardiac end appeared healthy; the ring of the pylorus was found as described at the operation, smooth, but tough and inexpandible, but easily admitting the forefinger, and projecting like the lips of the os uteri into the stomach cavity. The greater portion of the wall of the stomach in its immediate neighbourhood was ulcerated away, exposing the pancreas posteriorly and the liver in front, to both of which the stomach is very firmly adherent all round, so that there has been no extravasation.

REMARKS.—There can be no doubt, I think, that the roughened surfaces felt at the operation and suggestive of malignant disease were the ulcerated areas in which the rough surface of the pancreas could be felt; in fact, the stomach presented a threefold lesion (1) a hardening and loss of expansibility with some contraction

of the pylorus, with firm adhesions to neighbouring parts; (2) extensive ulceration of the wall of the stomach of a non-malignant kind in the neighbourhood of the pylorus; (3) great dilatation of the general stomach cavity.

Now it appears to me that these three conditions have a causal relationship to each other, and I would place the ulceration first, this leading to adhesions and then to chronic spasm, and eventual rigidity of the pylorus, this leading in its turn to the dilatation of the stomach; in fact, the condition of the pylorus may be compared to the chronic spasm of the sphincter and when irritated by an ulcer of the mucous membrane just within the rectum. Moreover, it seems likely that beyond mere constriction of the pylorus in cases which have been treated by Loreta's method, ulceration in its neighbourhood has an important share, for in the history of chronic illness which such patients give, a vicious circle has been established, the ulceration leading to constriction, and the pyloric constriction again preventing the healing of the ulceration. It is not easy to understand how real pyloric stricture comes about in the absence of any cause of irritation, such as the presence of an ulcerated surface, and the conditions found in this case, and verified by *post-mortem* examination, especially the degree and stages to which the different lesions had respectively arrived strongly support, I think, this view. If this be so, the probable presence of ulceration, either recent or healed, should lead to care in extreme dilatation of the pylorus in cases of Loreta's operation.

That great relief was afforded in this case is shown by the decrease in vomiting and pain, and by the increase in weight; it is not so easy to explain how the good result was brought about. Although the finger was passed through the pylorus, no force was used to dilate it. Whether any nerve filaments irritated by the ulceration were divided by the incision in the stomach wall, and so set at rest, or whether the plecting in of the walls of the stomach (which was considerable at the operation) helped to temporarily lessen the dilatation, must be a matter of conjecture. Dr. Ord has suggested that the prevention of apposition of the opposing gastric surfaces in the empty stomach in ulceration is an important cause of pain of an epigastric character, and this may have been present before operation in this case.

I would, however, suggest that moderate digital dilatation of the pylorus should be practised in cases of extensive chronic ulceration of the stomach with dilatation, even where there is not extreme pyloric stenosis, with a view of curing the ulceration by setting the pylorus at rest, and Professor Loreta's operation may thus have beneficial results in a further class of cases.

NOTE ON CHARCOT'S JOINT DISEASE.

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As Charcot's disease is a somewhat rare affection, I have thought that the following case, together with the few remarks suggested by the study of the causation of the deformities which accompany it, may be of interest to the profession.

A. B., aged 45. Patient was suffering from ataxy for twelve years. Illness was produced by a fall from a horse. No hereditary history of nervous disease. During the course of his malady he presented all the characteristic symptoms. The Argyll-Robertson pupil, the lightning pains, and peculiar gait were followed in due course by gastric crises, cystitis, complete loss of reflexes and cutaneous sensibility. Bedsores developed towards the end of his illness, and he died from exhaustion. For some weeks before his death he lost the perception of all but sweet taste, and refused all food which had not been sweetened. This phenomenon might be explained upon the supposition that the hypoglossal nerve was unaffected by the sclerotic changes. Unfortunately, it was impossible to examine the brain and spinal cord, as consent for such examination could not be obtained. The principal feature of interest, however, lay in the well-marked bone lesions which occurred during the later stages of the disease. About four years before the termination of the illness the joints of the feet and hands commenced to show traces of disease. There was no effusion into any of those affected, and but two of the finger-joints showed any inflammatory action; in these pus formed under the flexor tendons covering the joint. The pus was evacuated, the incision causing no pain, and the wounds healed. All the metacarpo-phalangeal joints became subsequently

enlarged and ankylosed. Owing to the bony enlargements, the same changes occurred in the feet, resulting in ankylosis of the tarsal joints. A small perforating ulcer formed at the head of the metatarsal bone of the left great toe. The sinus of this ulcer did not penetrate to the joint. It was successfully treated by paring away the indurated cuticle, thereby exposing the sinus, which was swabbed out with creasote and plugged with iodoform. The destructive process was arrested, and the sinus eventually dried up. In addition to the joints of the hands and feet, that of the left shoulder became affected; in this joint the destructive changes were best marked. Two years before death its movements were first observed to be limited. No pain was ever complained of in the joint, and no effusion ever took place either within or around it; it rapidly and painlessly underwent disorganisation. The bones occupied a position somewhat similar to that observed in a subglenoid dislocation.

Fig. 1, taken from a photograph of the joint with some of the

Fig. 1.

muscles still attached, shows the condition of affairs. The head of the humerus lay in the subscapular fossa, which was deepened for its reception. It was covered by a dense capsule composed of the subscapularis and part of the teres muscles, and strengthened by osseous and cartilaginous tissue. Fig. 2 shows the posterior view

Fig. 2.

of the joint, with a triangular-shaped osteophyte formed in the fascia covering the infraspinatus muscle. Upon opening the joint by a circular incision through the capsule, about two drachms of blood-stained serum escaped. The synovial membrane was thickened and infiltrated with fat; over the sharp bony points it had entirely disappeared. The biceps tendon was completely eroded, its external attachment to the capsular ligament enabling it to assist in flexing the limb. (See Fig. 1.) All the ligaments were greatly relaxed, and I fancy the dislocation was produced in the first instance either by the weight of limb or by mere muscular action. There were a number of fatty and fibro-calcareous nodules attached to the synovial membrane and the bones. Those nodules, with an external fatty covering and a central calcareous nucleus, were the most numerous; several are seen upon the eroded end of the bone. Three large osteophytes are shown in Fig. 3 sur-

Fig. 3.

rounding the head, or rather the upper extremity, of the shaft of the bone. One is evidently the separated head itself; the others are new formations. They are connected by the thickened capsule, out of which they have been partially dissected. The diseased scapula is well shown in Fig. 4. There are several bony

Fig. 4.

masses developed in connection with its processes. One is seen springing from the tip of the coracoid process; this measures two inches in length, and had the short tendon of biceps inserted into it. Another new formation springs from the bone beneath the glenoid cavity, which also measures two inches, and had the long tendon of triceps attached. The nutritive foramina in the spine were enormously enlarged, and so increased in number that, had the patient lived, the process would have become detached from the body of the bone. The nutritive vessels were very small, and lay imbedded in fatty masses, filling up the enlarged nutritive canals. The suprascapular nerve was much softened, and lay in a bony groove in the suprascapular fossa. It expanded in this situation, and its filaments were ultimately lost in the fibrous tissue connecting the osteophytic processes of the joint.

Peripheral nerve degeneration, the result of spinal cord disease, by cutting off the normal trophic influence travelling along the nervous paths, has been looked upon as the cause of these osseous lesions by their first describer. Waller has fairly proved that the trophic centre for the sensory root of cerebro-spinal nerves is not situated within the cord itself, as in the case of the anterior nerves, but within the ganglia placed upon the posterior nerve roots. Degeneration of these ganglia and their contained trophic centre is presumably more likely to occur in posterior sclerosis of the cord than in other spinal diseases. Now, if we believe that the normal trophic influence travels by a sensory rather than by a motor path, some explanation of the comparatively frequent occurrence of osseous lesions in ataxy may be

obtained. Nasse has found that section of nerves supplying bones has caused a diminution of all the osseous constituents with an increase of fat. These changes are similar to those occurring in the case here described, with the exception of the formation of the osteophytic processes. These formations are, of course, mainly the separated bony processes of the joints, but some are entirely new productions (see Fig. 2).

I have thought that in cases of nervous degenerative disease the normal phosphatic excretions are increased in quantity, and are prevented from leaving the system by the cystic disease so commonly accompanying these affections. These phosphates may consequently be reabsorbed from the urinary organs, and deposited as phosphates of lime or magnesia in the fatty, softened, and degenerating tissues affected by trophic disturbances. This explanation is, however, unsatisfactory when applied to those cases in which the osseous lesions occur before the vesical complications. In these cases there appears to be less bony formation than in those cases where Charcot's disease appears as a late phenomenon, the bones being absorbed and softened without the formation of osteophytes.

CHLORALAMIDE AS A HYPNOTIC.

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In his exhaustive account¹ of many of the new hypnotics, Professor Leech says of chloralamide that the observations upon it are so far few in number. I have recently given it to twenty patients suffering from various illnesses, in all of whom insomnia was a troublesome symptom. Brief notes are appended. It will be seen that the drug produced comfortable sleep in all the patients except two; one of these was suffering from delirium connected with cerebral hæmorrhage, and the other was admitted with rheumatic fever complicated by delirium tremens and salicylic poisoning. Both these patients died shortly after admission. It is noteworthy that some of the other patients were suffering from extremely painful diseases, and yet chloralamide produced sleep; thus a woman who had a thoracic aneurysm preferred it to morphine, and another patient who had carcinoma of the stomach also slept better with chloralamide than with morphine. The patient with carcinoma of the liver suffered intense pain, yet she dozed comfortably after chloralamide. The man suffering from cerebral softening who was quieted by the drug is also a striking case. Probably the house-physicians, sisters, and nurses are the best judges of hypnotics, as they see the patients frequently during the night. They all tell me that those who take chloralamide sleep well and comfortably after it, and the sisters of the three wards in which I have used it tell me that the patients sleep better after chloralamide than after any of the hypnotics which have been introduced during the last few years. My own experience, and what the patients themselves tell me certainly agree with this. In none of the twenty patients to whom I have given it—and many of them have taken several doses—have any effects followed that can be looked upon as contra-indications to its use. Never have I observed any depressing results, nor has headache followed its use. The time which elapses between its administration and the commencement of sleep varies between a quarter of an hour and two or three hours. If it is given in the evening, when once asleep the patient usually sleeps quietly till morning. Some writers have stated that occasionally after a dose in the evening the patient does not go to sleep till the next morning, and that he remains asleep all the day. This was so with one of my patients; but it must be remembered that, as the drug is feebly soluble in water—20 grains take five hours to dissolve in 2 ounces of water—it is often given as a powder with some milk. It was administered in this way to my patient who slept the next day, and I should think that some of these cases of delayed action were due to delayed absorption. Now I always prescribe it with spirit; 20 grains will dissolve in 1 drachm of rectified spirit in fifteen minutes, and water may be added to this solution without reprecipitating the drug. A good way of giving it is to tell the patient to dissolve it in a little brandy, add water to his liking, and drink it shortly before going to bed. If given in milk, not only is it insoluble, but it is difficult to swallow, for it sticks to the sides and bottom of the glass. The taste is slightly bitter, but by no means

disagreeable. It should never be prescribed with alkalies, for they decompose it, nor should hot water be mixed with it, for it decomposes at 148° F. For an adult, 20 to 60 grains—the exact amount depending upon the cause of the insomnia—is the dose; usually 30 grains will suffice. It has the advantage over sulphonal that it is only half the price, and it has the great advantage over paraldehyde that it has no nasty smell or taste, nor is it difficult to dissolve.

The few cases which have been published quite bear out the cases recorded here. It would seem that in chloralamide we have a safe hypnotic, which hardly ever has any depressing effects, which does not produce indigestion, and very rarely gives rise to any unpleasant results. We do not of course yet know what harm may result from its prolonged use. References to those authors who have studied the chemistry and physiological action of the drug will be found recorded by Leech, Paterson,² and in a leading article in the *Therapeutic Gazette* for September, 1889. Rabow³ considers 45 grains of chloralamide to be equivalent to 30 grains of chloral. Chloralamide has been used successfully as an enema by Feiper.⁴

CASE I.—Typhoid. A girl aged 4½. Very irritable and fretful, often keeps the other patients awake by her crying; 5 or 10 gr. of chloralamide always sent her to sleep a quarter of an hour after taking it. She slept quietly for many hours. She took it frequently for a fortnight.

CASE II.—Sarcoma of last rib, growing extensively into the tissues and organs around. A middle-aged man. He suffered intense pain, but 30 or 40 gr. of chloralamide always gave him sleep, often for the whole night. It relieved him as much as, or even more than, morphine.

CASE III.—Cerebral hæmorrhage with noisy delirium. An adult man. 30 gr. of chloralamide did not relieve the delirium.

CASE IV.—Thoracic aneurysm; a woman. She suffered great pain. Before the introduction of chloralamide she was treated with injections of morphine. For the last month of her life she had many doses of 30 gr. or 40 gr. of chloralamide. She always slept well after it, and she said she preferred it to morphine.

CASE V.—Subacute nephritis. A woman aged 27. Slept well and comfortably after a dose of 30 gr.

CASE VI.—Mitral regurgitation, pericarditis. A boy aged 12. Slept all night after a dose of 5 gr.

CASE VII.—Mitral regurgitation and albuminuria. A man aged 40. 30 gr. made him sleep well and comfortably.

CASE VIII.—Extreme ascites, probably due to cirrhosis. An elderly man much troubled with insomnia. 50 gr. made him sleep well and comfortably.

CASE IX.—Carcinoma of the liver. A woman aged 38. She suffered extreme pain, but 30 gr. of chloralamide caused her to sleep well and doze in comfort.

CASE X.—Erysipelas. A middle-aged woman. 30 gr. always made her sleep comfortably.

CASE XI.—Rheumatic fever. A boy aged 10. 15 gr. every four hours gave him quiet sleep, although he suffered much pain.

CASE XII.—Rheumatic fever, delirium tremens, salicylate poisoning. A man aged 40; died a few hours after admission. 3i of chloralamide had no effect upon him.

CASE XIII.—Brachial monoplegia, probably due to embolism and cerebral softening. An old man, who was very noisy and delirious. 30 gr. always quieted him and produced sleep lasting some hours.

CASE XIV.—Mitral disease. A middle-aged woman. 30 gr. always produced sleep.

CASE XV.—Carcinoma of the pylorus. A woman aged 56. 30 gr. always produced sleep better than morphine.

CASE XVI.—Chronic eczema. A man aged 40. 30 gr. caused comfortable sleep.

CASE XVII.—Mitral regurgitation and floating kidney. A woman aged 50. 30 gr. produced sleep, although the floating kidney caused much pain.

CASE XVIII.—Spastic paraplegia. A woman aged 56. 20 gr. caused comfortable sleep.

CASE XIX.—Phthisis, aortic disease, saturnine paralysis. A man aged 65. 20 gr. always produced sleep, but it took twelve hours to act, so that if the medicine were given in the evening the patient did not sleep during the night, but he slept all the next day.

CASE XX.—Mitral regurgitation. An adult man. A severe case. 30 gr. produced comfortable sleep.

² *Lancet*, October 26th, 1889.

³ *Centralblatt für Nervenheilkunde*, August 1st, 1889.

⁴ *Deutsch. med. Woch.*, August 8th, 1889.