In 30 cases in which we have been able to obtain information as to sex, 18 were males and 12 females. Drs. J. Thomson and J. Raglan Thomas (case recorded by Morley Fletcher) kindly gave us information on this point. Most of the cases were under 4 months old.

The causation of the condition has attracted a great deal of attention and speculation. Of the explanations given, two are the most prominent: (a) That it is a congenital redundancy; "Nature, in her extreme anxiety to provide an efficient pyloric sphincter, has over-exerted herself, and produced too great a quantity of muscular tissue" (Cautley); and (b) that of "congenital gastric spasm," that is, there is a functional disturbance of the nervous mechanism of the stomach and pylorus leading to over-action, and so to hypertrophy (Thomson). It seems reasonable to combine these views so far as to believe that there is some congenital hyperplasia of the pyloric sphincter, and that spasm supervenes on this, and is largely responsible for the symptoms manifested.

Diagnosis.—Obstinate vomiting associated with constipation in an infant whose dietary is carefully supervised may suggest the disease, but a diagnosis cannot be made in the absence of a tumour. In this case, though we were attracted by the idea, it did not seem justifiable to make a positive diagnosis of congenital hypertrophic stenosis of the pylorus. Such a conclusion would carry with it the question of surgical interference, and in the absence of a palpable tumour it would be impossible to exclude all other possible causes of vomiting in an infant.

Prognosis.—Cases in which this condition has been diagnosed, and in which recovery has occurred, have been mentioned by Finkelstein (3), Senator (1), Batten (1), and Barlow (1). Possibly some of these instances are, as suggested by Still, developmental variations in the amount of muscular tissue in the pylorus, in which the stenosis is not great. In Batten's case, where a male child had signs and symptoms of the disease when 11 weeks old, and recovered under careful treatment, but died from acute broncho-pneumonia and gastro-enteritis when 11 months old, the stenosis was not extreme.

A point of interest is whether these slighter cases, that do not prove fatal in early life, may subsequently present themselves as cases of simple pyloric stenosis. In the 31 cases described as congenital stenosis of the pylorus collected by Maier, and Landerer the ages ranged from 12 to 80 years, and though the type is certainly a different clinical one, it is possible that they are the outcome of slightly marked muscular hypertrophy in early life. It is conceivable that a primary muscular excess might in course of time undergo fibrous transformation, and result in fibrous stricture of the pylorus.

Treatment.—Rectal feeding led to temporary improvement in this case, but the condition of the pylorus, as shown in the woodcut, could hardly have been successfully treated by any means short of surgical.

Slighter cases, when recognised, should be treated, as Batten suggests, by nasal feeding, which, by obviating the peristalsis set up by deglutition, probably both (1) allows digestion and absorption to go on, and (2) tends to minimise the spasmodic stenosis of the hypertrophied pylorus. Probably a combination of rectal and nasal feeding, with a cautious and gradual return to ordinary means would be the best plan.

Six cases have been submitted to operation. Recovery followed Loreta's operation by Nicoll; the others were unsuccessful. Pylorotomy was performed in a case of Thomson's, and gastrostomy in Meltzer's, Stern's, Adler's, and Thomson's cases.

For the photograph illustrating this paper we wish to express our thanks to H. G. Drake-Brockman, M.R.C.S.

REFERENCE.

¹ In the earlier of his two contributions Meltzer speaks of having seen five cases. His second and usually quoted paper is devoted to a detailed account of one case.

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FURTHER OBSERVATIONS ON EPIDEMIC ARSENICAL PERIPHERAL NEURITIS.

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It has been very gratifying to me to have had my observations on the symptoms and cause of epidemic peripheral neuritis amongst beer drinkers so fully confirmed, and even more pleasing to know that the source of the arsenic has been traced, as was first done by Professor Delépine to the glucose and invert sugar, and ultimately to the commercial sulphuric acid, so that there is

Fig. 1, Case 1.—Paralysis of extensors of wrist and fingers.

every probability of a stop being put to the outbreak. At first Dr. Brooke and myself were of opinion that the source of the arsenic would be found in some connection with sulphur, and from the symptoms presented by a private patient of Dr.

Fig. 2, Case 1—showing "dropped feet" from paralysis of anterior tibial and peroneal muscles; slight keratosis of soles of feet.

Brooke's it seemed to be in the sulphur with which the hops were treated. I am glad that this has been found not to be the case, but that it has been found in the sulphur compounds from which the sulphuric acid is made.

In the many extremely interesting papers which have been published, a few new facts have been recorded. First and foremost the discovery of the arsenic in the urine, of the presence of which I was first told by Professor Dixon Mannon November 27th, who had obtained it the day previously from 6 ounces of urine passed by a woman who had been recently drinking beer. Diarrhoea has been noted in the acute cases, affection of the nails in several, and vomiting and diarrhoea in the suckling infant. All these observations I can confirm from my more recent cases.

A few symptoms which I have not yet seen reported may be of interest. The pigmentation does not occur in fair-complexioned patients, or, at any rate, only to a slight degree, perhaps in the form of a deeper pigmentation of pre-existing freckles. In many cases there seems to be a distinct sequence of events as regards the rash;

Fig. 3, Case 2.—Showing pigmentation and very marked general oedema.

it is first erythematous, especially on the hands, face, neck, and, as pointed out to me by Dr. Luff, the upper part of the chest; this becomes by degrees much darker, then passes into marked pigmentation, which finally desquamates in small branny scales, and so is removed after some weeks. The bullæ seem to be rare, as I have only seen four examples. Herpes zoster (which was indeed the one symptom which first led me to suspect arsenic as the cause of the epidemic) is only moderately common, and yet not very rare, as I have already remarked on the great relative increase of such cases in the last six months in this district, and since November 18th I have seen 6 fresh cases of herpes presenting also other symptoms of arsenical neuritis.

I have seen not a few cases of neuralgia affecting various spinal nerves which was not followed by herpes, although I expected it would be. In one case there was anæsthesia (not quite total) of the whole of the left fifth cranial nerve, but I have seen no other cranial nerve affected.

Of the circulatory and respiratory symptoms I have noted in several cases post-sternal—sometimes epigastric—pain, together with marked breathlessness and a feeling of great exhaustion on exertion, these symptoms being very similar to those occurring in beri-beri. So great and so lasting has cardiac asthenia been, that some of our cases in the Crumpsall Hospital have fainted after being allowed to get up, and have had to be put back to bed; I shall refer to this in speaking of the treatment. The bronchitis and dilated heart in many cases have been marked features, and a few patients have spat blood, there being no appearance or physical signs of phthisis. The hoarse voice seems to be entirely due to catarrh and swelling of the vocal cords, for on examination I found this, but no paralysis of the cords.

The vomiting and diarrhoea had been so profuse and marked in the earlier stages that several women have told me they had not been drinking beer for a month before admission, but had been taking spirits, and when asked the reason said that the beer did not seem to be "agreeing" with them and had made them sick; this was, of course, before the presence of arsenic was known. Dr. Judson, one of the resident officers at Crumpsall, tells me that two or three of his patients have had hæmorrhage from the bowel.

Since November 18th, 1900, I have seen about 150 fresh cases, and since that date 4 deaths have occurred at Crumpsall, all women; 1 died suddenly from heart failure, 1 from dilated heart with oedema and bronchitis (see photograph), and 2 from extensive paralysis, gradual heart failure and exhaustion.

Fig. 4, Case 2.—Legs.

The interesting questions raised by my colleague, Dr. Judson S. Bury, as to how many of the total symptoms we must ascribe to arsenic, and how much to alcohol can, I think, hardly be answered at present, and the same applies to the differential diagnosis and etiology of beri-beri. I can only refer to my original paper, and repeat that so far as I can see the skin lesions are the distinguishing features.

That beri-beri and so-called alcoholic paralysis with heart failure are extremely similar is undoubted. So long ago as 1890 Dr. Alcock (now of Goole), who had recently been one of the House-Physicians at the Manchester Royal Infirmary (when the late Dr. James Ross was investigating alcoholic neuritis), and had just returned from the China voyage, told me that he could see no difference whatever between beri-beri and alcoholic paralysis with heart failure. It would be a delightful induction if we could say that beri-beri and alcoholic paralysis and oedema were due to arsenic poisoning, but the facts known at present do not justify such a theory, for it would not explain the absence of the skin lesions in beri-beri (I can find no mention of them in the published accounts) and so-called alcoholic paralysis, nor would it explain the different relative frequency of those symptoms common to the three conditions. I agree with Dr. Bury that it seems very probable that arsenic in itself is quite sufficient to cause serious failure of the heart muscle, and I

Fig. 5, Case 3.—Very marked pigmentation, with branny desquamation of pigmented scales; wasting of arms from atrophic paralysis.

am inclined to think it may cause some mental confusion, but certainly not to the same extent as we ordinarily find in alcoholic paralysis.

Fig. 6, Case 3.—Showing marked wasting of anterior tibial and peroneal muscles of legs.

As regards the duration of the present epidemic, I think I can trace it back for over twelve months, for during this period I have been much struck and puzzled by the number of cases of pigmentation occurring at Crumpsall.

Treatment.—I trust I may be allowed to endorse, with a few exceptions, the most wise and excellent account of the treatment of these cases given by Dr. Judson Bury in the BRITISH MEDICAL JOURNAL for December 8th. The exceptions are that having seen such alarming heart symptoms and such feebleness of the pulse with low tension at Crumpsall, I should hardly like to give such depressing drugs as salicylate of soda and iodide of potassium, even though the latter drug is said by Sir William Gowers to aid in the elimination of the arsenic. For the same reason I should not recommend antipyrin or exalgin for the pain, though phenacetin might possibly be "safer." In the early stages we have been content with rest in bed, a little digitalis, tonic doses (3 or 4 minims) of liq. strychninæ, bismuth, and soda if the stomach symptoms were prominent, and carbonate of ammonia and senega for the bronchitis; for the pain, small doses of morphine have seemed to us to be the best treatment. With these exceptions, let me again say how cordially I agree with what Dr. Bury has said.

Finally, I must thank my assistants, Drs. Sissons, Anderton, and Judson, the resident medical officers at the Manchester Workhouse Infirmary, for their great help in what has been and still is a most arduous task.

Photographs of 3 cases, showing various types are here reproduced. One woman (unpigmented) is of fair complexion, but in addition to the paralysis, has the typical keratosis of the soles of the feet.

THE STRENGTH OF THE HYPERTROPHIED BLADDER.

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Mentone.

THE strength of the hypertrophied bladder, if I may judge from the descriptions given of it, would appear to be no greater as a safeguard against rupture than that of the bladder normally. Obviously, if this be so, the risk of rupture becomes considerably increased. The distribution of the muscular coat leaves the impression that "the mucous membrane between the fasciculi is insufficiently supported," and that the bladder hypertrophies foolishly, and to its own hurt.

A consideration, however, of the thin rubber ball covered with netting commonly attached to a spray apparatus should correct this impression. Such netting not only prevents a too great expansion of the ball, but materially strengthens the rubber seemingly unsupported between its meshes. Each little sacculus of rubber as it bulges out forms the segment of a sphere whose diameter is many times smaller than that of the rubber ball itself, by which without the netting it would alone be supported. The netting allows the sacculus to form, and pulls its walls inwards. Without the netting no succulus could form, and the walls of the same segment of rubber could only be supported by the almost tangential force of the adjacent rubber, which would have to be very great to afford a sufficient radially directed pressure-component to resist the outward pressure. The fasciculi, therefore, by altering the direction of pull, save the rubber from being exposed to a strain which otherwise would have increased as the pressure augmented, and *ceteris paribus* in the same proportion.

The strength of a segment is increased in the inverse ratio of the size of the sphere of which it forms part. Thus if when not fasciculated the bladder have a diameter of 4 inches, the force required to burst a certain segment of its walls may be represented by s . If, without the bladder altering in size, the segment be supported between muscular fasciculi so that the segment now is part of a sphere whose diameter is 1 inch,

the bursting force now becomes S , and $\frac{S}{s} = \frac{4}{1}$.

When the bladder, therefore, hypertrophies, its whole wall is strengthened. The interfascicular spaces, by becoming sacculated, are put into what is economically and mechanically the most ideal condition for supporting pressure. When large sacculi develop, or spontaneous rupture occurs, the causes are probably additional pathological complications rather than errors in mechanical principles during hypertrophy.