patients vary greatly in their sensitivity to the rays, and the dose which would cause slight erythema in one might procure intense redness and subsequent blistering in another. The best way to avoid this is to make a series of test exposures on small areas of skin. Four small windows, each being one inch square, should be cut fairly close together in a sheet of black paper. The paper should then be wrapped around the forearm and the four areas of uncovered skin exposed to the rays, the lamp being at a distance of two feet. These areas should receive one, two, three, and four minutes' exposure respectively. The same hour next day the areas should be examined. The length of exposure to the area in which there is distinct redness should be the length of treatment to the affected region.

The diseases which may be treated by ultra-violet rays I shall classify in three groups. The first includes those in which the treatment will always effect a cure. The second contains those in which the treatment will generally effect a cure, either alone, or in conjunction with other forms of treatment. The third includes a large number of diseases or cases, mostly common and obstinate, for which the treatment is now being tried. In these diseases the insufficiency of experience renders it difficult to say whether the rays are of real use. To these groups I shall add a fourth, which will include those in which the treatment is likely to produce harmful results.

Group 1.—The disease which must be given pride of place in ultra-violet ray therapy is rickets. For this the treatment may justly be regarded as a specific. I do not know any other disease for which it is a specific, although tetany and laryngismus stridulus—when occurring in rickety children—are said to respond equally well.

Group 2.—In this group I would place first the cases of infants who are unable to assimilate their food, and who consequently waste. If no organic disease is present the major number begin to thrive and put on weight. The treatment must be applied very cautiously and minute doses given. Children who have been the subjects of the fevers which commonly occur during their early years, but are left in a weak debilitated state, respond well to the treatment. Their health improves and they gain in weight. Nasal and bronchial catarrh lessens or disappears, and aural discharge loses its offensive character and may disappear. If the last-mentioned trouble is present, that most valuable form of treatment, zinc ionization, must not be forgotten. Adults who are left in a debilitated state after influenza or similar types of fever improve greatly after ultra-violet irradiation. In surgical tuberculosis the rays can play a valuable part in bringing the disease to an end; but additional treatment is generally required, such as surgery, application of splints, etc.; and residence at the sea or in the country are necessary for most cases. For this reason the treatment of surgical tuberculosis by ultra-violet rays in large towns is frequently unsuccessful. In dermatology the disease which is most often treated by the rays is lupus vulgaris. General treatment is more effective than local, though they can be combined with advantage. Local treatment requires the water-cooled lamp. The electro-thermic methods are very useful in local treatment, but general treatment should not be omitted. Other infective diseases of the skin, such as impetigo contagiosa and acne vulgaris, respond well. Patients who are liable to boils and carbuncles may be treated by the rays with advantage. Their low resistance to infection by staphylococci can thereby be increased. In seborrhoea I have had some good results in obstinate cases. In true eczema success is not often obtained. The so-called "weeping eczema" (which is really seborrhoea) improves under the treatment, and the "trade eczemas" (which are really forms of dermatitis caused by chemical irritants) can be cured if the causes are removed. Non-specific indolent ulcers should receive a trial of the treatment, both local and general, and success will often be obtained. Erythema pernio is said always to respond to the rays, but I prefer diathermy. In true psoriasis I have had no success with ultra-violet rays. Some cases of pruritus ani and vulvae can be freed from the itching. These cases require local treatment, and when it is given every particle of mucus and exudation and previously applied unguents must be removed. Some

writers have described good results in the treatment of erysipelas by the rays. I have had no personal experience. In alopecia areata the hair grows again after a course of local treatment by the rays. A long course may be required.

Group 3.—This includes a large number of chronic maladies and obstinate cases which resist drug treatment. The "rheumatic material," such as chronic fibrositis and arthritis, with which the various departments of physiotherapy are so plentifully supplied, has been treated by the rays, but I have had infinitely better results—at any rate in women—by pelvic diathermy. Treatment of chronic fibrositis and arthritis by the rays will generally be followed by disappointment. The ultra-violet rays have been tried in infection of the uterus and its adnexa and the pelvic supporting tissues, erosion, and some forms of dysmenorrhoea, but here again pelvic diathermy gives far better results. In obstinate cases of sciatica and brachial neuritis I have had little success with the rays after other forms of physiotherapy had failed, although it is possible that the cases which had been cured by the latter would have responded to the rays. Hyperpiesis, chronic bronchitis, bronchial asthma, hay fever, Raynaud's disease, acrocyanosis, and other diseases are being treated by ultra-violet rays, and success has been claimed by different writers, but sufficient experience has not been gained to enable a definite statement to be made regarding the therapeutic value of the rays in these diseases.

Group 4.—This includes the diseases and patients for which the rays should be administered with extra caution or not at all. Patients with general pyrexia should not receive ultra-violet ray treatment. In acute local infection the rays should not be applied locally, nor should general treatment be given if the body temperature is raised. If pus is present the treatment should not be given until it has been evacuated. If it is suspected, the treatment should be postponed. In pulmonary tuberculosis the treatment should not be given, except by an expert; quiescent phthisis has been rendered active. It is inadvisable to apply the rays to patients who are suffering from failing hearts or who are the subjects of Bright's disease. It is generally inadvisable to give the treatment to very old people. During menstruation ultra-violet ray treatment should be omitted.

In concluding a short lecture on a very large subject I would insist on treatment by ultra-violet light being under the direction of medical men. Unqualified people, even if they are trained, can only administer the rays, but the doctor can employ them to treat disease.

CONGENITAL OESOPHAGEAL OBSTRUCTION.

BY

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CONGENITAL malformations of the oesophagus are by no means extremely uncommon, and usually manifest their presence within a few days of birth; almost all prove rapidly fatal. Life may, however, be saved in some of the rarer cases by prompt intervention, and I propose to give details of a case of complete oesophageal obstruction, operation, and recovery, followed by pyloric stenosis, a second operation, and recovery. Before doing so I will outline the development and the congenital malformations of the oesophagus.

DEVELOPMENT.

From the primitive foregut the pharynx, oesophagus, and stomach are developed. In the third week the oesophagus is a mere sphincter, or constriction, between the pharynx and the stomach.¹ When the neck is differentiated during the second month the oesophagus undergoes a rapid elongation; the chief factor in producing this elongation is the development of the lungs and pleural cavities, whereby the stomach is pushed backwards into the body cavity. Up to the third week the trachea and oesophagus form a single structure; the lung-buds have not yet appeared. There is, however, a histological differentiation of the two parts, which will be described later.

Separation of the oesophagus from the trachea takes place during the third and fourth weeks by the growth of two lateral septa, which eventually meet and fuse in the mid-line to form a partition from which is differentiated the posterior (membranous) wall of the trachea and the anterior wall of the oesophagus (Fig. 1). The last portion to fuse is the caudal extremity of the partition—that is, at the level of the bifurcation of the trachea. In the fourth week the lung-buds arise from the distal extremity of the respiratory tube, and by their rapid increase in size cause the elongation of the oesophagus referred to above.

Differentiation of the Histological Structure.

In a 4 mm. embryo the oesophagus is an epithelial tube, flattened from side to side, with a ventrodorsal lumen. It is lined with two layers of cells, the nuclei nearest the lumen showing mitotic figures. The upper portion of the oesophagus shows a slightly different structure; here the dorsal portion of the tube is lined with a single row of cells, while the ventral portion shows three or four layers; this thickened portion is destined to form the respiratory tract. The tube is surrounded by a mass of undifferentiated mesenchyme.

After the trachea and oesophagus have been separated by the fusion of the septa the oesophageal lumen becomes greatly narrowed by a proliferation of the epithelial lining; some authorities consider that it becomes entirely obliterated;² others maintain that the original lumen is never entirely lost.^{3, 4} The former view is probably correct. In 20 mm. embryos vacuoles appear in the epithelium. There are two theories as to their origin: (1) that they are due to accumulation of intracellular fluid; (2) that they are due to an active moving apart of the cells through a transference of mitotic activity from the inner to the outer layer of cells. The latter is regarded as being the most probable explanation of their formation. The result is an enlargement of the lumen due to coalescence of neighbouring vacuoles.

FIGS. 1 TO 9.—Diagrams showing congenital anomalies of oesophagus.

CONGENITAL MALFORMATIONS.

There are seven types of congenital malformation:

1. Total absence of the oesophagus.
2. Doubling of the oesophagus.
3. Oesophago-tracheal fistulae.
4. Partial obliteration of the oesophagus.
5. Diverticulum formation.
6. Simple congenital strictures.
7. Membranous or valve-like strictures.

1. *Total Absence of the Oesophagus.*—This occurs only in acardiac monsters, in whom the head also is usually missing, and this type is of no importance.

2. *Double Oesophagus.*—Blasius (1674)⁵ records a case of a newborn infant in which the middle two-fourths of the oesophagus was double; this, however, did not cause any obstruction. I can find no record of any similar case.

3. *Oesophago-tracheal Fistulae.*—As was stated in the section dealing with the development of the oesophagus, the two lateral septa of the cephalic extremity of the foregut, which meet and fuse in order to differentiate the upper half of the oesophagus from the trachea, have their point of ultimate fusion at the lower end of the trachea. In this situation they may fail to meet, and hence an oesophago-tracheal fistula results. (a) If the oesophago-tracheal septa develop in their normal plane, apart from the fistula, both organs are normal (Fig. 2). It rarely

happens that a fold of mucous membrane covers this opening. (b) If the plane of development of the oesophago-tracheal septum is faulty, the latter becomes attached to the posterior wall of the foregut and so leads to obliteration of the upper part of the oesophagus, together with a fistulous communication of the lower part of the oesophagus with the trachea.⁶ This is the commonest congenital anomaly of the oesophagus (Fig. 3).

4. *Partial Obliteration of the Oesophagus.*—(a) Simple blind ending of the upper part of the oesophagus may occur, possibly as a result of a deviation in the lines of development of the two lateral septa. In these circumstances the proximal end of the lower part of the oesophagus is blind also. Another explanation is that the rapid growth of the lung-buds has ruptured the continuity of the slender tube which is the primitive oesophagus.⁷ It sometimes happens that the upper and lower blind ends are united by a strip of muscle tissue (Fig. 4). This would appear to be due either to a rupture of the inner layer of cells or to a failure of the epithelium in a portion of the oesophagus to undergo vacuolization, and therefore the lumen, which, according to some observers, has been entirely obliterated at one stage of development, never again becomes patent. The outer layer of cells, however, persists, and forms a thin strand of muscle. (b) Obliteration of the oesophagus may occur only from the level of the tracheal bifurcation downwards, the oesophagus above this level being normal (Fig. 5).

These varieties of malformation of the oesophagus are of interest because of their frequency and because of the embryological factors concerned in their production. Clinically, however, they are of small importance because no treatment is of any avail. These anomalies are often associated with other congenital malformations—for example, Meckel's diverticulum, imperforate anus, and duodenal atresia.

In Types 3 and 4 above severe interference with swallowing is occasioned; gurgling sounds are heard when fluid enters the trachea as soon as the infant swallows, followed by violent attacks of coughing and vomiting. If feeding is persisted in the child may be asphyxiated or pulmonary complications may supervene. Death occurs either from these or from inanition.

Curable Types.

I shall now describe the varieties of congenital anomalies of the oesophagus which, although very rare, yet are of great clinical importance because their early recognition and prompt treatment may make all the difference between the life and death of the patient.

5. *Congenital diverticulum* at the pharyngo-oesophageal junction is extremely rare, occurs in the same position as the acquired Zenker's pulsion diverticulum, and eventually produces similar symptoms. It is possibly atavistic in origin, being present normally in the hog and some other animals.^{8, 9, 10} It differs from a true Zenker's diverticulum by having a complete muscular covering, but its treatment is the same as that of the acquired form.

6. *Simple Congenital Strictures.*—In these cases there is a definite narrowing of the whole circumference of the oesophageal wall at one or more places. This type is very uncommon. The narrowed portion may be either circular and confined to only a small area (Fig. 6) or may extend through more or less of the length of the oesophagus (Fig. 7). Rarely is the upper end of the gullet the affected region;^{11, 12, 13} more commonly it is the lower segment.¹⁴⁻¹⁸ The symptomatology is similar to that of the succeeding variety.

7. *Membranous or Valve-like Strictures.*—Membranous or valve-like strictures are, according to Kraus,¹⁹ very rare. The membrane may form a ring-like fold (Fig. 8) or a complete diaphragm (Fig. 9), and is generally situated a very short distance above the cardia. The condition has been known to be associated with an oesophago-tracheal fistula, but may be the only abnormality.

The *etiological factors* concerned in the formation of simple congenital strictures and of membranous strictures, whether complete or incomplete, would appear to be one or more of the following errors of development.

1. The "mere sphincter" between the pharynx and the oesophagus in a 4 mm. embryo referred to above does not elongate equally, but leaves a stenosis of one portion of the canal.

2. The obliterated tube fails to canalize at one place.

3. Although canalization occurs, yet the inner layer of cells fails to transfer its mitotic activity to the outer layer, and therefore a local area of oesophageal wall remains without coalescence of the local vacuoles, and stenosis results.

SYMPTOMS AND DIAGNOSIS.

In both simple congenital and annular membranous strictures cases have been recorded in which symptoms were not produced until, after weaning, the child was found to be unable to take solid food, which was almost immediately regurgitated. By feeding solely upon fluids, chiefly milk, life has been maintained, and patients have lived for many years before the true nature of the lesion has been discovered. These patients are very underdeveloped and thin, and males are said to show only slight masculine characteristics.

On the other hand, depending upon the degree of atresia, symptoms of oesophageal obstruction, notably dysphagia and the regurgitation of fluids, may make their appearance as soon as fluid is given to the newborn infant.

If the membranous stricture is not annular but in the form of a complete diaphragm, symptoms of complete oesophageal obstruction are always present from birth. The precise diagnosis of the actual type of oesophageal malformation which is present can only be arrived at by means of a careful x-ray examination and oesophagoscopy, and should not be left until the *post-mortem* examination is reached.

COMPLICATIONS.

If left untreated all cases of complete occlusion die of inanition; also the more severe stenoses. With an inadequate lumen the stagnated food may produce a marked oesophagitis in the portion of the oesophagus which is dilated above the stricture. The ulcerative process may actually cause perforation. In rare instances a pressure diverticulum is formed above the stenosis, as in one of my cases. Probably apart from this secondary dilatation of the oesophagus a true congenital dilatation does not occur.

TREATMENT.

(a) For Simple Congenital and Annular Membranous Strictures.

Simple strictures are best treated by slow gradual dilatation by means of bougies. This produces a more permanent result, and much less damage, than when dilatation is performed rapidly. Rapid dilatation causes ulceration at the site of the lesion, and is followed by fibrosis, contraction, and a tighter stricture than was present before treatment commenced. In addition, too rapid dilatation may cause perforation or rupture of the oesophagus.

Treatment, therefore, consists in the passage of carefully graduated bougies. At the first sitting a bougie which only comfortably fits the stricture is passed under direct vision through the oesophagoscope. At subsequent sittings, which should take place once or twice a week, bougies may be passed blindly provided there is not a great deal of dilatation of the oesophagus or diverticulum formation above the stricture. Commence with a bougie of the same size as the one passed through the oesophagoscope, and when this has remained *in situ* for a few minutes replace it by a bougie of one size larger; and at each subsequent sitting pass, if possible, one bougie a size larger

than the largest previously used. In no circumstances should any force be used.

Annular membranous strictures may respond to similar treatment, but, as with this type of stricture considerable dilatation of the oesophagus is usually present, and the lumen is always eccentric, the bougies must invariably be passed under direct oesophagoscopy. This type of stricture lends itself to internal oesophagotomy, whereas with simple strictures involving a considerable length of the oesophageal canal this procedure is fraught with much danger. A ring-like stricture may also be treated successfully by electrolysis.

In an emaciated, dehydrated patient, or if in spite of great care and patience improvement does not occur, a gastrostomy is necessary. This allows rest to the oesophagus, permits feeding of the patient, and a retrograde oesophagoscopy may be performed. By this means, or by peroral endoscopy, a filiform bougie to which is attached a silk thread is passed through the stenosis. The thread is drawn upwards or downwards, as the case may be, until one end projects out of the mouth and the other through the gastrostomy opening. Each end of the thread is attached to the two ends of a fine piece of rubber tubing, and therefore a continuous or endless chain is present. By means of the thread the tubing is pulled hard enough to render the diameter of the rubber small enough to pass the stenosis. When tension on it is relaxed its length is diminished and its diameter increased, and it then exerts continuous pressure on the stricture and causes further dilatation. The rubber is left in position for an hour or

FIGS. 10 AND 11.—Congenital stricture of oesophagus treated by rubber tube.

two every day, and then by pulling upon the thread the rubber is displaced from the oesophagus and its place taken by the thread. Every few days the tubing must be replaced by one of larger calibre until sufficient dilatation is obtained (Figs. 10 and 11).

(b) A complete membranous diaphragm in a newborn infant may be perforated by means of the oesophagoscope or a bougie. In this way I have recently treated with success an infant less than two days old suffering from this condition.

CASE OF COMPLETE MEMBRANOUS DIAPHRAGM.

The patient was a female child, who was born on the evening of January 18th, 1928. The mother was a primipara, and the labour, which was not abnormal in any way, lasted twenty-one hours. The child weighed 7½ lb., and appeared to be perfectly healthy.

Throughout the whole of the following day attempts were made to feed the child at regular intervals. At no time would she suckle the breast, and when given teaspoonfuls of milk and water the infant appeared to have great difficulty in swallowing, and regurgitated the fluid as soon as it had been taken. On one occasion only was there a small quantity of blood, but on each occasion she coughed and choked a great deal and became very cyanosed.

I saw the child with Dr. Henry Robinson about noon on January 20th, when she was just forty-two hours old. Outwardly she appeared quite normal; but on feeding with a teaspoonful of water she coughed, became cyanosed, and retained none of it. The child obviously had a congenital abnormality of the oesophagus, and this was most likely to be in the nature of an oesophago-tracheal fistula. The infant was twenty-five miles from London, and x-ray examination was not immediately available; but, having an oesophagoscopic outfit, I decided to perform oesophagoscopy forthwith. This was done without, of course, any

anaesthetic, using a 4 mm. tube (Jackson's pattern), which passed quite freely down to and beyond the level of the bifurcation of the trachea. About 1 in. below this level, however, the lumen became obstructed by a glistening membrane with a "mother-of-pearl" appearance, which was at first thought to be inspissated mucus. Careful swabbing, however, showed it to be a solid structure, and no opening could be found in it (Fig. 12).

By pressing firmly with the endoscope the membrane was ruptured and normal oesophageal mucosa was seen below it. Gastric mucosa was easily recognized after the tube had been pushed about 1 in. further. On withdrawing the oesophagoscope a few drops of blood obscured the view of the wall of the gullet at the level where the membranous diaphragm had previously been seen.

FIG. 12.—Membrane as seen by oesophagoscope.

No other abnormality was detected in the pharynx, larynx, or oesophagus.

One ounce of exhausted breast milk was now given to the infant, who swallowed it normally and retained it. This appeared very satisfactory, and we thought it worth while to investigate the case still further by means of x rays. With Dr. Robert Knox we examined the infant the same afternoon with the fluorescent screen while she swallowed an emulsion of bismuth, and a definite narrowing of the lumen of the oesophagus, together with a slight delay in the passage of the bismuth, was noted.

about an inch above the stomach. No other abnormality of the upper alimentary tract could be detected, and we were able to give a fairly good prognosis. This was borne out by the fact that the child, although she persisted in refusing the breast, swallowed and retained fluid normally, and scarcely vomited again. Temperature, pulse, and respiration were normal throughout.

The baby, which had lost about $\frac{1}{2}$ lb. in weight, made steady progress until four weeks later, when she weighed 8 lb. She then began to vomit forcibly through both the nostrils and the mouth. A gastric tumour was visible and a pyloric tumour palpable on the second day of these symptoms, and hypertrophic pyloric stenosis was diagnosed. Laparotomy, on February 18th, confirmed this, and no other abnormality of the stomach or duodenum was found. Rammstedt's operation under gas and oxygen anaesthesia was followed by an uninterrupted recovery. For some considerable time after this the child was difficult to feed, but has now settled down and is doing well; at 5 months old she weighed 11 $\frac{1}{2}$ lb.

The points which I wish to emphasize with reference to this case are:

1. So far as I know, this case is unique; I can find no record in the literature of any previous case which has survived.
2. Congenital oesophageal malformations are not so rare as might be supposed.
3. The symptoms of all varieties are almost identical.
4. Some cases which are curable are probably diagnosed as being incurable.
5. A careful examination by means of prompt oesophagoscopy, with or without x-ray examination, should lead to a cure of a number of cases for which at present no treatment is undertaken.

REFERENCES.

- 1 Keith, A.: *Human Embryology and Morphology*, fourth edition, 1921.
- 2 Kreuter, E.: *Die Angeborenen Verengerungen u. Verschlüssungen des Darmkanals*, Leipzig, 1905.
- 3 Forssner: *Anat. Hefte*, 1907, xxxiv, 1.
- 4 Johnson, F. P.: *Amer. Journ. Anat.*, 1910, x, 521.
- 5 Blasius: *Observata Medica Rariora*, 1674, Table xv, Fig. 6.
- 6 Keith, A., and Spicer, J.: Three Cases of Malformation of Tracheo-oesophageal Septum, *Journ. Anat. and Physiol.*, 1907, xli, 55.
- 7 Shattock, S.: Congenital Atresia of Oesophagus, *Trans. Path. Soc. Lond.*, xli, 87.
- 8 Albrecht, W., and Brünings, W.: *Neue Deutsche Chirurgie*, 1915, Band 16, Direkte Endoskopie.
- 9 Negus: *Journ. Anat., Otol. and Laryng.*, 1925.
- 10 Jackson, C.: *Bronchoscopy and Esophagoscopy*, 1927.
- 11 Home, E.: *Practical Observations on Treatment of Stricture in the Urethra and in the Oesophagus*, 1821, ii, 398.
- 12 Cassan: Quoted by Cruveilhier, vide infra.
- 13 Zenker: *Hand. Spec. Path.*, 1878.
- 14 Cruveilhier: *Anatomie Pathologique*, xxxviii, 1.
- 15 Fagge, C. Hilton: *Guy's Hospital Reports*, Series III, xvii, 413.
- 16 Mayo, C. H.: *Annals of Surgery*, 1923, lxxvii, 267.
- 17 Hirschsprung: *Hosp. Tid.*, 1895, iii, 1037.
- 18 Schneider: *Angeborene Speis. Dissertation*, 1900.
- 19 Kraus, F.: *Spec. Pathologie u. Ther.*, Nothnagel, xvi, pt. i, div. ii, Die Erkrankungen der Speiseröhre, 1902.

ADDITIONAL BIBLIOGRAPHY.

- Brenneman, J.: Atresia of the Oesophagus. *Amer. Journ. Dis. Child.*, 1918, xvi, 145.
- Brown, K. P.: A Case of Congenital Narrowing of the Oesophagus. *Edin. Med. Journ.*, 1922, N.S., xxix, 145.
- Cautley, E.: Malformation of the Oesophagus. *Brit. Journ. Child. Dis.*, 1917, xiv, 1.
- Griffiths, J. P. C., and Lavenson, R. S.: Congenital Malformations of the Oesophagus; with Report of a Case. *Arch. Pediat.*, 1909, xxvi, 161.
- Gross, F., and Sencert, L.: *Rev. de Chir.*, 1907, xxxv, 1.
- Guizez, J.: *Diagnostic et traitement des rétrécissements de l'oesophage*, p. 46, 1923.
- Hess, J. H.: Stenosis of Lower Esophagus at Cardia in a Child aged 2. *Med. Clinics of N. America* (Chicago Number), 1923, vii, 499.
- Hirsch, I. S.: Congenital Atresia of the Esophagus; Report of Two Cases. *Journ. Amer. Med. Assoc.*, 1921, lxxvi, 1491.
- Huntingdon, J. L., Young, J. H., and Foot, N. C.: Report of Case of Congenital Atresia of Oesophagus, *Boston Med. and Surg. Journ.*, 1919, clxxx, 354.
- Hutchison, R.: Congenital Stricture of Oesophagus. *Proc. Roy. Soc. Med.*, 1923, xvi, Sect. Dis. Child., 42.
- Kastner: Congenital Atresia of the Esophagus, *Arch. Pediat.*, 1920, xxxvii, 670.
- Keith, A., and Spicer, J.: On Developmental Factors. *Journ. Anat. and Physiol.*, 1906, xli, 52.
- Marcus, J. H.: Atresia of the Anus, Rectum, and Esophagus. *New York Med. Journ.*, 1923, cxviii, 374.
- Plass, E. D.: Congenital Atresia of the Esophagus with Tracheo-oesophageal Fistula, associated with Fused Kidney: a case report and a survey of the literature on congenital anomalies of the esophagus. *Johns Hopkins Hosp. Reports*, 1919, xviii, 258.
- Reynolds, R. P., and Morrison, W. W.: Congenital Malformations of the Esophagus, *Amer. Journ. Dis. Child.*, 1921, xxi, 339.
- Richter, H. M.: Congenital Atresia of the Oesophagus, with Report of Two Cases. *Surg., Gynecol. and Obstet.*, 1913, xvii, 397.
- Sargnon, M.: Congenital Stricture (Membranous) of Oesophagus in Region of Cardia in a Child aged 10 $\frac{1}{2}$ years. *Lyon Med.*, 1923, cxxxii, 538.
- Shattock, C. E.: Specimen of Congenital Malformation of Oesophagus. *Proc. Roy. Soc. Med.*, Sect. Dis. Child., 1922, xv, 2.
- Shaw, H. L. K.: Congenital Atresia of the Esophagus; with Report of Case. *Amer. Journ. Dis. Child.*, 1920, xx, 507.
- Taglicht: Case of Malformation in Stillborn Infant. *Virchow's Archiv f. Path. Anat.*, 1921, 229, 322.
- Turner, F. C.: Congenital Stenosis of Lower End of Oesophagus. *Trans. Path. Soc. Lond.*, 1885, xxxvi, 185.
- Vinson, P. P.: Congenital Strictures of the Esophagus; with Report of a Case. *Journ. Amer. Med. Assoc.*, 1923, lxxx, 1754. *Collected Papers of the Mayo Clinic*, 1923, xv, 3.
- Weiss, E.: Congenital Atresia of Esophagus with Esophagotracheal Fistula: Report of Three Cases, with illustrations of two. *Journ. Amer. Med. Assoc.*, 1923, lxxx, 1.
- Willard, H. G.: Congenital Atresia of the Esophagus. *Journ. Amer. Med. Assoc.*, 1922, lxxviii, 649.

PRIMARY SPASMODIC DYSMENORRHOEA AND ITS TREATMENT.

BY

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PAIN at the commencement of the menstrual period is very common, and is met with in varying degrees of intensity. It may be only a transitory sensation, or it may be severe enough to force the patient to bed. The more severe types present a trying problem, not only by reason of the distress, but also because of the interference with the patient's livelihood as a result of her inability to follow her normal occupation, which may be more important from her point of view. Patients are nearly always nullipara, though there are exceptions.

In typical cases the pain may come on a few hours before, and end with the menstrual flow, or it may continue for forty-eight hours of that period. Nearly always cessation of pain coincides with a freer flow. There are, of course, cases in which pain lasts longer, even throughout the period, but in suggesting a treatment it is better to consider only the typical cases which constitute the majority. The atypical yield to treatment devised on the same lines.

The patients, as a rule, are anaemic and listless, but the rapid improvement which follows removal of pain suggests that these are, in a large part, secondary symptoms. The increased menstrual flow which follows the usually sudden cessation of pain indicates that an obstruction has been overcome. That obstruction occurs at the junction of the body of the uterus with the cervix. When menstruation commences, in the normal subject, the potential uterine cavity becomes a real one through separation of its walls by accumulating menstrual fluid. Real resistance is met