two or three times the calcification of the skeleton was either not increased at all or only slightly.

3. The ingestion of even very large quantities of calcium did not cure the rachitic calcium impoverishment of the skeleton of rats on a diet deficient in the fat-soluble factor.

4. When the exhaustion of the fat-soluble factor in the body of rats is not complete, an excess of calcium in diets deficient in fat-soluble factor may in some cases slightly increase the amount of calcium in the skeleton, though never sufficient to make the calcification normal.

5. On the basis of the data of the chemical and histological examination, the skeletal affections in rats on —A diet must be classed in the group of rachitic affections, which develop even with an excess of calcium in the diet.

INTUSSUSCEPTION SUPERVENING ON CONGENITAL STENOSIS OF THE ILEUM.

BY

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CASES of intussusception as the result of congenital constriction of the small intestine form a group often emphasized in textbooks, but records of such cases are comparatively infrequent. In a series of 400 cases of intussusception at the London Hospital, Perrin and Lindsay¹ found only two due to this cause. According to Leichtenstern² intussusception as a result of congenital stricture is uncommon, as the dilated hypertrophied proximal segment is not easily invaginated into the small empty distal portion. The following case, therefore, seems of sufficient interest to record. I am indebted to the late Mr. T. H. Kellock for permission to publish it.

A boy aged 11 years returned home from school on June 30th, 1922, complaining of pains in the abdomen; administration of castor oil was followed by vomiting; the sickness and pain continued throughout the night; next day the vomiting ceased though the pain persisted, becoming more severe at night. His condition became progressively worse, vomiting recurred, and he was admitted to the Middlesex Hospital on July 3rd. He was a tall thin boy of poor muscular development; his face was pale, drawn, and anxious, the tongue dry and furred, the lips and teeth were crusty. The temperature was 97°, pulse 124, respirations 28. The abdomen was distended and tender, especially in the lower part, and showed no movement on respiration. In the centre of the lower part of the abdomen was an indefinite swelling dull to percussion. No note was made as to visible peristalsis.

Operation (by Mr. Kellock).

A right paramedial incision, 4 inches long, was made below the level of the umbilicus. The peritoneum was injected, and the cavity contained much blood-stained fluid. The distended and engorged small intestine which presented was followed down until the cause of the obstruction was reached in the lower ileum. This proved to be an intussusception. The greater part of it was reduced, but a portion some 4 inches long was irreducible, and showed gangrenous patches. Resection of this part was performed, and, as the patient's condition was bad, anastomosis was effected by a Murphy's button. The abdomen was closed except for a small gauze drain.

On the evening of that day the temperature rose to 99.4°, the pulse and respiration rate being 140 and 28. His condition remained critical for some days. Removal of the gauze drain on the fifth day released a discharge with faecal odour. By the eighth day the general condition showed slight improvement, but a fistula, freely discharging intestinal contents, had developed. The Murphy's button was not recovered until the twenty-eighth day. The escape of intestinal contents slowly diminished and the surrounding skin, which had been reddened and excoriated, began to heal.

The patient was discharged to a convalescent home seven weeks after the operation, with the fistula practically closed. His general condition was very satisfactory. He is now completely recovered, has put on weight, and looks robust and well.

The grant of the Medical Research Council and the hospitality of the laboratories of the Lister Institute have enabled me to carry out this work, and to them my thanks are due. I wish also to express my sincere gratitude to Professor C. J. Martin for his continuous support in this investigation, and to Miss M. Carr for her assistance in chemical analyses.

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The Specimen.

Examination of the specimen removed showed a pure enteric intussusception. When the two outer coats were divided longitudinally it was found that the intussusception had commenced by the invagination of a portion which was the seat of a congenital stricture into the part below. The stricture was over 2 inches in length, and the lumen of the gut at its narrowest part was not more than an eighth of an inch in diameter. The wall of the gut at the stricture is not much thickened, the external diameter being about a quarter of an inch. A nodule about the size of a hazel nut, which was incorporated in the wall of the gut, appeared to be the most advanced point of the entering layer; it was an effusion of blood into the tissues, probably secondary to the intussusception.

The whole specimen showed the congestion and ulceration usual in cases of intussusception of some days' standing.

The different ways in which a marked congenital stricture of the ileum may affect the life and health of an individual are well shown in the notes on three specimens in the Middlesex Hospital museum. Bland-Sutton,³ who applies the name "im-perforate ileum" to such deformities, has illustrated two of the specimens in his article in the *American Journal of the Medical Sciences* and in his book on tumours. In the first

specimen, which shows the condition to which he gives the name "septate ileum," the lumen is interrupted 36 inches above the ileo-caecal valve by a perforated diaphragm which reduces the diameter of the channel to a quarter of an inch. Externally a slight constriction marks its site. This septum had caused no symptoms, and was found *post mortem* in a man, aged 62, who had died from myocarditis and emphysema. The second specimen shows complete interruption of the ileum in an infant; the patient was under the care of Sir John Bland-Sutton, who diagnosed the condition and operated; death occurred, as it almost

The specimen removed at operation. (From a drawing by Miss D. Clephan.) The outer and middle layers of the intussusception have been opened up to show the congenital constriction, A, in position. The most advanced point, B, shows a rounded nodule projecting on the peritoneal surface; this on section consisted of blood clot. C, ulcerated or gangrenous portion of the outer layer.

invariably does. Only two cases are recorded in which an infant with complete stenosis in this region has recovered. In both of these the lesion was single, and it was possible to perform entero-anastomosis. When the condition is multiple death has so far invariably been the result. In many cases, according to Clogg,⁴ even where the lesion is single, the lumen beyond the occlusion is either blocked with inspissated mucus or so narrow that the passage of meconium along it is still prevented.

The third specimen, recorded by Leopold Hudson,⁵ was obtained *post mortem* from a boy aged 8, who died of peritonitis; until twelve months previous to his death the boy had been perfectly healthy and free from abdominal trouble. The specimen shows a segment of gut situated 38 inches above the ileo-caecal valve, so narrow for a length of three-quarters of an inch that it barely admitted a probe. This stenosed segment connected a hypertrophied and distended proximal portion of small intestine with a distal part apparently normal. A small perforation had occurred just above the narrow segment, causing a fatal result. The autopsy revealed an intussusception considered agonal in nature, formed by the invagination of the upper loop into the lower for a distance of approximately 2 inches.

Sir Arthur Keith⁶ collected and grouped specimens of constrictions and occlusions of the alimentary tract of congenital

or obscure origin found in the museums of the Royal College of Surgeons and various medical schools. The total number of specimens was 86: the jejunum or ileum was affected in 15 (those having an associated Meckel's diverticulum were not included). Sir John Bland-Sutton has found that these stenosing abnormalities of the ileum are associated with the obliteration of the stalk of the yolk sac, and are sometimes complicated by a persistent Meckel's diverticulum.

Keith also collected from the same museums a total of 95 cases of Meckel's diverticulum, in 27 of which marked constriction or narrowing was found in the neighbourhood of this abnormal structure. The constrictions were of two kinds—those due to pressure or traction of the mesentery of the diverticulum as it crossed the ileum, and those due to irregular growth or expansion of the Meckelian loop of the ileum, the last part of the ileum to be differentiated off from the yolk sac. Of the 95 preparations, intussusception was present in 3, but whether any of these were due to constriction, as distinct from the diverticulum, is not stated.

Meckel's diverticulum is said to be present in 2 per cent. of all individuals. Since the rarer specimens showing a secondary lesion are likely to have been preserved at the expense of the more common ones, it would appear that intussusception associated with Meckel's diverticulum is somewhat rare. In the collection of Perrin and Lindsay, enteric intussusceptions form less than 7 per cent. of the total, and Meckel's diverticulum is responsible for 5 of these.

Bland-Sutton has pointed out how often congenital constrictions are found where embryonic diverticula occur. In the duodenum the common congenital occlusion is caused by a septum just above the entry of the bile duct, and is probably associated with the outgrowth of the embryonic buds of the liver and pancreas. In this situation, therefore, it occurs at the junction of the two different morphological parts of the alimentary tract, the foregut and midgut. In the ileum, stricture occurs most commonly in close relation to the site of a Meckel's diverticulum. It is not easy to see how this explanation accounts for all the occlusions of the ileo-jejunal segment.

The following cases illustrate the varieties of deformity that may occur in that region:

Keith's series include 3 cases of multiple stenosis (in 2 the jejunum was affected, in 1 the ileum), 1 case of complete occlusion just above the ileo-caecal valve, and 1 case in which continuity was interrupted just at the valve. In the 6 other cases of jejunal stricture of his series the deformity varied from local narrowing to complete absence of a segment of gut and mesentery.

Rock Carling⁷ records a case of stricture just below the duodeno-jejunal flexure. Morley⁸ describes a case of occlusion at the ileo-caecal valve.

The theories advanced in explanation of the strictures have been admirably expounded by Clogg, but the more important points may be recalled.

1. Relation to Diverticula.

Meckel's diverticulum can, according to Leichtenstern, occur at varying points along the jejunum and ileum. He gives cases in which a single diverticulum has been found near the mid-point of the ileum, at the ileo-jejunal junction, and even in the jejunum. Crymble⁹ records a case of persistent vitelline duct connected with the appendix. Lamb¹⁰ records cases where diverticula were seen in the jejunum (14 cases) and in the duodenum (7 cases).

More recent authorities state, however, that many true diverticula in the upper part of the ileo-jejunal segment are associated with accessory pancreatic buds. The relation of these diverticula to stenoses and occlusions has not yet been worked out. It is possible that, as in the development of the normal pancreatic buds, conditions which permit the formation of septa may be present. However, I can find no reported cases of developmental septa or constrictions in relation to such diverticula.

2. Relation to Proliferation of Mucous Membrane.

Fandler,¹¹ who first pointed out that the duodenal lumen in the human embryo of 30 to 60 days is more or less completely obliterated by proliferated epithelium, suggests that a similar epithelial occlusion may exceptionally be found in all parts of the embryonic intestine. Keibel,¹² in an embryo of 11.5 mm., found that the epithelium of the lower part of the intestines blocked the lumen in two places, but did not observe similar conditions in other specimens.

Although occlusion by septa, and even narrowing of the gut, might be accounted for by the persistence of this

obliteration of the lumen, it is not easy to attribute absence of a segment or absence of the mesentery to this cause. The narrowing which occurs in the duodenum when a septum is present merely indicates that development has stopped at an early period, without explaining the cause.

3. Failure in Evolution of the Intestinal Tract.

The jejunum, ileum, and proximal part of the colon are all developed from that portion of the midgut which overlies the yolk sac in embryos up to the end of the third week. It is possible, therefore, that strictures are brought about by irregular closing of this portion, but if such irregularities occurred, congenital dilatation of this segment might be expected to exist more commonly. It seems more likely that strictures arise during the subsequent elongation of the midgut and hindgut. Certainly irregularities occur in this process.

In Abernethy's case of a boy aged 12 the small bowel was only 2 feet in length, while the colon measured 4 feet.

Sir Alexander MacCormick¹³ reports a still more remarkable case of a well developed man of 28 who had always had good health except for periodic attacks of vomiting. At an exploratory operation the stomach was found to fill the entire abdominal cavity, and to reach down to the pelvis. The small intestine was represented by a short segment 6 inches long, reaching from the duodeno-jejunal flexure to a caecum situated in the left hypochondriac region. No appendix was found, and a modified colon followed the course of the portion of the normal large intestine distal to the splenic flexure.

Failure in development of a small segment would give all varieties of stricture from mere narrowing to complete absence of a portion of gut with its mesentery. Certain factors have to be considered in relation to this lack of development.

(a) *Vascular Anomalies.*—Arrest of growth of the intestines, according to Jaboulay, depends upon vascular lesions—for example, embolism and thrombosis of the mesenteric vessels. It is easy to see that such lesions would cause such abnormalities, but proof of the statement is difficult. The same effect might be produced by congenital vascular defects, by dragging on the vessels by adhesions, or by pressure of tumours upon them. Davis and Poynter¹⁴ found small lumina and sclerosis in the arteries of the segments affected in a case of multiple atresia of the jejunum.

(b) *Peritonitis* has been widely accepted as a cause of congenital stricture. Tubercle and syphilis provide the majority of such cases, but other forms are said to exist. The focal effect necessary for the production of single stenosis (the 'commonest type') is unusual in any such infection of the peritoneum.

4. Mechanical Causes.

(a) *Blocking of the lumen* by means of mucous plugs has been described as a cause of intestinal obstruction in infants, sometimes in relation to narrowing of the lumen, sometimes with a normal lumen. Its occurrence is rare, and the condition has little or no claim to consideration as a general cause of stricture.

(b) *Adhesions* formed in development or as a result of peritonitis are said to cause congenital strictures, either by compression of the gut by bands or by dragging on the vessels of the mesentery. They may also initiate a volvulus.

(c) *Volvulus* is believed to account for some cases. Morley attributes his own case to this cause, and suggests that it may explain Waterston's¹⁵ case also.

It must be remembered that the intestines undergo a complete counter-clockwise turn in passing from the foetal to the adult position. The stalk of the vitelline duct has usually disappeared before rotation commences. If, however, involution of the duct fails, so that attachment to the umbilicus persists, the possibility of a clockwise strain at the point of attachment of Meckel's diverticulum must be borne in mind. A specimen of a Meckel's diverticulum retaining its umbilical attachment is illustrated by Evans.¹⁷ The same possibility is present whenever a peritoneal band attaches the foetal gut to the anterior abdominal wall at an early stage. The question arises as to whether a case of multiple congenital stricture may have arisen from multiple volvuli.

Volvulus, when present, is usually clockwise, but many turns are present. It may be that it is a continuation of the process started as above, but there is no proof of such an origin. It may equally well have arisen in a more haphazard way at an early stage, or the volvulus may be a secondary factor in a gut already the subject of atresia.

The theory that congenital constrictions arise from torsion might account for those cases in which there is irregularity

or absence of the mesentery; it does not, however, account for those specimens which show mere narrowing, but no suggestion of torsion, and no disturbance of an orderly vascular arrangement. It can only play an occasional part in the formation of intestinal abnormalities.

(d) *Intussusception* was undoubtedly present in the constricted portions found in the cases examined by Braun and Chiari,¹⁶ but again the normal anatomical arrangement of the vessels present in many cases of simple narrowing is against this as a universal cause.

In considering the recorded cases of congenital stricture of the ileo-jejunal segment two points are striking: First, that the segment may be affected in any part of its length by single or multiple constrictions, which, when considered as gross anatomical lesions, resemble one another closely, and in their difference in degree tend to form definite steps in a series. Secondly, that these apparently similar conditions have been credited with widely divergent origins.

The causes given above in the mechanical group do not apparently occur sufficiently often to disprove the possibility of their being associated lesions—secondary developments in portions of gut already malformed. It is difficult, therefore, to avoid the conclusion that there is some common primary etiological cause underlying all these lesions.

The two factors that would appear to account for the majority of these cases are: deficient evolution of the intestinal tract and excessive involution of the vitelline duct, but the details of the processes underlying these departures from the normal are at present little understood.

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OPTIC ATROPHY.*

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"OPTIC ATROPHY" is a term applied to a pale condition of the disc when some or all of the fibres of the visual path as they pass through the foramen sclera are degenerated. Pallor does not always mean atrophy, but when associated with a permanent defect in the visual field it probably represents atrophy. The cause of pallor is not always very obvious; in some instances it is determined by emptiness of capillaries at the disc; in the grey pallor of tabes it is difficult to say what the coloration is due to; in post-papillitic pallor the fibrous tissue elements decide the whiteness.

In a degeneration of the nerve such as is met with in tabes or disseminated sclerosis there is a wasting of the nerve elements due to constriction by the proliferation of the supporting structures with the minimum of inflammatory change. In this type of case the neurone is affected in the course of its axis-cylinder process. Should the neurone be affected at the cell level—that is, in the retina—the axis cylinder undergoes degeneration, which may produce a very similar picture as, for instance, in alcohol-tobacco combined poisoning.

A direct compression of the neurone between the disc and the lower visual centres (for example, pituitary enlargement) gives rise to a simple degeneration which takes place fairly rapidly towards the retina as well as towards the brain. A different picture results from a stasis at the nerve head, produced by pressure of cerebro-spinal fluid, such as occurs in brain tumour. Here there is much swelling of the supporting tissues; the pressure affects the neurone, but not for some time, and, although inflammatory products are not in evidence, the subsidence is attended by proliferative changes of the glial and endothelial elements; formation of fibrous tissue and compression of the nerve elements ensue. In neuritis the fluid in the subarachnoid space round the nerve may not be under increased pressure, but there may be an inflammatory condition of the meninges clothing the nerve and of the pial

septa with vasculitis and cellular changes. Here the subsequent proliferation is marked and perivascular proliferation leads subsequently to marked contraction of the vessels. Even in a simple occlusion of the central artery, where the neurone suffers from sudden deprivation of nutrition and death at the level of the retina, there is a perivascular proliferative change at the disc productive of fibrous tissue. The amount and distribution of the fibrous tissue give a clue to the cause of the atrophy, and the appearance of an atrophic disc is determined by the relative changes which take place in nerve element, interstitial tissue, and vessels respectively. It may be an advantage to think of the general causation under three heads:

1. Initial damage to the neurone itself (parenchymatous).
2. Initial damage to the supporting tissues, with or without oedema or inflammatory exudate, followed by secondary damage to the neurone (interstitial).
3. Initial interference with the blood supply with secondary damage to the neurone (vascular).

Of all the causes of atrophy, congested disc is chief. It is of two main distinct types (different in method of production but very similar in ophthalmoscopic appearance), frequently referred to as papillitis and papilloedema. Both are associated with redness and swelling; but the one is active, the other passive. If a localized inflammation takes place sufficiently far back along the nerve the disc may not show any change, but this is not very common. With these points in mind, I show a rough tabular grouping of different types of optic atrophy, giving in brackets some of the common terms used in the books to designate different types. There is, of course, much overlapping in such a grouping and it cannot be regarded as accurate, but from a clinical point of view it may be thought useful.

Types of Optic Atrophy.

1. Optic atrophy following direct damage to neurone by pressure or selective poisons, including subinflammatory increase in interstitial elements, excluding inflammation and oedema.
At the level of the retina: Methyl alcohol, alcohol, and tobacco; diabetes. (Axial atrophy; partial atrophy.)
At the level of the papilla and pars arteria centralis: Glaucoma; tabes, insular sclerosis. (Simple atrophy; spinal atrophy; primary atrophy; genuine atrophy; tabetic atrophy; grey atrophy; non-inflammatory atrophy.)
At the level of the orbito-cranial portion of nerve and tract: Pituitary diseases; insular sclerosis; tabes; fracture; hydrocephalus; carotid pressure. (Simple atrophy; secondary atrophy; descending atrophy.)
2. Optic atrophy following damage to neurone through post-inflammatory or post-oedematous change in supporting tissues.
At the level of the retina: Retinitis pigmentosa; retinitis (syphilitic) and choroiditis (renal). (Consecutive atrophy; ascending atrophy.)
At the level of the papilla and pars arteria centralis: Syphilis; renal disease; brain tumour; oxycephaly, etc. (Post-neuritic atrophy; post-papillitic atrophy; secondary atrophy.)
At the level of the orbito-cranial portion of nerve and tract: Acute true retrobulbar neuritis; periostitis; orbital cellulitis and abscess; nasal sinusitis; influenza; encephalitis; acute infective diseases. (Secondary atrophy.)
3. Optic atrophy following damage to neurone and supporting tissues by deprivation of blood supply.
At the level of the retina: Arterio-sclerosis (with and without high blood pressure; embolism of central artery; quinine. (Retinitic atrophy.)
At the level of the papilla and pars arteria centralis: Retrobulbar growth (?); haemorrhage into sheath; disease of central vessels (?).
At the level of the orbito-cranial portion of nerve and tract: Vascular disease.

The commonest variety of optic atrophy which we meet with in India is the post-neuritic atrophy of syphilis. Although a post-neuritic atrophy apart from the papilloedema and papillitis of cerebral tumour or meningitic disease may not be thought very common, in Madras there is no doubt that our most frequent atrophy is one which follows a mild insidious bilateral neuritis, which in no way resembles a papilloedema, inasmuch as all that can be seen during the neuritic stage is an almost flat dull-red disc with perivascular disc changes. Colonel Kirkpatrick laid stress on this variety during the period of his superintendency. In 284 cases of optic nerve diseases collected between 1914 and 1918, of which he took careful notes, syphilitic neuritis preceded this atrophy in by far the greatest number of cases. Of 58 cases of optic atrophy admitted to the wards in 1919 he states the bulk was syphilitic. The mildness of the neuritis and its insidious progress to a post-neuritic atrophy with a white

* Abstract of an address delivered to the Madras Branch of the British Medical Association on February 5th, 1923.