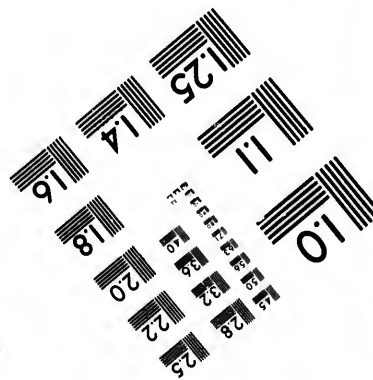
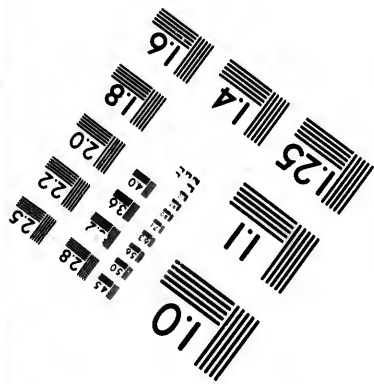
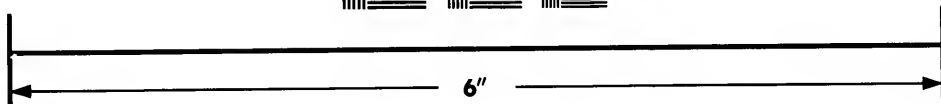
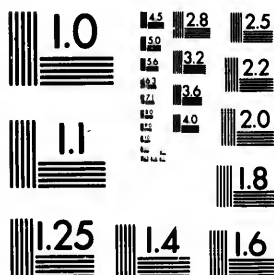


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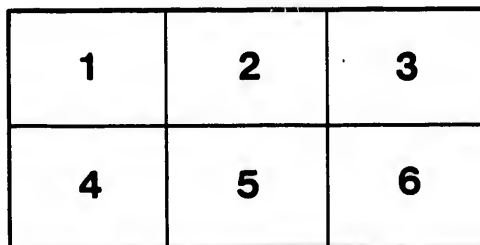
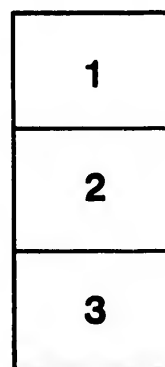
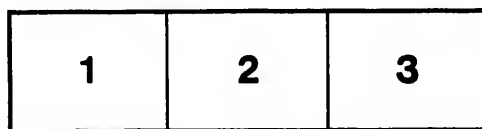
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ON  
SO-CALLED IDIOPATHIC DILATATION OF THE LARGE  
INTESTINE,<sup>1</sup>

BY

C. F. MARTIN, B.A., M.D.

Lecturer on Pathology, McGill University; Assistant Physician, Royal Victoria  
Hospital, Montreal.

Medical literature of the past three years contains a few remarkable cases of unexplained dilatation of the colon and sigmoid flexure, which are of unusual interest to the pathologist and clinician. The condition has, without a single exception, proved fatal, baffling the skill of physician and surgeon alike, and yet the failure of methods



hitherto employed, has stimulated suggestions in regard to treatment which may in future cases be of some benefit. A similar instance of the kind, which occurred a few months ago in the Royal Victoria Hospital, had, in addition, interesting features worthy of record in connection with the others of its kind so rarely seen by the clinician.

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<sup>1</sup> Read before the Montreal Medico-Chirurgical Society, November 20th, 1896.

It is interesting to note the marked similarity in the clinical histories of almost all the recorded cases. In nearly every instance there has been observed a more or less persistent constipation or other "trouble with the bowels" commencing within the first few days of life—necessitating the use of purgatives or enemata, which treatment is attended by varying success. Following upon this, comes either very early or within a few years, a noticeable abdominal distension—sometimes with frequent passage of flatus and progressively increasing constipation, resisting more and more the measures employed to evacuate the large intestines. Pain accompanies most of these cases and occasionally there are vomiting and intermittent liquid stools.

Examination of the patient reveals a distended tympanitic abdomen, with occasional localised lateral dullness from impacted faeces, and vermicular movements may often be seen. As a rule there is some pain on deep pressure. In every case, moreover, a rectal examination fails to reveal evidence of stricture, organic or otherwise, and there is never any appreciable obstruction to the insertion of catheters or syringes. It is true that in a few instances the clinician has suspected a spasm of the rectum and anus, but the observations made were never positive on this point. There is often progressive emaciation, though sometimes death is very sudden and unexpected, and the autopsy reveals no satisfactory reason therefor. The suggestion made by Peacock may, in a general way, be taken as satisfactory. "Death resulted from a disturbance of the vital powers in consequence of the mechanical injury of the intestine resulting from its extraordinary distension." The condition, moreover, is rarely associated with the usual symptoms of rachitis.

While as a rule the malady is fatal at an early age, it is nevertheless not incompatible with moderate health for many years, and cases which appear to have been congenital, have persisted for 28 years, as described by Peacock. Idiopathic dilatation of the colon may be present at birth, or appear later on in life, and in many of these latter cases the condition is regarded, though it would seem wrongly so, as congenital in nature. If a condition of dilated colon develop in adult life and the autopsy reveal no apparent cause, it is scarcely justifiable on this basis alone to regard the state as one undoubtedly congenital. Thus for example, in a case reported by Strahan, there was no disturbance of the bowels until adult life, and then within a few years a fatal issue resulted from colitis with dilatation, and no organic lesion was found to explain the origin of the trouble. The patient, however, was a lunatic, and, as was to be expected, disregarded entirely the necessary precautions to improve his intestinal condition, and hence constipation and

irregularities were present which could readily induce the existing abnormalities. Indeed, Van der Kolk, as noted by Hurd, found a slight dilatation of the colon so common in the insane, as to associate the conditions very intimately, and the cause is doubtless that above given. The mere fact that no organic obstruction was found is not sufficient in itself to make one regard the condition as congenital, particularly when no symptoms whatever develop until the patient attains adult life.

It is of course of prime importance to distinguish between purely congenital cases and those which have been acquired at a later date, for dilatation of the colon as a result of (or at all events following upon) koprostasis is by no means uncommon. Many instances are recorded of dilatation of the colon coming on late in life, but these are nearly all associated with some condition interfering directly with the evacuation of the bowels, and are obviously due to chronic constipation with or without constriction of the intestinal lumen. Such for example is the case recorded by Little and Callaway, and that above mentioned by Strahan, while in the same connection may be cited the interesting condition described by Mr. Gay, where as a result of atony of the bowel following typhoid fever, chronic constipation supervened and with it marked dilatation of the colon.

So far as the congenital cases are concerned, it is not an unfrequent circumstance that antenatal stenosis or imperforate condition of the anus will lead to dilatation of the large bowel, either diffusely or in the form of diverticula. Cases of this kind may show equally well a series of symptoms and morbid anatomy resembling those seen in the apparently idiopathic congenital dilatations. Many instances are recorded, such for example, as those by Vulpian, Lacave, Duplex and others; but all these are quite evidently not idiopathic in the true sense of that word. (We understand by the term "idiopathic" in reality a confession of our ignorance, or at all events our inability to find the true organic lesion associated with the morbid anatomical condition present. For if "nothing is that errs from law," there must be some etiological factor to explain every case of abnormal dilatation of the colon).

In endeavouring to differentiate between cases that are acquired and those which are purely congenital in nature, no small difficulty arises, and while some would apply the term congenital to those instances only where the intestine has never from birth acted normally, the definition, it would seem, is too restricting. Several cases for example exist in the literature where within the first few months of life this condition has arisen and the autopsy has



revealed no satisfactory explanation. Indeed, all gradations exist between the congenital idiopathic dilatation and that developing after several years.

Dr. Rolleston, in his interesting paper on this subject (to which I am indebted for several references), differentiates between acquired and congenital cases, admitting, however, the impossibility of absolute divisions. He includes under the latter group only those where the bowels have never acted naturally and distension appeared soon after birth, while all other cases are classed as acquired. In the statistical table, however, which is included in his article, this classification is scarcely adhered to, inasmuch as the case of Little and Callaway above mentioned, is regarded as congenital in origin, although the symptoms developed only late in adult life, and if such a case be regarded as antenatal, it would seem unjustifiable to exclude at least six or seven others where the condition has arisen even in earlier childhood, though perhaps not co-incident with birth. There can be no question that in the majority of these cases some mechanical hindrance to the evacuation of the large bowel is the main immediate etiological factor, and though these may frequently be antenatal, it could scarcely be regarded as associated with changes which have arisen in the earliest days of life in the embryo.

The cases of idiopathic dilatation of the colon or sigmoid which occur, might perhaps be classified as follows :

1. Those in which the symptoms occur at birth, either as constipation alone, or combined with distension of the abdomen (Rolleston's congenital cases). Of these there are very few on record, apparently only four cases about which there can be no doubt, viz. : those of Generisch, Osler, Hirschsprung and Peacock ; my own case is also to be herein included.

2. Those in which the symptoms develop shortly after birth (*i.e.*, within a few months), or where it is stated that "from earliest infancy" there was one or other sign present.

These cases are more numerous, and though they cannot be definitely called congenital, they so closely resemble them as to be practically identical, and are more than probably of congenital origin. These are recorded by Walker and Griffiths, Eisenhart, Hughes, Formad, Osler, Bristowe, Gee (2 cases) and Rolleston.

3. Those developing after several years and associated with no pathological lesion. Such is Gee's, subjoined, in which the symptoms appear to have commenced only after 4 years of age.

4. Those cases which occur only in adult life, which can likewise be explained only as idiopathic (*i.e.*, with no organic lesion to suggest a clue as to the cause). Of these, there are many cases too numerous to mention, *e.g.*, those of Herringham and Bruce Clarke, Little and Callaway, Hadden, Lewitt and many others.

The following table will explain these various cases and include a few details concerning each :

## CASES OF "IDIOPATHIC" ENLARGEMENT OF LARGE INTESTINE.

	Date	Sex	Age	Date of Onset	Earliest Symptoms	Early constip'n.	Operation.	Death.	Part affected
<i>Undoubted Congenital Cases.</i>									
Paçoek.....	1872	M.	28	At birth	Constipation.....	Yes.....	No.....	Sudden	Whole large bowel.
Hirschsprung.....	1882	M.	2 months	"	" and Dist'n.....	Yes.....	"	Yes.....	Chiefly transverse
Oster.....	1883	M.	7 "	"	"	"	"	In statu quo.	colon—rest less so.
Genesich.....	1893	M.	11 years	"	"	"	"	Yes.....	Colon.
Author's	1886	M.	3 $\frac{1}{2}$ "	"	Constipation.....	"	Two	Yes.....	Sigmoid chiefly.
<i>Probable Congenital Cases.</i>									
Walker and Griffiths.....	1893	M.	11 years	A few weeks after birth	Distension.....	No.....	No.....	Yes.....	Colon and sigmoid.
Hughes.....	1887	M.	3 "	Early infancy	Constipation.....	Yes.....	"	"	Sigmoid chiefly.
Formad.....	1892	M.	23 "	Before 1 $\frac{1}{2}$ years	Irregular bowels—slight abdominal enlargement.	Irregular	Exploratory only	Sudden	Colon mainly.
Gee.....	1884	M.	4 $\frac{1}{2}$ "	3 months	Constipation.....	Yes.....	No.....	Yes.....	Sigmoid.
Gee.....	1884	M.	4 "	1 month	Distension.....	"	"	"	Sigmoid and colon.
Bolleston and Haward.....	1896	M.	12 "	2 months	"	"	"	"	Colon and sigmoid.
Oster.....	1883	M.	10 "	"Early in life"	" and cons'p'n	"	Yes.....	"Recovery for 2 years	Sigmoid mainly.
Bristove.....	1885	F.	8 "	"Long time constipated"	Constipation.....	"	No.....	Yes.....	Sigmoid and colon.
Hidden.....	1893	M.	11 weeks	"	"	"	Yes.....	Yes.....	Small and large intes.
<i>Dubious Cases.</i>									
Gee.....	1884	M.	5 $\frac{1}{2}$ years	4 $\frac{1}{2}$ years	Distension.....	Yes.....	No.....	Yes.....	Sigmoid and colon.
Lewitt.....	1867	M.	21 "	12 (9) years	Constipation.....	At 12 yrs.	"	"	Colon and sigmoid.
Eisenhart.....	1894	F.	35 "	Always constip'd.	"	"	"	"	Sigmoid.
Strahan.....	1893	M.	32 "	17 years	Sudden pain and distension.	No.....	"	"	Sigmoid and colon.
Little and Callaway.....	1850	M.	34 "	(?)	Distension and constipation marked at 33 years.	No.....	Puncture	Yes.....	Sigmoid and colon.

\* Operated on two years later for obstruction and died.

As will be observed from the above table, the cases which have certainly arisen coincident with birth or before it are but five in number, while at least eight are recorded where the onset of the symptoms appeared so soon after birth that it is difficult to ascribe to these an origin different from the five undoubted congenital cases. In several instances the conditions are so similar in all respects that one is scarcely justified in regarding them in the light of non-congenital (*i. e.* acquired) cases in contradistinction to the five instances mentioned.

So far as constipation alone is concerned, it is difficult to believe that this in itself is the cause of the malady, but it is much more probable that the gas developed in the intestine would equally well induce a kinking and functional closure of the intestines, particularly when with a lax meso-colon the bowel may become twisted. The cause is obviously purely mechanical, and while numerous theories have been advanced in practice, none have proved entirely satisfactory as applying to every case on record.

Among the most commonly cited of these are the following:

1. An unduly lax meso-colon, by means of which there arise kinks in the bowel, consequent constipation, and gradually increasing distension.
2. Immoderate or anomalous development of the tissues of the sigmoid flexure or colon.
3. Undue length of the sigmoid flexure in early infancy, combined with habitual constipation.
4. Defective innervation of the intestinal muscles.
5. Spasm of the rectum.
6. Adhesions.
7. Colitis, and hence weakened intestinal wall.
8. Contracted meso-colon at one place.

#### CASE REPORT.

Without discussing at the present time the value of these different theories, I will now pass on to describe the case that has come under my notice, after which it will be possible to discuss which of these theories best accord with the conditions therein recognized.

For the notes of the clinical history I am indebted to Dr. H. S. Shaw, senior resident surgeon.

W. E., aged 3½ years, a male Canadian child, was admitted on January 3rd, 1896, to the surgical wards of the Royal Victoria Hospital on account of persistent constipation. The history given by the mother was briefly as follows: At birth the patient seemed to be

well in all respects except that the bowels did not move for five days. A purgative was then administered with good effect. During the first part of his life, the child was constantly constipated, sometimes for as long a period as eleven days. This necessitated the periodical administration of glycerine suppositories and castor oil, which generally produced the desired effect, flatus also passing frequently and freely. For the first year no enlargement of the abdomen was observed; the child suffered no pain and was in all respects a quiet baby. At the end of the first year, however, gradual and progressive enlargement of the abdomen supervened until the child had attained the age of  $3\frac{1}{2}$  years. From time to time the abdominal distension lessened, and flatus passed freely, but the bowels were inactive unless purgatives or injections were persisted in. Pain was observed merely a few months before admission to the hospital, and was then associated only with defecation or injections. The stools presented at first large scyballous masses, and, later, semi-solid evacuations, with two intermitting attacks of diarrhoea.

At the end of December, 1895, the constipation became alarming and no flatus was passed; the distension and pain became more marked than ever before, while purgatives and enemata seemed useless; friction over the abdomen proved equally futile. Two days before admission, however, a large evacuation was obtained, and, with it, a large amount of flatus was passed.

The *personal history* was negative.

The *family history* was of considerable interest in one respect, namely, that one other child of the family had been observed at birth to present an unusually large abdomen, and likewise to require enemata and purgatives from time to time. This child lived but three months, with this condition unaltered, the fatal issue being brought about by an acute broncho-pneumonia. No autopsy had been made.

*Condition on admission*—The child is pale, fairly well nourished, sleeps well, and suffers but little pain, except at stool. The tongue is clean, the appetite good, and the bowels obstinately constipated.

The abdomen is very greatly distended, having a girth of 68 cm., and is uniformly enlarged. From the ensiform cartilage to the pubes the measurement is 33 cm. Vermicular movements are distinctly seen over the lower half of the abdomen and apparently associated with the distended colon. This part of the bowel presents as an elongated prominence, running obliquely across the abdomen, and varying in position from time to time, and particularly with the vermicular movements. The movements are both spontaneous and easily excited by examination. On palpation there is marked gurgling; the

abdomen is soft, unresistant to the touch, and there is no tenderness. Fluctuation is sometimes elicited in the flanks, but seems to vary greatly and is probably associated with fluid in the intestines. Percussion yields a tympanitic note throughout, except for a modified dulness on each side low down.

The hepatic and splenic dulness are normal. On auscultation there are borborygmi heard, but otherwise no abnormality. The rectum showed no signs of disease nor stricture. The urine is normal. In the chest there is no evidence of disease, beyond that induced by the pressure upon the diaphragm from below. An operation was performed by Dr. Bell on January 16th, and the abdominal cavity opened in the median line, giving exit to a small quantity of serous fluid. The distended bowel was found to consist of sigmoid flexure, whose diameter was 9—10 cm. There was elsewhere no signs of collapse of the bowel or stricture, nor could any obstruction be detected. The rectum was examined again, but found perfectly free, and the abdomen was therefore closed and nothing further done.

The patient recovered well, and the bowels moved by means of enemata and the distension was markedly diminished, though the girth was never less than 55 cm. The condition, however, otherwise remained in *statu quo*, so a second operation was performed on January 30th, and an incision made in the left inguinal region. Bougies were introduced per rectum to empty the large bowel, but failed to move some impacted feces. Accordingly large trochars were employed and the bowel punctured in the abdominal cavity, and semi-solid feces were then pushed out of the dilated sigmoid flexure, and the wound thereafter sutured in the usual manner.

For five days the patient did well, but on the sixth day symptoms of perforative peritonitis developed and death ensued.

The *autopsy* was performed 17 hours after death and the following notes from the report are abstracted concerning the conditions of the abdominal cavity :

The operation wounds are both almost entirely healed. On opening the abdominal cavity fetid gas escapes, and about 400 cc. of creamy, greyish fluid having a faecal odour is removed. It is for the most part free, but in portions is sacculated off by the recent plastic adhesions in the peritoneum. There is a very recent general peritonitis ; the omentum for the most part covers the small intestines and is adherent to the lower end of the sigmoid flexure. The visible intestines are covered with a plastic exudate, and the coils are loosely adherent to each other. The lower half of the abdomen is occupied by the enormously distended and hypertrophied sigmoid flexure, which

lies completely across the abdominal cavity with its concave border looking to the left. The upper half of the loop is the wider and longer, measuring 20 cm. x 8 cm., while the lower half is 15 cm. long x 5 cm. in diameter.

There are fairly loose adhesions between the two portions of the loop, as also between the end of the sigmoid flexure and the left abdominal incision. There seems to be a thickening of that part of the meso-colon which approximates the two ends of the loop and causes traction on the lower end, thus creating a partial narrowing of the lower portion. The sigmoid immediately above this narrowed part is dilated with a pouch formation on its inferior surface, evidently caused by gravitation of accumulated faeces.

On the outer and lower surface of the sigmoid are a number of Lembert sutures to which the omentum is adherent. Union is complete, except at one median point where a small perforation, 2 mm. in diameter, indicates the evident origin of the peritonitis.

On opening the sigmoid, solid faeces weighing 850 grms. are removed, the wall is greatly thickened, measuring on an average 5 mm. The mucosa itself is normal without any ulceration. The rectum is of about normal size and thickness, and there is nowhere any evidence of constriction.

The descending colon is also greatly thickened, somewhat dilated and its mucosa congested. Elsewhere the large intestine shows but slight hypertrophy and no ulceration. The total length of the colon from caecum to sigmoid flexure is 50 cm. The sigmoid itself measures 38 cm. The caecum, appendix and small intestines appear normal in all respects except for the plastic peritonitis on their serous coats. So far as the other organs and structures are concerned, there was nothing of importance detected.

The interest in the above case then concerns the same features as those already observed in the first four cases found in the above table as being undoubtedly congenital in origin.

The condition is most like that described by Curschmann in his article on topographical clinical studies, where he specially notes that in early life the sigmoid is relatively larger than the rest of the large intestine and that we often find a persistence of the infantile condition. This he describes as often monstrous, and records 15 cases out of 233 examined. From his observations it would seem that the sigmoid is very prone to congenital volvulus, especially when there is a marked approximation of the two ends of the limbs of the loop. The meso-colon thus becomes fan-shaped, the widest portion of the fan

corresponding to the loop within, the narrowest part being where the two ends of the loop are approximated (as in the figure). This portion, according to Curschmann, is nearly always more opaque and thick than other portions, because of chronic inflammation, hence retraction, chronic constipation and dilatation of the intestine above. It is worth noting in this connection that Jacobi in his clinical lectures on pædiatrics likewise considers this antenatal condition of the sigmoid flexure as an important factor in the production of infantile habitual constipation.

The *diagnosis* rests upon the combination of symptoms and signs already referred to, the main features being :

1. Constipation in early infancy, sometimes with intermittent diarrhoea.
2. Early distension of the abdomen with exacerbations and remissions according to the success in evacuating the flatus and intestinal contents.
3. Evidence of a dilated colon from physical examination.
4. Absence of any constriction at the anus or in the rectum. Sometimes emaciation.
5. Pain and tenderness, not usually marked, except when copious enemata are employed.

Rolleston has pointed out that the condition is much more common in males, a fact confirmed by the additional cases from the literature which have been included in the above table.

The *prognosis*, as indicated above, is most grave, not a single case being permanently cured. The case under Dr. Osler, which was operated upon and an artificial anus created in the dilated sigmoid, remained well for nearly two years, but it appears from references made to this by Dr. Rotch, that the symptoms again returned and the patient shortly afterwards died, a second operation being performed. Sometimes death is very sudden and the autopsy fails to explain the reason. As a rule, however, gradually increasing constipation and distension prove so serious that nutrition fails, and either a fatal peritonitis ensues, or the patient dies from what was referred to above as "disturbed vitality from the mechanical injury."

The *Treatment* in all the cases has proved most unsatisfactory and the usual means of purgation are believed by most authorities to only aggravate the existing conditions. In the present instance it is certain that the enemata increased the child's sufferings, rendered it much more nervous and did little to give more than very temporary relief. Purgatives were equally useless. Massage was employed by the child's father but was likewise found futile; in fact Rolleston em-

phasizes the danger of such a method and the liability of rupture of the bowel where ulcers have existed.

All operative measures have hitherto been unsuccessful and the various incisions and punctures produced relief for but a short time and death resulted either from shock or peritonitis. But the rationale of an operation performed upon the enlarged intestine itself, may well be questioned inasmuch as the paralysed condition of the muscle wall would render the evacuation of the bowels still difficult, and it is evidently for this reason that Rolleston has made what seems an admirable suggestion viz :—To open the lower end of the small bowel where there will be no obstruction to the out-flow of fæces and by this means to relieve the distention and accompanying symptoms.

Where any kink has formed or where there is contraction of the meso-colon one might expect by relieving these conditions to obtain a good result only in those cases where the dilatation had not become excessive, otherwise it would seem that the diseased condition induced by the stretching and hypertrophy would be irremediable.

Where indeed very much hypertrophy and distention have occurred and the symptoms are in no way relieved, it may truly be said that the large intestine where affected is to all intents and purposes a foreign body and it may therefore be reasonably considered whether total extirpation of the affected portion is not commendable and whether an operation for intestinal anastomosis could not easily be performed between the two healthy ends remaining. This could be attempted either between the unaffected colon and the rectum, or if the whole upper colon be involved an anastomosis could be accomplished between the lower end of the ileum and the remaining portion of the healthy rectum. In this way fæces would have a ready escape and the sphincter action of the rectum and anus would remain intact. This could be done either by means of an end to end anastomosis, or, as my friend Dr. A. E. Garrow has suggested, better still by a lateral anastomosis which would thus obviate the liability to intussusception and prolapse.

My best thanks are due to Prof. Adami for the appended drawing, and to Dr. Patrick for the accompanying photograph.

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