THE HUNTERIAN LECTURES ON THE PATHOLOGY AND SURGERY OF INTUSSUSCEPTION.

Delivered at the Royal College of Surgeons of England.

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[ABSTRACT.]¹

LECTURE I.—SOME POINTS IN THE MINUTE ANATOMY OF INTUSSUSCEPTION.

[MR. D'ARCY POWER began his course of lectures with an apology for selecting so time-worn a subject as intussusception, but he justified his choice by saying that it was one which had occupied the thoughts of John Hunter, the great master in pathology, after whom the chair he occupied was named. Very little seemed to be known about the minute anatomical changes occurring in intussusception, though the various museums attached to the medical schools and hospitals throughout the country contained an abundance of material for its study.

The earliest changes were shown in sections from the intestine of a child, aged 8 months, upon whom the lecturer had performed an operation successfully 13½ hours after the appearance of the first symptoms of intussusception. The child died a few days later, with symptoms of paralysis of the bowel, but without peritonitis. The mucous membrane of the invaginated colon was healthy except for an increased number of goblet cells in its crypts of Lieberkühn. Much blood had been extravasated into the submucous tissue, though no clotting had occurred. The circular layer of muscle was healthy, but the longitudinal layer was oedematous, and was separated from the serous coat by many large venous and lymphatic channels.

The next section was taken from an experimental invagination made in a cat a week previously. The histological changes were again more marked in the mucous and submucous layers than in the muscular coat, but the extravasation of blood was chiefly in the mucous membrane whilst the swelling of the submucous tissue was due to oedema. The sections from the experimental invagination in a cat were compared with a spontaneous intussusception which had killed a dog. The extravasation of blood in the dog's intestine had done serious injury to the mucous membrane and to the submucous tissue, but the chief alteration in its structure had taken place in the muscular coat, where the circular fibres were separated by the oedema and had degenerated.

Some of the later changes in intussusception were illustrated by sections taken from John Hunter's own specimen, which was particularly well preserved, because at the time the patient died spirit was so cheap and strong that its retailers used to advertise that their customers might be "drunk for a penny, dead drunk for twopence, and straw to lie upon for nothing." Hunter's specimen, which led him to write his celebrated paper upon "Introsusception," was taken from the body of a child who died sixty hours after the first symptoms of illness had been recognised. Microscopical examination showed that the mucous membrane of the intestine was healthy, but that parts of its surface were covered with blood clot. The whole of the submucous tissue was engorged and oedematous. The circular layer of muscle was more injured than the longitudinal layer, whilst the mesocæcum was thickened by bands of fibrin. A lymphatic gland which had been involved in the invagination was congested.

The latest changes occurring in intussusception were shown in sections taken through the ilea of children in whom the submucous and serous coats of the intestine were suppurating whilst the mucous and muscular layers were comparatively healthy.

The changes which take place in the intestines of adults

as a result of intussusception were next shown to be identical in some points with those occurring in the more delicate tissues of children, though in other points they differed widely. The frequent association of adult intussusception with polypi and other growths in the intestinal canal led to the changes being of a more chronic nature. The villi of the mucous membrane, therefore, became converted into mere tags of connective tissue, but the extravasation of blood into the submucous tissue remained a marked feature in the invaginated bowel. In a case of chronic intussusception taken from the body of a man, aged 27, the submucous and muscular layers had become converted into dense fibrous tissue, whilst the villi were congested but had not undergone any mummifying change into connective tissue.

Sections from portions of bowel which had been exfoliated, with and without the recovery of the patient, were then exhibited. It appeared that the intestine occasionally underwent a sclerosing process before it was cast off, but that more frequently it had undergone so much tryptic digestion as to leave only a skeleton of connective tissue (reticulin) which was still sufficiently coherent to be passed as a complete cylinder. An examination of the scarred intestine at the point of separation showed that the mucous membrane repaired itself, and that the submucous tissue became continuous with the serous coat by layers of scar tissue which ran through the interrupted muscular coat. The exact method of repair in these cases required more careful examination, for the specimens which had been examined were not sufficiently well preserved to allow a satisfactory microscopical examination to be made.

The rarer forms of intussusception were then considered, and sections were shown from a case of ascending and retrograde intussusception occurring in the same patient. It was clear that in this instance the descending or ordinary form of invagination was the primary one, whilst the retrograde invagination had been produced much later, though it was not of the "agonic" type. Sections were then shown from a polypoid tumour associated with an intussusception occurring in a woman. The tumour proved to be a sarcoma, but the intestinal villi contained numerous oval bodies which seemed to be thrombosed vessels. The thrombosis was limited to the villi, for the blood vessels in the deeper part of the mucous membrane and in the submucous tissue contained ordinary *post-mortem* clots.

Mr. Power concluded his lecture, which was illustrated throughout with microphotographs, by an allusion to Dr. Vierhuff's and Dr. Sutherland's cases of intussusception occurring in the course of attacks of purpura. He also pointed out the various ways in which a Meckel's diverticulum, whether it was open or closed, might cause the rarest forms of intussusception; and he showed a specimen in which an obscure case of intussusception in an old person was proved by microscopic examination to be associated with carcinoma, that most usual cause of invagination in late adult life.]

LECTURE II.—THE PATHOLOGY OF INTUSSUSCEPTION.

In the former lecture, Sir, we ascertained some of the minute structural changes which take place in invaginated portions of the intestine, and we were then on the firm ground of fact. In the present lecture I propose to consider some points in the pathology of intussusception, and we shall find that we are almost at once driven to theorise. Every advance in pathology must be made by combining the results obtained from a consideration of anatomical, physiological, and clinical facts, for the science is the mistress of all these its handmaidens. It will therefore be necessary to follow intussusception along these lines, and afterwards to draw certain conclusions from the premisses thus obtained.

ANATOMICAL.

It behoves us to make a careful anatomical examination of the ileo-cæcal portion of the alimentary canal, because 40 to 60 per cent. of all the recorded cases of intussusception are said to occur at this part of the intestine. My work, therefore, has been limited to the ileo-colic angle, and I have been very greatly assisted by Dr. Pickard and Dr. Hayne, successively the senior resident medical officers at the Victoria Hospital for Children. These gentlemen have given me

[1885]

¹ The full text of this lecture will be published in the forthcoming number of the *Journal of Pathology and Bacteriology*.

Table of Measurements at the Ileocolic Angle.

Number.	Age.	Sex.	Type of Cæcum.	Length of Mesentery.	Diameter of Ileum, mm.	Diameter of Colon, mm.	Breadth and Direction of Ileocaecal Slit (mm.)	Direction of Axis of Ileum.	Arrangement of Peritoneal Folds and Mesenteric Glands.
1	Fœtus near term.	—	A.	—	6	6	—	Upwards	{ Figured and measured by Professor Struthers, <i>Edinburgh Medical Journal</i> , October, 1893 (Figs. 1 and 2).
2	"	—	—	—	9	12	—	Upwards	
3	5 days	F.	E.	—	11	12	8, horizontal	Horizontal	A well marked ileocolic fossa; wide ileocaecal pouch.
4	5 weeks	M.	C.	38	11	16	8, horizontal	Horizontal	Shallow ileocolic fossa; no ileocaecal pouch. Many small glands in ileac mesentery.
5	8 weeks	F.	D.	46	13	20	12, horizontal	Upwards	Deep ileocolic fossa; no ileocaecal pouch. A collar of lymphatic glands in ileocolic fold (Fig. 6).
6	15 weeks	F.	A.	47	15	19	10, puckered	Curved downwards	Good ileocolic fossa; wide and deep ileocaecal pouch. One large gland in ileocolic fold (Fig. 4).
7	5 months	M.	C.	37	13	22	7, horizontal	Upwards	No ileocolic fossa; ileocaecal pouch is behind and above instead of behind and below, as is usual. Capacity of colon 10 ounces.
8	5 months	M.	A.	34	15	25	10, oval	Horizontal	Very deep ileocolic fossa; no ileocaecal pouch. Glands in ileocolic fold and in ileocolic mesentery.
9	6 months	F.	E.	50	14	20	10, horizontal	Horizontal	Well-marked ileocolic fossa; deep ileocaecal pouch, with crescentic edge. One gland in ileocolic mesentery.
10	6 months	M.	A.	—	14	25	8, gaping	Upwards	—
11	7 months	M.	E.	41	14	20	10, oval	Horizontal	Shallow ileocolic fossa; deep ileocaecal pouch. Glands in ileac mesentery and one in ileocolic fold over colon.
12	7 months	F.	A.	50	18	22	8, horizontal	Horizontal	Deep ileocolic fossa; very deep ileocaecal pouch with thin crescentic edge. Capacity of colon 40 ounces.
13	9 months	M.	E.	47	11	15	8, circular	Curved downwards	Shallow ileocolic fossa; no ileocaecal pouch. One lymphatic gland in ileocolic fold.
14	9 months	M.	D.	55	13	22	7, oval	Upwards	Deep ileocolic fossa; very shallow ileocaecal pouch. Lymphatic glands few and small.
15	9 months	M.	A.	44	15	24	12, horizontal	Horizontal	Deep ileocolic fossa; well-marked ileocaecal pouch. A large mass of glands in ileocolic mesentery.
16	11 months	M.	A.	47	16	20	9, semicircular	Upwards	No ileocolic fossa; no ileocaecal pouch. One large gland in ileocolic mesentery.
17	11 months	M.	E.	50	14	26	9, horizontal	Horizontal	Shallow ileocolic fossa; very large and deep subcaecal pouch. One lymphatic gland in ileocolic mesentery.
18	15 months	M.	A.	56	15	25	9, horizontal	Horizontal	No ileocolic fossa; ileocaecal pouch narrow and deep. No glands.
19	16 months	M.	A.	38	11	15	6, circular	Horizontal	Shallow ileocolic fossa; deep ileocaeca pouch with wide mouth. Glands in ileocolic mesentery; none in ileocolic fold.
20	16 months	F.	D.	38	22	29	8, horizontal	Upwards	No ileocolic fossa; shallow and wide ileocaecal pouch. A chain of lymphatic glands lies parallel with the colon.
21	18 months	M.	A.	—	17	20	9, horizontal	Semicircular downwards	Shallow ileocolic fossa; deep ileocaecal pouch opening far forwards beneath ileum. Glands of ileocolic junction.
22	23 months	M.	A.	63	14	21	6, very puckered	Vertically upwards	No ileocolic fossa; ileocaecal pouch wide and shallow. Two glands in ileocolic mesentery. Cæcum sharply bent upon itself to pass behind the ileum (Fig. 2).
23	2 years	M.	E.	45	24	28	12, horizontal	Horizontal	Shallow ileocolic fossa; well-marked and wide ileocaecal pouch. Glands in ileocolic mesentery and fold.
24	2½ years	F.	A.	45	19	32	9, horizontal	Curves upwards	Deep ileocolic fossa; deep and wide ileocaecal pouch. Large gland in mesentery; none in ileocolic fold.
25	2½ years	M.	A.	—	15	26	13, horizontal	—	Shallow ileocolic fossa; deep ileocaecal pouch. Glands in ileocolic fold.
26	2½ years	F.	E.	—	16	25	11, horizontal	Horizontal	Shallow ileocolic fossa; deep ileocaecal pouch, with small opening. Glands in mesentery at a distance from ileocolic fold.
27	2½ years	M.	A.	53	15	30	15, horizontal	Convex upwards	Shallow ileocolic fossa. Mesentery of appendix is derived from lower border of ileum. It is double, and forms a very definite pouch ½ inch in depth. No glands in ileocolic junction; large one in mesentery (Fig. 3).
28	3 years	M.	A.	46	17	30	8, vertical	Upwards	Shallow ileocolic fossa; very deep ileocaecal pouch. Glands greatly enlarged; ileocolic fold thickened.
29	3½ years	M.	A.	50	16	23	12, oval	Horizontal	Shallow ileocolic fossa; wide and deep ileocaecal pouch. Many lymphatic glands.
30	4 years	F.	A.	—	15	42	17, horizontal	Horizontal	Good ileocolic fossa; ileocaecal pouch very deep, with pinhole opening. Glands in ileocolic fold and in mesentery. Ileocaecal valve admits tip of little finger.
31	4 years	M.	B.	—	16	20	9, oval	Upwards	Shallow ileocolic fossa; deep ileocaecal pouch, with a thin edge. Glands in mesentery; none in ileocolic fold.
32	4 years	M.	A.	57	18	30	18, horizontal	Upwards	Shallow ileocolic fossa; very complex ileocaecal pouch. Two large glands at ileocolic angle with a few smaller ones. Intestinal wall thick and muscular. Died of colocolic intussusception.
33	4½ years	F.	A.	45	20	35	22, horizontal	Upwards	No ileocolic fossa; no ileocaecal pouch. No glands.
34	5 years	F.	—	100	16	28	—	—	—
35	6 years	M.	D.	—	18	29	—	—	—
36	9 years	F.	A.	50	17	33	15, horizontal	Upwards	Fair ileocolic fossa; shallow and wide ileocaecal pouch. Glands in mesentery at a distance from ileocolic junction.
37	9 years	F.	E.	57	17	45	18, horizontal	Upwards	Well-formed ileocolic fossa; very deep ileocaecal pouch, with minute orifice. Several glands in mesentery, and one in ileocolic collar.
38	9 years	M.	A.	—	26	40	20, horizontal	Horizontal	Shallow ileocolic fossa; deep and complex ileocaecal pouches. No glands.
39	10 years	F.	C.	81	23	63	10, circular	Horizontal	No mesenteric attachment to upper border of ileum. Ileocolic junction concealed by the cæcum. Ileocolic fossa deep. No ileocaecal pouch. Capacity of large intestine 70 ounces.
40	11 years	F.	A.	58	15	47	21, horizontal	Horizontal	A singularly deep ileocolic fossa; no ileocaecal fossa. Glands in mesentery only (Fig. 1).
41	13 years	M.	E.	50	16	34	20, horizontal	Upwards	No ileocolic fossa; no ileocaecal pouch. One gland at junction of ileocolic in the ileocaecal fold.
42	14 years	F.	E.	100	17	42	19, horizontal	Horizontal	Shallow ileocolic fold; complex ileocaecal fossa. Large gland in mesentery. Capacity of large intestine 40 ounces.
43	14 years	F.	D.	82	22	87	24, horizontal	Horizontal	Very deep and complete ileocolic fossa; no ileocaecal pouch. Ileocaecal valve easily admits the forefinger (Fig. 5).
44	15½ years	F.	A.	72	20	49	14, horizontal	Horizontal	No ileocolic fossa; large and deep ileocaecal fossa, with shallow subcaecal fossa. Very few lymphatic glands.

many opportunities to examine the ileo-cæcal region in the bodies of children from a few days old up to the age of 15 years. The results of our observations are set out in the preceding table.

It should be stated that the mesentery was measured as soon as the body was opened, but the measurements are only approximately correct, because it is difficult to obtain constant points from which to measure and because its tissue is very easily stretched. All the other measurements were made when the specimen had been hardened for twenty-four hours in Müller's or in Foa's solution; and afterwards in 30, 60, and 90 per cent. alcohol, remaining in each strength for twenty-four hours. I believe that by adopting so uniform a method of hardening more accurate results have been obtained for purposes of comparison than could have been gained by measuring the fresh intestines, a proceeding which is difficult on account of the extensibility of their walls. The actual width of the intestines seemed to be unimportant, as I was only interested in their relative diameters. The mesentery in each case was measured at a point in the colon 2 inches away from the top of the ileo-colic junction; the ileum was measured at the same distance above it, and the colon at the same distance below it. All the measurements are expressed in terms of millimetres for the sake of ease and accuracy.

a. THE MESENTERY.

The length of the mesentery is a matter of interest in considering the pathology of intussusception, because the size of the invagination depends materially upon the length of the mesentery or upon its extensibility. Time does not allow me to consider the matter in detail, but it is obvious from a consideration of the table that the length of the mesentery is singularly constant in all the specimens measured; and as the age of the individuals varied considerably, this means that its proportion to the length of the body was much greater in infants than in older children. Increased length of mesentery is necessarily associated, other things being equal, with an increased range of movement in the intestine. The intestines should therefore be capable of a greater range of movement in children than in adults. This result is confirmed by the practical experience of all surgeons who have had to open the abdomen for the relief of intussusception in children, for the intestines are nearly always so freely movable that it is easy to bring the tumour into the wound. A few cases are recorded, however, in which the bowel seems to have been anchored with undue firmness.

Careful examination of the ileo-colic mesentery shows that the ileo-colic artery is always present, running parallel with the ileum and sending off numerous branches to it and to the lymphatic glands in its neighbourhood. These glands vary greatly in their size and distribution; sometimes they lie parallel to the colon, sometimes parallel to the ileum, and sometimes they have no orderly arrangement. They are discrete and seed-like in many specimens, in others they are aggregated to form a mass of lymphoid tissue which often forms a collar or band thickening the edge of the anterior ileo-colic fold, whilst in children who die of tuberculous disease they are characteristically enlarged.

Treitz,² Lockwood,³ and others have drawn attention to a suspensory muscle of the duodenum and mesentery. It springs from a band forming the right side of the oesophageal opening in the diaphragm, and is prolonged onwards with the superior mesenteric artery into the mesentery. The presence of such a band of muscular fibres might prove an important factor in the causation of intussusception if it extended as far as the ileo-colic junction, by hindering the movement of one border of the intestine. I therefore took the trouble to stain and make a microscopical examination of a piece of the ileo-colic mesentery in each of the specimens, but in none was I able to find a single bundle of muscular fibres.

b. THE ILEO-COLIC ANGLE.

The point of union of the small intestine with the large intestine next engaged my attention. The axis of the ileum at this point was horizontal (Fig. 1) in 20 cases; in 15 it sloped upwards to open into the large intestine (Fig. 2); in 3 it was curved concavely downwards (Fig. 3), and in one it was

Fig. 1.—A simple but complete form of ileo-colic angle from a girl, aged 11 years. The ileo-colic fossa is well formed, and the axis of the ileum is almost horizontal. (a) The ileo-colic fold. (b) The ileo-cæcal fold.

Fig. 2.—The ileo-colic angle of a child, aged 23 months. The ileo-colic fossa is absent, and the axis of the ileum is directed upwards to end in the large intestine.

Fig. 3.—The ileo-colic angle from a child, aged 2 years and 8 months. The ileo-colic fossa (a) is incomplete, but there is a very large and deep ileo-cæcal fossa (b). The axis of the ileum is concave downwards.

curved convexly downwards (Fig. 4). The actual line of union of the ileum with the colon is hidden both in front and behind by folds of the peritoneum, whose arrangement has been made familiar to us by the works of Mr. Treves,⁴ Dr. Rolleston and Mr. Lockwood,⁵ in England, and of Dr. Pérignon.⁶

² *Vierteiljahrsschr. f. d. prakt. Heilkunde*, Prag, vol. i, 1853, p. 113.

³ *Hunterian Lectures on Hernia*, London, 1889, p. 24.

⁴ *The Anatomy of the Intestinal Canal and Peritoneum in Man*, 1885.

⁵ *Journ. of Anat. and Physiol.*, xxvi, 1892, p. 130.

⁶ *Thèse de Paris*, 1892, No. 4.

Fig. 4.—The ileo-colic angle of a child, aged 15 weeks. The ileo-colic fossa is incomplete, and the axis of the ileum is convex downwards.

Fig. 5.—The ileo-colic angle of a girl, aged 14 years. The ileo-colic fold (a), and the ileo-cæcal fold (b) are unusually well developed. The axis of the ileum is nearly horizontal.

Fig. 6.—The ileo-colic angle of a child, aged 8 weeks. The ileo-colic fossa is incomplete, but its roof is studded and thickened with lymphatic glands. The axis of the ileum is horizontal.

in France. The folds give rise to pouches whose extent and complexity vary in each specimen, but, as a rule, those in front of the ileo-colic junction are simpler than those behind and below it. The ileo-colic fossa resolves itself into two great types, the complete (Fig. 1) and the incomplete (Fig. 6), whilst in some cases (Fig. 2) it is entirely absent. The complete fossa (Figs. 1 and 5), which is usually coexistent with the simpler types of cæcum, is that in which the ileo-colic fold stretches in a wide sweep from the ileo-colic mesentery across the ileum to form the mesentery of the appendix. This fold is attached to the cæcum externally, and terminates internally in a crescentic margin which forms the mouth of the ileo-colic fossa. This border is sometimes thick and rounded (Figs. 1 and 6), at other times thin and membranous (Fig. 5), and the ileo-colic fold may often contain one or more lymphatic glands (Fig. 6). The lower wall of the pouch is formed by the ileo-cæcal fold in the most simple instances. This fold is attached for a varying distance along the lower border of the ileum, but, like all the other structures in this region, it varies greatly in its

development, and is often wholly absent. Numerous modifications of this type are seen (Figs. 1 and 5), but they agree in having a pouch of greater or less depth which involves the whole width of the ileum. The incomplete form of pouch (Figs. 3, 4, and 6) occurs when the ileo-colic fold terminates on the anterior surface of the ileum. When there is no pouch (Fig. 2) the ileo-colic fold is attached to the ileum in its whole extent.

The peritoneal folds lying behind the ileo-cæcal junction are much larger and more complex than those situated in front of it, and the fossæ formed by them vary so greatly in width, depth, and structure that I have no time at present to describe them adequately. It must suffice to say that they are divisible broadly into the ileo-cæcal (Fig. 3, b) which runs upwards behind the ileo-colic junction and the subcæcal fossæ, which are situated behind the cæcum, and are less constant than either the ileo-colic or the ileo-cæcal pouches. The course run by an ileo-cæcal intussusception will depend materially upon the arrangement of these peritoneal folds and their fossæ. When the folds are simple the fossæ are small, and if lymphatic glands are absent the case is probably only a simple invagination which can easily be reduced by irrigation of the bowel in its earlier stages, or, if it be left unreduced, will run a chronic course, killing the patient by exhaustion, or ending in sloughing and separation of the intestine. When the peritoneal folds are complex the fossæ are large, and if the lymphatic glands are numerous so much tissue is necessarily invaginated in addition to the intestine that if early strangulation does not occur, extensive adhesions of the puckered mesentery must necessarily be formed which will soon cause the intussusception to become irreducible. Such cases will therefore run an acute course. The nature of the mouths of the pouches must also be taken into account, for in some cases they are so wide that the fossæ are merely depressions, whilst in others they are no bigger than pinholes, and the fossæ are then closed sacs, whose walls would either easily be brought into apposition by pressure, or would become distended by the products of inflammatory exudation.

c. RELATIVE DIAMETERS OF ILEUM AND COLON.

It is of importance, too, in considering the anatomical points connected with an ileo-colic intussusception to ascertain the relative diameters of the ileum and colon. The width of the large intestine at birth is only a few millimetres greater than that of the small intestine, and before birth its diameter is usually the same, or even a little less; at the age of 15 it is from two and a half to three times as large. The colon begins to grow in girth directly after birth, as might be expected when we remember that its function is to store up and consolidate the refuse food materials until they can be cast out of the body at convenient times and places. It appears, however, that while the colon grows in width it remains at first almost stationary in length, whilst, on the other hand, the ileum grows both in length and breadth. The question of the growth of the large intestine in length was very carefully studied by Mr. Treves,⁷ who occupied this chair in 1885. He executed his task with such skill and assiduity that I did not think it worth while to work over the same ground, but I took the liberty to measure the widths of the large and small intestines in each of my specimens. These transverse measurements show that the colon increases very rapidly in capacity, though for a time its length may remain stationary. Thus in the specimen obtained from a child aged 5 weeks the small intestine has the same transverse diameter as in a child at birth, but the diameter of the colon has increased by a quarter, and in a child of 5 months old, the small intestine remaining of almost the same diameter (No. 7 in the Table), the large intestine has nearly doubled its width. Individual measurements like this prove nothing, but a general review of the figures shows that whilst the ileum only rarely doubles its diameter, if 11 or 12 millimetres be assumed to be a fair average width at birth, the large intestine not only often doubles its size, but may even treble or quadruple it.⁸

⁷ *Anatomy of the Intestinal Canal and Peritoneum in Man*, 1885, pp. 9 and 10.

⁸ Intussusception is said to be so uncommon amongst the native population of India, that I asked Surgeon-Major Gibbons, I.M.S., and Surgeon-Captain O'Kinealy, I.M.S., to obtain for me the ileo-colic angles of Hindu children. After much trouble they have been successful in sending me two, of which the measurements were: (1) M., aged 2 years, ileum, 17 mm.; colon, 27 mm.; no ileo-colic fold. (2) M., aged 3 years, ileum, 18 mm.; colon, 24 mm.; no ileo-colic fold.

These facts seem to have an important bearing upon the question of the origin of intussusception. The colon is growing rapidly and continuously in width from birth onwards, but at about the age of 4 months it also begins to grow in length. The small intestine has grown steadily in length and breadth from the beginning, but the increase in its circumference is less rapid than the increase in its length. During the early months of life, therefore, there is a rapidly increasing disproportion between the transverse diameters of the large and small intestines. Physiology teaches us that too rapid growth is often associated with perversion of function, especially when, as in this case, the increased rate of growth affects both the muscular and the nervous tissues, but to this we shall recur presently. Clinical evidence shows that spontaneous intussusception occurs most frequently between the fourth and sixth months, when the large intestine is beginning to grow in length as well as in width.

d. THE ILEO-CÆCAL VALVE.

The last point of anatomical interest to which I directed my attention was the ileo-cæcal valve. The description found in the ordinary textbooks of anatomy is substantially correct in a large number of cases, but in many the arrangement differs. The orifice is often oval or circular in the hardened specimens, instead of being horizontal, and a part of the valve is frequently so defective that the opening appears to be horseshoe-shaped when it is seen from cæcum. In one or two cases the orifice very closely resembled the pyloric opening in the stomach. The ileum nearly always projected for 4 to 7 millimetres into the lumen of the intestine; sometimes as a conical process, at other times more on one side than upon the other, and in a few cases the mucous membrane of the ileum was distinctly prolapsed into the large intestine.

PHYSIOLOGICAL.

Let us turn now from the anatomical to the purely physiological side of the question as to the cause of intussusception. Experimental work seems more likely to help in the elucidation of the problem than simple observation. Observation tells us, however, that the peristaltic wave passing along the small intestine ceases at the ileo-cæcal valve, and that a new wave is developed there by the contraction of the coats of the large intestine. It is quite possible, therefore, for the end of the ileum to be contracted or even to be absolutely quiescent at the instant when the colon is contracting energetically.

The experimental work of Dr. Leubuscher,⁸ elaborated and carried to a more successful issue by Professor Nothnagel,⁹ has taught us that intussusception is due to one piece of gut swallowing, so to speak, a neighbouring piece, which has become constricted in consequence of local or other causes producing a vigorous contraction of its circular layer of muscle. An intussusception, therefore, is essentially and usually spasmodic in origin, and though there may be occasionally a paralytic form, which is secondary to other causes acting upon the intestinal wall, I do not believe that it ever causes a spontaneous invagination.

The important part which is played by the muscular coat of the intestine in producing an intussusception is well seen in two specimens¹⁰ which came from the museum of Mr. George Langstaff. They were taken from dogs which had been dosed with turpith mineral for the cure of the distemper. The signs of intussusception began almost directly after they had taken the drug. The first dog lived two days, the second dog three days. These specimens led me to experiment with turpith mineral to discover whether the intussusceptions were produced by it or were merely of accidental occurrence, and they formed the beginning of a series of experiments upon the effects of increasing the peristalsis of the intestines. The experiments were made upon healthy cats, rabbits, and guinea-pigs, as well as upon similar animals whose intestines had been invaginated by drawing the colon over the cæcum and ileum, the neck of the artificial invagination being afterwards fixed with a few point sutures of silk or horsehair. Turpith mineral, black sulphide of mercury,

eserin, barium chloride, calomel, rhubarb, magnesium sulphate, croton oil, and castor oil were administered with varying results. Some of these drugs, like croton oil, turpith mineral, and the black sulphide of mercury, were administered on account of their irritant action; some, like rhubarb, Epsom salts, barium chloride, and castor oil, because they increased in various ways the peristaltic action of the bowel; whilst others, like eserine, were given because they produced a tonic contraction of the coats of the intestine.

Time fails me to detail the experiments I have performed; it must suffice for the present to give results. Turpith mineral and black sulphide of mercury cause enteritis, and so modify the irritability of the intestine as to lead to its muscle being thrown into a series of localised contractions separated from each other by relaxed portions of the intestine, an arrangement which is most favourable to the formation of a series of short intussusceptions. Eserine, on the other hand, produces so uniform a contraction of the intestinal muscle that no intussusception could possibly be produced. I endeavoured to modify this uniform contraction by excising pieces of the mesentery and by administering purgatives to the animal before the eserine was injected, but without any satisfactory result. The effects of barium chloride were of great interest. The increased peristalsis produced by the injection of 6 minims of a 2 per cent. solution of barium chloride was sufficient in a cat to reduce the ileac portion of an artificial invagination, though the cæcum remained completely invaginated within the colon. A different result occurred in another invagination, for the increased peristalsis led to the intestine becoming M-shaped at the point of invagination, the kink being so sharp that the animal died of complete intestinal obstruction.

A simple invagination of the intestine in a cat produces little or no discomfort to the animal, for it leads neither to gangrene, strangulation, nor acute symptoms. This specimen (Fig. 7) was taken from a cat in which the

Fig. 7.—An ileo-ileo-colic invagination produced experimentally in a cat. The invagination was removed, and the cut ends of the intestine were united by Maunsell's operation a week after the intussusception had been produced. The ileo-cæcal valve is seen in section at *a*, whilst *b b* mark the position of the sutures which a week previously were inserted at the neck of the invaginated intestine.

ileum was invaginated into itself, and was pushed into the colon, where it was secured with three interrupted sutures. The sutures were of silk, and, as it was my first operation upon a cat, I did not allow for the great thickness of the muscular coat in these animals. The sutures therefore hardly passed into the submucous layer, and they consequently held badly and threatened to cut their way out of the brittle muscular coat when I tied them. The intestine was replaced in the peritoneal cavity and the abdomen was closed.

The cat appeared to be so well on the day after the operation that I felt sure the sutures had cut their way out, and

⁸ Virchow's Archiv, vol. lxxv, 1881, p. 83.

⁹ Beiträge zur Physiol. und Pathol. des Darmes, Berlin, 1884, p. 42; Spec. Path. and Therap., Bd. xvii, Theil ii, 1886, p. 297.

¹⁰ Museum of the Royal College of Surgeons of England, Nos. 2,725 and 2,726.

that the invagination had reduced itself. For a week the animal took its food and behaved exactly like a healthy cat, except that it would not jump, and got down steep places by scrambling. Eight days after the first operation it was again anaesthetised, and a second incision was made to the right of the first one, which had nearly healed. The invagination still existed, so I removed it by cutting through the intestine above and below the tumour. The ileum and colon were then joined by Maunsell's method, the operation being completed in thirty-five minutes from the first incision, and half an hour later the cat was lapping milk.

The invaginated portion of the intestine (Fig. 7) bears out the theory that intussusception is due to the swallowing of a piece of constricted intestine by a neighbouring portion, whose peristalsis is still active, whether the contraction be a descending one, as is usual, or retrograde, as is less often seen. The invaginated intestine has been divided longitudinally. The sutures are seen in position, but instead of being at the neck of the sac, where they were inserted a week previously, they are now situated in the very middle of the intussusception, at a distance of 32 millimetres from its beginning, as measured along the lesser curvature. The whole length of the intussusception is 75 mm., and as the sutures mark the original neck, the invagination has advanced 32 mm. in eight days—that is to say, it has nearly doubled itself in this time. The intussusception is an excellent example of the ileo-cæco-ileo-colic variety, for the cæcum is intact, and the ileo-cæcal valve is normal, and has not become invaginated. The intussusception is pervious throughout, a fact which explains the freedom of the cat from any symptoms of obstruction. The invagination is of the chronic form, for the apex of the invaginated ileum is alone congested. The intussusceptum, as is usual, is curved towards the side of its mesenteric attachment. The two apposed serous surfaces can be separated from each other as low as the point of suture, but below it they are intimately blended by the union of the mesenteric folds. The adhesions are much better marked along the concave surface, where the mesentery is crowded into a small space, than along the greater curvature, where the two serous surfaces can be separated in their whole extent, except at the point where they have been sutured.

Amongst the other interesting points which this experiment, with its resulting specimen, teaches us is the important one that the force exercised by peristalsis is extremely slight even in an animal with so muscular an intestine as a cat. The sutures were barely capable of maintaining their position when they were tied, yet as the invagination increased they must have turned over the edge of the intussusception without losing their hold. The feeble force of peristalsis has also been noticed by Professor Cash,¹¹ of Aberdeen, who says that in dogs a weight of 8 to 10 g. is sufficient to prevent a well-developed peristaltic wave from executing its object, whilst the traction exercised by a weight of 5 g. produced much hindrance and caused spasmodic contraction of a propulsive character which created most distinct discomfort or colic.

PATHOLOGICAL.

A considerable number of the cases of intussusception are merely invaginations of one piece of bowel into another which is usually of larger calibre, and, if there is a great disproportion in width, spontaneous reduction may take place, as in a case recorded by Mr. Thomas,¹² occurring in a boy aged 16 months, in whom he had distinctly felt the invaginated intestine *per rectum*. When the disproportion between the diameters of the ensheathed and ensheathing intestine is less marked or when much additional tissue is invaginated, adhesions are formed, the intussusception becomes irreducible, and in process of time secondary changes take place in the intestine, which lead to strangulation, to gangrene, or to ulceration of the neck of the bowel.

1. STRANGULATION.

Strangulation in intussusception is more often chronic than acute. Its exact cause is unknown even in hernia, though its mechanism is well understood. It is of the true or elastic variety when it occurs in intussusception—that is

to say, the invaginated bowel is enclosed in a lumen which is either no bigger than itself, as in the enteric form, or only slightly larger, as in the ileo-cæcal variety; whilst in the ileo-colic type, where the ileum passes through the ileo-cæcal valve, the valve may form an additional constricting agent—for, as I showed in my first lecture, hæmorrhage may take place into its substance or it may become inflamed. During the actual invagination the lumen of the ensheathing intestine must be dilated to its utmost extent. The wall of this portion of the intestine then recoils and exercises sufficient pressure upon the vessels of the mesentery to cause a marked congestion in the tissues of the intussusceptum. The congestion leads to a stagnation in the blood flow, and so to a cellular infiltration, which is best marked at the two points where the vessels are doubled upon themselves—that is to say, at the end of the intussusceptum and at the point where the pressure is exercised, which is the neck.

In children the tissues are so soft, and the peristaltic wave engulfing the gut is so feeble, that in some cases the intestine merely slips on and is imprisoned, for, as I have endeavoured to show, there is usually a very considerable difference in the relative diameters of the large and small intestines even in children between the ages of 4 and 6 months, the time when statistics show that intussusception is most common. Clinical evidence shows that there is so little interference with the contractile power of the muscular coat of the intestine, that in many cases the intussuscepti certainly, and the intussusceptum probably undergo rhythmic peristaltic contraction, for if the tumour be carefully watched in a well-marked case, its consistency will be found to vary greatly, at one time hard and easily felt, at another it is quite unrecognisable. The continuance of peristaltic contractions show that the blood flow is no more stopped through the intestine than it is in the gravid uterus, for if it were the muscle would be paralysed and there would be no longer any peristalsis. In an intussusception, however, there is no arrangement for storing up the blood during the contractions as there is in the uterus. The intestinal tissues, therefore, become engorged, oedema takes place, and the congestion leads to the extravasation of blood which is so marked a feature in the microscopical sections taken from tissues thus affected.

2. GANGRENE.

The inflammatory changes which result from a continuance of these conditions lead to gangrene which may spread slowly from the apex towards the neck of the invaginated piece of gut, though in many cases it appears early and may involve considerable portions of the invaginated bowel. Gangrene, however, is less common in intussusception than it is in hernia, and there are several reasons for this difference between the two conditions which have otherwise many pathological affinities. Intussusception is essentially an affection of children, and in children gangrene of any part is rare, even after the most serious injuries, partly because their vascular system is so healthy that it adapts itself readily to the most unfavourable conditions; partly because the blood supply to all growing parts is very abundant, and partly because all young tissues are soft and yielding. In an intussusception, therefore, the bowel passes through an elastic ring which, even when it is inflamed, never offers a resistance in any way comparable with that exercised by the rigid fibrous structures which compress a hernia or an internal strangulation. When gangrene takes place early in intussusception, it often follows upon a particularly violent onset in cases where the invagination is ascribed definitely to some sudden muscular effort; to increased, prolonged, and violent peristalsis, as after the administration of purgatives; and occasionally to such rare conditions as the blocking of the narrowed lumen of the intussusceptum with blood,¹³ or portions of undigested food, or even to the grafting of a second intussusception upon an earlier one which was running an acute course. Gangrene occurring in the course of an intussusception is more limited, is more chronic, and is less likely to set up suppurative peritonitis than a similar condition in strangulated hernia. It is more limited because the tissues are less liable to injury, as it is impossible to perform taxis for the relief of an intussusception.¹⁴ It runs a more chronic course because it is asso-

¹¹ *Proc. Roy. Soc.*, vol. 41, 1887, p. 230.

¹² *Lancet*, ii, 1886, 1219.

¹³ *Trans. Path. Soc.*, xxviii, 1877, p. 131.

¹⁴ *Holmes's System of Surgery*, Ed. 3, vol. ii, p. 767.

ciated with less perfect interference with the blood supply, and the intestine is thus enabled to protect itself more perfectly by reparative changes. It is less likely to set up general peritonitis, because the gangrenous portion is enclosed in a sheath of healthy intestine, for the gangrene in chronic cases nearly always begins in the intussusceptum, though it may afterwards involve the intussusciptens.

3. ULCERATION.

The occurrence of ulceration or of necrosis of the intussusciptens is a much greater danger than gangrene of the intussusceptum, but it is fortunately somewhat less common. It is, however, met with at either end of the intussusception, as well as in the bowel above and below that portion which has been invaginated. We do not yet know the exact course taken by the micro-organisms as they pass through the wall of the bowel, and, though I have been engaged in an endeavour to trace their passage, my results are not yet sufficiently advanced to warrant any definite statement. But there seems to be no doubt that micro-organisms begin to traverse the intestinal wall when a loop of bowel has been constricted for a period of from 4 to 48 hours, and that the more completely the blood supply is arrested the more rapidly do they pass. Suppurative peritonitis is not usually set up in anything like so short a space of time, and when it does occur it is more often associated with rupture of the gut from the progress of ulceration due to accidental or additional causes than to the simple extension of suppurative processes through the wall of the intestine. The experiments of Grawitz¹⁵ and of Halstead¹⁶ have shown that even pure cultivations of pyogenic organisms as well as suppurating tissues and particles of faeces do not necessarily cause suppuration when they are introduced directly into a healthy peritoneal cavity. Accessory conditions must be present to enable pus-producing substances to set up a purulent peritonitis, and these accessory conditions seem to have in common the power of preventing absorption or removal of the pyogenic substances from the peritoneal cavity. The whole subject of absorption from the peritoneal cavity wants to be worked out, for Drs. Adler and Meltzer¹⁷ in their excellent essay Upon the Path by which Fluids are carried from the Peritoneal Cavity into the Circulation, have done enough to show that it bristles with difficulties, though it is full of interest.

CLINICAL FACTORS.

There are good reasons to believe that intussusception is associated with peculiarities of structure and function in the alimentary canal. In a certain proportion of cases intussusception has occurred in more than one member of a family¹⁸ and in the children of parents who have themselves suffered from conditions associated with organic or functional disturbances of the bowels. A remarkable instance of this came under the notice of Mr. Pick¹⁹ in the case of a child, aged 15 months, who was admitted under his care into St. George's Hospital for the relief of an intussusception. The grandfather and the father of the child had been operated upon for strangulated hernia, and one brother had died of strangulated hernia. The defective condition, whether of structure or of function, may be repeated in different members of the family, or it may be peculiar to the individual. In the latter case the symptoms of intussusception may be preceded for months by an increase in the peristaltic contraction of the bowels, or they may be repeated at long intervals²⁰ even when a *post-mortem* examination shows that the invagination has not been associated with any gross lesion in the alimentary canal like a polypus, sarcoma, or cancer.

Many cases of intussusception have been cured by injection or inflation of the bowel through the rectum, even when the apex of the intussuscepted gut has protruded beyond the anus. In a few cases such advanced intussusceptions have receded spontaneously, whilst in others when the abdomen has been opened the surgeon²¹ has rejoiced to find that the

invaginated bowel unfolded itself spontaneously as soon as the merest touch had released the constriction at its neck. The effects of the constriction in untreated cases are essentially chronic, for strangulation, gangrene, and ulceration are secondary changes which may never make their appearance in the course run by an ordinary intussusception.

CONCLUSIONS.

The evidence derived from anatomical, physiological, pathological, and clinical data render it legitimate to assert that spontaneous ileo-cæcal intussusceptions occur where the colon is considerably larger than the ileum, and is so unduly movable that it readily allows itself to become invaginated when once the process has begun. This variety of intussusception is essentially an affection of childhood, and such an undue increase in the width of the colon implies either a congenital abnormality or an unduly rapid growth, for at birth the diameter of the large intestine is practically the same as that of the ileum. Such a rapid increase in the width of the large intestine may, perhaps, in some instances, render the ileo-cæcal valve less competent to guard the end of the ileum, because the valve is not capable of very rapid growth if it is to be firm, but this is probably a factor of very rare and subordinate importance.

Though anatomical peculiarities are important factors in the production of an intussusception, the physiological factors are no less important, for they apply in all probability to every form of spontaneous invagination whether it is of the enteric variety, the ileo-cæcal, or the colic. The physiological factor is much less easy to specify than the anatomical, for it is almost certainly an individual peculiarity. It may be stated broadly, however, that as regards the ileo-cæcal portion of the intestine the increased mobility, coupled with the unduly rapid growth in the width of the large intestine is probably associated with increased and irregular peristaltic movements of the large intestine. Everyone who has had much experience in watching children must have noticed how subject they are to an irregular twitching of the muscles, and how greatly such inco-ordinate movements vary in different children and in different families. They are most common at the instant when the child drops off to sleep and during the act of awakening. They occur both in the voluntary and in the involuntary muscles, and are due, as physiologists think, to a contraction of the interfibrillar sarcoplasm, whilst the ordinary tetanus and twitch is caused by an additional shortening of the contractile fibres. This theory explains why the symptoms of intussusception so often appear at the instant when a child awakens, why it is that boys rather than girls, and the most active and the best grown children are attacked as often as those who suffer from chronic inflammatory affections of the alimentary canal. The muscular system in the active and well grown is undergoing its greatest development, and its sarcoplasm is in an unstable condition as in the puny where attacks of enteritis have unduly heightened the irritability of the intestinal muscle, and have thus led to the irregular twitchings which, with suitable anatomical conditions, may form the starting point of spontaneous intussusception.

The cause of intussusception is obscure, but clinical evidence shows that it may be produced mechanically though the spontaneous form is the more common. Amongst such mechanical causes are direct injury to the belly,²² sudden and violent muscular efforts,²³ gymnastic movements,²⁴ jolting or dandling,²⁵ paroxysmal coughing as in whooping cough,²⁶ or it may even be the reward of greediness, as in the cases recorded by Max Baur²⁷ and Leichtenstern,²⁸ in which men were suddenly seized with symptoms of intussusception shortly after eating a quantity of cherries—stones and all. It is, indeed, no matter of surprise that such causes should produce an intussusception, for any of them might lead to a sudden and limited constriction of the intestine associated with an active peristaltic movement of a neighbouring portion sufficient to draw the receiving layer of the gut over the contracted portion, which then becomes

¹⁴ *Charité Annalen*, xi, 1884, p. 770.

¹⁶ *American Jour. of the Med. Sci.*, xciv, 1887, pp. 437 and 451.

¹⁷ *The Journal of Experimental Medicine*, i, 1896.

¹⁸ Hutchinson, *Med.-Chir. Trans.*, vol. lix, 1876, p. 100; Bryant, *Harveian Lectures*, p. 12, Case 17.

¹⁹ *Lancet*, 1891, i, p. 1312.

²⁰ Pick, *Lancet*, i, 1891, p. 1312; *Archives of Pediatrics*, xiii, 1896, p. 616.

²¹ Marshall, *Med.-Chir. Trans.*, vol. lix, 1876, p. 81; Fagge and Howse, *Med.-Chir. Trans.*, vol. lix, 1876, p. 88; Shepherd, *Lancet*, ii, 1892, p. 1155.

²² *Guy's Hosp. Rep.*, 1869, p. 290; *Trans. Clin. Soc.*, xvi, 64; *Lancet*, ii, 1888.

²³ *Lancet*, ii, 1892, 482.

²⁴ *Lancet*, ii, 1888, 315.

²⁵ *Lancet*, i, 1893, 651.

²⁶ *New York Medical Record*, vol. xlix, 1896, p. 73, Case XIV.

²⁷ *Trans. Clin. Soc.*, xxii, 1889, p. 282.

²⁸ *Berl. klin. Woch.*, No. 33, 1892, p. 817.

²⁹ *Deutsch. Archiv. f. klin. Med.*, xii, 1874, p. 381.

the apex of the intussusceptum. As soon as the intussusception has been started the anatomical peculiarities again become of paramount importance, for they determine the character of the intussusception. In the ileo-cæcal forms a wide colon with few and simple ileo-colic folds, devoid of lymphatic glands, will allow the intussusception to run a chronic course, even though the amount of bowel invaginated is very great. Complex fossæ, with numerous glands at the ileo-colic angle and prolongations of mesentery along the end of the ileum will, no doubt, so far steady the ileum as to render its intussusception less likely. But if it should occur, the additional amount of tissue invaginated will render the impaction so peculiarly tight that if gangrene is not produced at once early adhesions will be formed and the intussusception will soon become irreducible.

REMARKS ON THE MEDICAL ASPECT OF A SYSTEM OF COMPULSORY INSURANCE OF THE WORKING CLASSES.

By F. PARKES WEBER, M.D., M.R.C.P.LOND.

As the subject is comparatively little known to the majority of English medical men, in order to make the medical remarks intelligible, I must first give a short account of the German National Workmen's Insurance System, and this account I shall give as nearly as possible in the words of the *Guide to the Workmen's Insurance of the German Empire*,¹ compiled by Dr. Zacher, permanent member of the Reichs-Versicherungsamt. It will, however, be unnecessary to enter into any complicated details or figures.

The work of social reform was practically initiated by the Message of the German Emperor, William I, to the Reichstag on November 17th, 1881. The Message, as communicated by Prince Bismarck, was expressed in the following words:

We consider it Our Imperial duty to impress upon the Reichstag the necessity of furthering the welfare of the working people. We should review with increased satisfaction the manifold successes with which The Lord has blessed Our reign, could We carry with Us to the grave the consciousness of having given Our country an additional and lasting assurance of internal peace, and the conviction that We have rendered the needy that assistance to which they are justly entitled. Our efforts in this direction are certain of the approval of all the federate Governments, and We confidently rely on the support of the Reichstag, without distinction of parties. In order to realise these views a Bill for the Insurance of Workmen against Industrial Accidents will first of all be laid before you, after which a supplementary measure will be submitted providing for a general organisation of Industrial Sick Relief Assurance. But likewise those who are disabled in consequence of old age or invalidity possess a well-founded claim to a more ample relief on the part of the State than they have hitherto enjoyed. To devise the fittest ways and means for making such provision, however difficult, is one of the highest obligations of every community based on the moral foundations of Christianity. A more intimate connection with the actual capabilities of the people, and a mode of turning these to account in corporate associations, under the patronage and with the aid of the State, will, We trust, develop a scheme to solve which the State alone would prove unequal.

Thus the working man, unfitted for work by sickness, accident, invalidity, or old age, was to have a legal right to a provision sufficient to render him independent of public charity or Poor-law relief. This end could only be attained by a system of general and compulsory insurance, based on mutuality and self-administration, and the endeavours to attain this end have resulted in the system of working-men's insurance now employed in Germany. This system consists of three component parts: (1) Insurance against sickness; (2) insurance against accidents; (3) insurance against invalidity and old age. These component parts, which supplement each other to form the insurance system, must be separately considered.

INSURANCE AGAINST SICKNESS.

The insurance is intended to secure the insured sufficient relief in case of illness during at least thirteen weeks. All the insured have a legal claim to:

(a) Free medical attendance and medicines from the beginning of the illness; likewise spectacles, trusses, bandages, etc.

(b) (From the third day of any illness which incapacitates from work) a daily sick pay amounting to one-half the daily

wages on which the contributions have been based; or else (in special cases) free admittance to a hospital, together with half the sick pay for the family.

(c) Burial money.

(d) In the case of women, sick relief during four weeks following confinement.

The insurance against sickness, contrary to that against accidents, is restricted to local organisation, because cases of less importance are continually occurring in which relief to be efficacious must be prompt. The law has therefore authorised the formation of various associations for obligatory insurance against sickness. By the associations being local and self-administrative, the management of their funds is more easily controlled, and simulation on the part of the insured is more difficult. The contributions of the different classes of workmen vary from 1 to 3 per cent. of their average daily wages. The law binds the employers themselves, when depositing the contributions of their workmen, to pay a sum equal to one-half the contributions of the employed, so that two-thirds of the whole are furnished by the workmen and one-third by their employers.

INSURANCE AGAINST ACCIDENTS.

The liability law of June 7th, 1871, had not the desired effect, partly because the burden of proof was laid on the poor workman seeking redress, and partly because the person—sometimes a fellow workman—responsible for the accident was often unable to pay damages. Moreover, this law did not provide for accidents caused by chance or by the fault of the workman himself.

The present system of insurance against accidents is carried out under the guarantee of the empire, on the mutual system, by the employers united in trade associations, which may embrace the different branches of industry in certain districts or in the whole empire.

The object of the insurance arrangements is to secure compensation for bodily injury or for death, in consequence of an accident to the workman whilst working for his employer, injuries sustained on other occasions being excluded. As a matter of course the injured man must not have brought about the accident purposely, though the injury may be due to his own fault. The compensation includes the cost of the cure, and in addition a fixed allowance during the period of incapacity for work, or in fatal cases burial money and an allowance to the survivors (widows, children, parents) from the day of death. When the injured person is totally disabled the compensation amounts to two-thirds of his average year's earnings; if only partially incapacitated for work a fraction of this amount will be granted. For the first thirteen weeks after the accident the associations for insurance against sickness (or, in their absence, the employers) have to step in.

In the interest of a fair and suitable treatment of sufferers from accidents the trade associations are, however, legally authorised either to commit at their own costs the care of the injured to the associations for insurance against sickness beyond the thirteenth week, until a complete cure is effected, or they may themselves undertake the charge of the patients at any time during the first thirteen weeks, with the understanding that their outlays of sick pay shall be refunded by the associations for insurance against sickness.

The amount of the compensation is fixed, after a police investigation, by the organs of that trade association in whose district the accident happened. Against this decision an appeal may be made to an arbitration court, composed of two members of the trade association, two representatives of the insured workmen, and a presiding magistrate. This court is invested with the character of a special court of law; in more complicated cases an appeal from its verdict may be made to the Reichs-Versicherungsamt.

Every employer contributes to the burdens of the year in proportion to the risks to which he exposes his association.

As it is evident that both the trade associations and their individual members have a strong interest in diminishing the chances of accidents, the law confers on the associations the important prerogative of prescribing regulations for the prevention of accidents. Through these regulations the employers can be compelled, under penalty of higher assess-