of tonsillitis several of the cows at the farm had suffered from mastitis and other inflammatory infections of the udder, and it is thus possible that the bailiff may have conveyed to one or other of such cows the infection which in some way he had brought from the patient at the house he visited daily.

Incidentally, it may be mentioned that following the cases at the farm there was a small outbreak among the occupants of this house and that the doctor who treated patients both at the farm and at the house considered that all of them were of the same nature.

Both the outbreaks I have described, the one of scarlet fever and the other of sore throat, were due to haemolytic streptococci of closely allied though distinguishable types and in both the infection was spread by milk. While, however, the scarlet fever epidemic lasted only for a few days and the attacks, numbered by tens, were almost limited to persons served by a single roundsman, himself a carrier of infection, the epidemic of sore throat was spread over several weeks and the patients, numbering many hundreds, received their milk from many roundsmen, none of whom, so far as is known, was infected. The difference in the duration of the outbreaks is doubtless attributable, at any rate in part, to the circumstance that scarlet fever is notifiable, this permitting of prompt preventive measures, while sore throat is not. The difference in their magnitude, however, is only to be explained, I think, by the different ways in which infection was spread. The scarlet fever patients owed their infections, I believe, to the direct infection of the milk by the infected roundsman by whom they were served, while to account for the many hundreds of attacks of sore throat occurring among persons living in all parts of the area served by a large dairy, some gross infection of the milk must be assumed. It has been seen that when the milk from a single farm of the many supplying the dairy was discontinued the outbreak ceased, and it would thus seem that for this milk to have infected so many consumers, greatly diluted as it was with other milk before it reached them, it must have been grossly infected. Such gross infection of milk with organisms pathogenic to human beings is, I think, best to be explained, in the light of the experiments of Davis and Capps, by the infection of cows, already susceptible owing to the condition of their teats and udders, with organisms derived from a human source.

¹ Miller and Capps: *Journ. Amer. Med. Assoc.*, Vol. lviii, 1912.

² Davis and Capps: *Journ. Infect. Dis.*, Vol. xv, 1914.

INTUSSUSCEPTION ILLUSTRATED BY THREE UNUSUAL EXAMPLES

BY

JAMES CROOKS, M.B.ED., F.R.C.S.ENG.

SURGICAL REGISTRAR, HOSPITAL FOR SICK CHILDREN,
GREAT ORMOND STREET

Three cases of intussusception recently operated upon at the Hospital for Sick Children have certain points of interest. In the first I was able to discover the exact cause; in the second a recurrence was shown to have the same point of origin as the previous intussusception; and in the third, recovery followed intestinal resection and anastomosis in a baby aged 8 months.

CASE I

This baby girl, aged 6 months, provided an opportunity of obtaining a good specimen of bowel indicating the cause of the intussusception. As a rule the intestine of such of these children as come to the post-mortem room is gangrenous, and it is impossible to determine precisely the exact starting-point of the intussusception by an examination of it.

This baby, however, died twenty-four hours after operation from respiratory failure, the cause of which was proved by the necropsy to be mal-development of the diaphragm.

At the operation the apex of the intussusception reached half-way down the descending colon; on following it backwards the colon was found to pass under the right costal margin, so that the incision had to be prolonged upwards. The liver lay completely in the right side of the chest. The point of origin of the intussusception, which reduced easily, was six inches proximal to the ileo-caecal valve, and could be felt as a definitely localized and thickened patch in the bowel wall, of the area of a sixpenny bit. The surrounding gut was not obviously thickened.

The necropsy showed that the muscle of the diaphragm was absent, save for a few fibres in its left posterior part; the rest was solely represented by a thin complete fibrous sheet; this lay over the liver, which was herniated into the right side of the chest. The right lung was unexpanded and occupied only the apex of that side of the chest. The Peyer's patches in the ileum were large, and the thickening in the bowel wall corresponding to the starting-point of the intussusception was an almost polypoid hypertrophy of the lower end of one particularly big patch. This is well shown in the figure. There could be no doubt that this thickened part of the patch projecting into the lumen of the bowel had acted like a polyp in causing intussusception. The mesenteric glands were moderately enlarged.

Half-scale drawing of bowel of Case I. The upper figure shows the large size of the lymphoid patch, and the lower profile sketch indicates how it projected into the lumen.

CASE II

Operation on this boy, aged 6 months, revealed an intussusception which had reached the splenic flexure. Reduction was difficult, but the intestine was considered viable. The starting-point, which was unusually high, being eighteen inches proximal to the ileo-caecal valve, was indicated by a definite dimple, but the whole of the affected bowel was so oedematous that no localized thickening could be felt. There was no evidence of a Meckel's diverticulum.

At the age of 12 months this boy was again presented to me by his mother, within two and a half hours of a recurrence of his symptoms. Even after this short time the intussusception, which had again reached the splenic flexure, was difficult to reduce. Healed scars of the peritoneum covering the bowel showed clearly the extent of the previous invagination, and this second intussusception had the same starting-point as the first. Again there was a dimple, but no localized thickening could be felt, the whole of the bowel involved being oedematous. A year has passed since the second operation, and there has been no recurrence.

CASE III

A baby girl, 8 months old, was brought to the Hospital for Sick Children with a history of having been seized by abdominal pain seven days before. She had vomited once at the onset, but since then had taken her food well, and had not apparently had further pain. She passed one stool on the first day, but since then the bowels had not opened, except for the passage of a little dark blood on the fourth day.

On examination she did not appear to be in pain, but was pale and listless. The abdomen was distended, particularly in its upper part, where it was marked by the pattern of underlying coils of bowel. No localized tenderness was discovered, nor could a tumour be felt apart from the general

distension. Rectal examination revealed that the bowel was empty; there was no tumour, and no blood on the examining finger.

At the operation some turbid fluid was found in the peritoneal cavity, and the small bowel was greatly distended. An intussusception reached half across the transverse colon. It reduced easily to within an inch or two of its apex, which was eight inches proximal to the ileo-caecal valve. The last inch was gangrenous and perforated. Three inches of ileum were resected, the ends closed, and a side-to-side anastomosis of ileum to ileum, just above the ileo-caecal valve, was performed.

For the first thirty-six hours after the operation 5 per cent. glucose in normal saline solution was given subcutaneously every six hours; the baby was kept under the influence of morphine, and nothing was given by the mouth. Thereafter feeding was rapidly resumed. There was no vomiting, and the bowels opened three times on the second day. Further progress was uneventful but for a mild wound infection, until the morning of the eighteenth day, when the child vomited and became collapsed. She remained so, and appeared to have periodic abdominal pain. In the evening the abdomen was opened again through the original incision. There was generalized turbid exudate, and in the right iliac fossa lay a coil of distended small bowel adherent to a loop of collapsed ileum, above the anastomosis. On dividing the adhesion the obstruction was relieved. The turbid exudate was removed, and the abdomen was closed; 150 c.cm. of 10 per cent. glucose-saline solution was injected into a vein. Uninterrupted recovery followed, and during the past year the baby has been perfectly well.

It will be noticed that these three examples are ileo-colic intussusceptions. This type has its point of origin in the small intestine; it is primarily enteric, but when the ileum has passed into the colon it is conveniently classified as ileo-colic. Out of thirty-seven consecutive operations, in which I made a careful examination of the point of origin of the intussusception after reduction, it was in the ileum in thirty-four; thirty-three of these were ileo-colic in type, and one was enteric. Of the remaining three, the ileo-caecal valve formed the apex in two; the third had a dimple in the caput caeci, and, according to Perrin and Lindsay, should be classified as ileo-caecal also. These surgeons, in their monograph in the *British Journal of Surgery*, 1921, based on the surgical records of 400 cases and not upon their personal experience, give the incidence of the ileo-colic type in intussusception as 31.5 per cent., in marked contrast with my small series, where it is over 89 per cent. Each of the thirty-four cases showed a definite thickening or dimpling in the ileum to indicate the point of origin or apex, for these two terms are synonymous. I am in entire agreement with Perrin and Lindsay when they find no evidence to support Leichtenstern's theory that an intussusception may grow at the expense of its entering layer only. If such were the case, there would be no thickening or dimpling to indicate the apex, for this would be continually changing. Thirteen of the thirty-seven children were girls; the average age was 13 months; the youngest was 3 months old, and the oldest 4 years and 10 months.

This high incidence of intussusception in young children starting in the ileum confirms the belief that the common cause is enlargement of a Peyer's patch, which excites uncontrolled peristalsis. This is illustrated by Case I. Case II suggests that such a patch may remain hypertrophied, or become so again, and then lead to a recurrence of intussusception. Case III shows that resection of a gangrenous intussusception may be confined to small intestine, involving an easier and safer operation than the removal of the colon. Further, it is the enteric intussusception which has become ileo-colic that is most liable to be gangrenous, since both the intussusceptum and its vessels have passed through the tight ileo-caecal valve.

I wish to thank the surgeons to the Hospital for Sick Children for allowing me to operate on these children; and for permission to publish this note.

Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

EXOTOXINS IN RELATION TO VACCINE THERAPY

I think it may be fairly stated that where a germ forms, as the major part of its poisoning properties, an exotoxin,—that is, a powerful diffusible poison secreted into the liquid culture medium in which it grows—a serum is usually the most successful antidote. Examples of this are tetanus, diphtheria, botulism, and dysentery. On the contrary, when the endotoxic factor is almost wholly predominant—that is, when the toxin is contained firmly within the germ and only liberated after autolysis—a vaccine is indicated as the appropriate antidote.

The clinical pathologist probably makes more vaccines from the staphylococcus and streptococcus than from any other germs, and in respect of these two organisms I would like briefly to point out that diffusible exotoxins are secreted by both, as well as endotoxins, as also in dysentery of the bacillary type; albeit in the last the exotoxic factor is by far the more important, and so important, in fact, that serum therapy is chiefly useful, rather than vaccines. *Staphylococcus aureus* and some strains of *Staphylococcus albus* form haemolysins and leucocidins, the former being quite powerful, as cultures on blood media show. The zone of haemolysis around the colonies of *Staphylococcus aureus* is markedly conspicuous. The streptococcus also forms exotoxins—streptolysin, which is a very powerful diffusible toxin whose effects we are aware of in fulminating cases, and also a haemolysin, well shown in the haemolytic strains.

Kolmer¹ has shown that the body not only contains antitoxins to these poisons, but that it can be stimulated to form more, if so desired, by appropriate and graduated inoculation.

The average autogenous vaccine ignores these important toxins. The staphylococcal emulsion is washed off the agar slope and suspended without the least regard for the exotoxin. That is the usual procedure. As a matter of fact, with the immunogen of Ferry and Fisher,² actually the reverse process is utilized and the washings are utilized alone, and the very important endotoxic factor is ignored. I maintain that either process is irrational, and I have endeavoured to combine the two factors in vaccine therapy as applied to the above two organisms as follows. The ordinary saline emulsions ignore the potent haemolysin and less potent leucocidin, and therefore it is more rational to make one's vaccine from liquid media and to include a certain proportion of that liquid medium in the vaccine.

The technique is quite simple. Twenty-four to forty-eight hour glucose serum broth emulsions are prepared and centrifuged. The emulsion of germs is made up in saline, and 5 per cent. of the supernatant fluid containing the endotoxins is added to the vaccine. I am sure that the results by this technique are superior to those formerly obtained, and that this method constitutes a more rational way of making vaccines from the organisms in question.

GEOFFREY SHERA, M.A., M.D.,
Consulting Pathologist to the Eastbourne
Hospitals and East Sussex
County Council.

¹ Kolmer, J. A.: *Infection, Immunity, and Specific Therapy*, 1917, pp. 110, 118, and 119.

² Ferry, N. S., and Fisher, B. S.: *Brit. Journ. Exper. Path.*, 1924, v, pp. 185 and 205.