references to the literature.¹ Since then two more cases have come under my care, and details of other cases under the care of my colleagues seem to indicate that localized spasm of the uterine muscle is a difficult subject, and one which is, at present, very imperfectly understood. Eden, in the 1915 edition of his *Midwifery*, recognizes: (1) Excessive retraction, with the formation of a retraction ring due to the violent efforts which the uterus makes to overcome some mechanical obstruction; (2) general tonic contraction from unsuccessful efforts of a strong uterus to overcome obstruction, or from irritation by drugs or manipulations; (3) local tonic contraction leading to a contraction ring.

The details of the two cases at present unrecorded are as follows:

CASE IV.

Primipara, aged 30, sent into Queen Charlotte's Hospital on August 2nd, 1913, by a doctor who had attempted to hasten delivery by manual dilatation of the cervix and high forceps, which failed. She had been in labour for forty-eight hours.

On admission: Pelvis $9\frac{1}{2}$, $10\frac{1}{2}$, $7\frac{1}{2}$ in. Child in L.O.P. position, dead. Head in cavity of pelvis. The resident medical officer's notes say: "No pains. It was decided to wait for pains and then perforate." When the fundus occasionally contracted the head was perforated, and with great difficulty he pulled it outside the vulva with the cranioclast, but all efforts to deliver the shoulders failed even after cleidotomy, and the fundus of the uterus now became "irritable."

When I saw her an hour later she was suffering from severe shock, so a 7 lb. weight was tied to the cranioclast, and she was left for transfusion, hot bottles, etc., for $1\frac{1}{2}$ hours. It was then very easy to deliver by bringing the arms down over the head and making light traction, whereas previously the strongest traction had failed to move the fetus at all. The fundus was now "flaccid," and the placenta temporarily retained, so it was removed manually and an intrauterine douche was given. There was no obvious ring obstructing the exit of the placenta, the delay being due to other causes.

The child weighed $8\frac{1}{2}$ lb. The mother recovered, but there was considerable sloughing of the vagina, resulting in the formation of a urethro-vaginal and uretero-vaginal fistula during the second week of the puerperium.

CASE V.

Patient aged 41. Fourteen previous pregnancies without difficulty. Admitted on August 26th, 1915, after being in labour twenty hours. Child dead and an offensive discharge coming from uterus. Previous repeated attempts at delivery had been made, and it was thought to be a case of locked twins, as a foot and hand could be felt high up, but nothing could be brought down below the brim.

I found a very tight contraction ring round the thigh of the child, which did not yield to continuous pressure. The mother being obviously infected, I did not risk a simple Caesarean section but excised the whole gravid uterus. As the vagina was cut through the child fell out, and so a detailed description of the specimen is useless. The mother made an uninterrupted recovery.

RETRACTION AND CONTRACTION RINGS.

It is obvious that these cases have no connexion with the retraction and thinning that occur, for instance, in a case of labour obstructed by a hydrocephalic head, where a retraction ring may form late in labour.

The conclusions arrived at from a study of the numerous recorded cases in 1912 may be summarized as follows:

DIFFERENTIAL DIAGNOSIS.

Contraction Ring.

1. A localized thickening of the wall of the uterus due to the contraction of the circular fibres over a point of slight resistance, most frequently over a depression in the child's outline or below the presenting part.

2. The uterine wall at the site of the contraction ring will therefore be thicker than it is either above or below.

3. The wall below is neither thinned nor over-distended.

4. The presenting part is not forcibly driven into the pelvis.

5. The child may be wholly or mainly above the contraction ring.

6. The body of the uterus above a contraction ring is usually relaxed and not tender.

7. Round ligaments are not tense.

Retraction Ring.

1. The junction of the thinned lower uterine segment with the thick retracted upper uterine segment.

2. The uterine wall above the retraction ring is much thicker than it is below it.

3. The wall below a retraction ring is both thinned and over-distended.

4. The presenting part is or has been jammed into the pelvis.

5. Part of the child must be below the retraction ring.

6. The body above a retraction ring is tonically contracted and hard.

7. Round ligaments stand out.

Contraction Ring.

8. A contraction ring may occur in the first, second or third stage of labour.

9. A contraction ring does not vary in position as labour goes on.

10. A contraction ring is rarely felt on abdominal examination.

11. The patient's general condition is good.

12. Causation.—Premature rupture of the membranes. Intrauterine manipulations.

Retraction Ring.

8. A retraction ring practically always occurs late in the second stage of labour.

9. A retraction ring gradually rises as retraction of the upper uterine segment proceeds.

10. A retraction ring may frequently be felt per abdomen (?).

11. The patient's general condition is bad.

12. Causation.—Obstructed labour.

The position of the ring is, roughly speaking, round the neck in 45 per cent.; below the whole of the child in 21 per cent.; round the arm in 15 per cent.; round the thorax in 9 per cent.; and round the after-coming head in 9 per cent. of recorded cases.

The difference between the condition of the lower uterine segments was well seen recently when attempting to excise a grossly infected tonically contracted gravid uterus without opening it. The tense thinned lower segment would not come up, and on attempting to put on curved clamps, in order to retain the infected fetus within the uterus, the lower segment cracked and ruptured. Whereas in the two cases where similar excision of infected contraction ring cases has been necessary, no such difficulty was met with.

In Dickinson's series of 90 cases, multiparae and primiparae were equally affected; in those that I collected it was twice as common in multiparae. Excluding laparotomy cases the maternal and fetal mortalities were 58 and 63 per cent.

TREATMENT.

Expectant treatment usually fails in a severe case. [Budin waited thirty-six hours, but no relaxation occurred.] Anaesthetics, morphine, and, in fact, all drugs are useless.

Dilatation.—In a slight case manual dilatation has succeeded. In severe cases the method of continuous weight traction advocated by Willett has been successful in several recorded cases. In my fourth case a fistula resulted, but she had had prolonged and repeated efforts made to deliver her before weight traction was used.

Forceps are useless. If the head is suspended by a ring round the neck, they usually slip off. If they do not fall off, intermittent traction merely pulls the child and uterus down together. Embryotomy is always particularly difficult, partly owing to the high position of the child; it is attended by a high maternal mortality and often fails altogether. Cleidotomy, as well as craniotomy, is necessary if the ring is round the neck, but often fails. Attempts to cut the ring from inside have been made, but are obviously futile. In a severe case, if manual dilatation and weight traction fail, laparotomy will be necessary. Laparotomy, in the cases collected in 1912, gave a maternal mortality of 7 out of 19. Now, including Hicks's three cases and my own last, it would be 9 out of 23, but in most of the old cases Caesarean section was not tried till all else had failed. If performed early, there is no reason why its death-rate should be higher than when performed for contracted pelvis.

REFERENCE.

¹ *Proceedings of the Royal Society of Medicine, Obstetrical and Gynaecological Sections, January, 1913.*

ON THE LIFE-HISTORY OF ASCARIS LUMBRICOIDES.

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In papers published in the *BRITISH MEDICAL JOURNAL* on July 1st and October 7th, 1916, the life-history of *Ascaris lumbricoides* was traced from the hatching of the eggs in the alimentary canal of rats and mice to the arrival of the larvae in the mouth of these animals. It has been shown that the larvae reach the mouth on the eighth day after infection and are still present in abundance in the lungs and trachea on that day.

Guided by later experiments, it is now possible to trace the course of the parasite through a further stage. Larvae have been found to persist in the lungs and trachea of

mice on the ninth, tenth, eleventh, and twelfth days after infection and to migrate down the alimentary canal on these days. This migration commences on the ninth day, small numbers only descending the bowel. A proportion of the migrants die during this day, and there is no accumulation in the large intestine. On the tenth day the migration is fully established. The organisms traverse the stomach and small intestine with some rapidity and accumulate in the caecum and large intestine. The process is continued on the eleventh and twelfth days. Up to the present I have not traced it beyond the twelfth day. After arrival in the large intestine, a large number of the larvae are voided in the faeces. I have not, however, worked out this emigration fully as yet, and therefore propose to leave its description to a later date. With this summary the experiments may be described in detail:

Mouse K on September 3rd received a dose of eggs of *Ascaris suilla* which were twenty-five days old. On September 12th, nine days after infection, it was killed. One moribund ascaris larva was found in the stomach, one active and one moribund larva in the upper portion of the small intestine. The caecum and adjacent portion of the large intestine contained very many examples of *Oxyuris*. One ascaris larva was found among them. The faecal pellets from the rectum did not contain any larvae. Numerous larvae were present in the trachea; none were found in the mouth.

Mouse L received a dose of eggs of *Ascaris suilla* thirty-four days old on September 13th. On the 22nd, ten days after infection, it was killed. No larvae were found in the stomach; two were found alive and active in the small intestine, between forty and fifty in the caecum, and about twenty in the large intestine. The caecum and large intestine contained also many *Oxyurids*, both adult and immature. The ascaris larvae can be distinguished readily from these forms even under a $\times 10$ magnification. The distinguishing marks are (1) their greater activity—they swim actively with a sinuous movement; (2) their dark colour due to the numerous granules in the intestinal walls; (3) their narrower body form.

The faeces passed by Mouse L on the 22nd were examined after twenty-four hours, and the following numbers of larvae were found in successive faecal pellets: Pellet 1, none; pellet 2, three larvae; pellet 3, two larvae; pellet 4, one larva; pellet 5, twelve larvae; pellet 6, none; pellet 7, one larva; pellet 8, six larvae. The faecal pellets were dry. The larvae did not move in any case, but half were perfectly fresh and unshrunk in appearance. The remainder were only slightly shrunk.

Mouse M received a dose of eggs thirty-five days old on September 13th, and was killed on the eleventh day thereafter. Numerous larvae were found in the trachea and roots of the lungs, none in the bases of the lungs. The stomach and small intestine were not examined. The caecum and large intestine contained many larvae together with *Oxyurids*. In the faeces of the previous night and of the eleventh day no larvae were found.

Larvae from the large intestine placed in tap water were alive and active after two hours, but had died after twenty-four hours.

Mouse P received a dose of eggs forty-two days old on September 20th, and was killed on the twelfth day thereafter, October 2nd. Active larvae were found in the lungs and caecum. One live and active larva was found in recent faeces, one dry and inactive larva in faeces twenty-four hours old.

Mouse R received a small dose of eggs twenty-four days old on September 26th, and was killed on October 6th, ten days after infection. One larva was found in the lungs, none in the trachea, caecum, large intestine, or faeces.

Measurements of the Length of the Larvae at Different Dates after Infection.

The larvae measured were in all cases killed in weak corrosive sublimate solution either cold or boiling, and were measured in this solution or in normal salt solution.

- 8th day: From lungs and trachea (alive), 1-1.8 mm.
- 9th day: From stomach (moribund), 1.3 mm.
- 10th day: From lungs (alive), 2 mm. (Mouse R). From intestines (alive), 1.3-1.65 mm. From faeces (inactive), 1.4 mm.
- 11th day: From the trachea (alive), 2.1 mm. From the large intestine (alive), 1.9-2.1 mm.
- 12th day: From the lungs (alive), 1.25-1.9 mm. From the caecum (alive), 2.02 mm. From fresh faeces (alive), 1.6 mm.

The words in brackets indicate the condition of the specimen before fixation.

It will be observed that the results given above probably supersede two statements made in my second paper on ascaris infection, published in the BRITISH MEDICAL JOURNAL on October 7th, 1916. The statements in question are: (1) "No larvae were found in the respiratory tract later than the eighth day after infection." (2) "It is probable that they" (the ascaris larvae from the lungs of rodents) "emigrate in the saliva of the rodent on to food substances such as bread."

The first statement was based on the following observations:

1. Rat B, killed sixteen days after the first infection and twelve days after the last. No larvae were found in the lungs, trachea, stomach, or intestines.

2. Rat F, killed twelve days after the first and nine days after the last infection. No larvae were found in the respiratory tract; one dead larva was found in the stomach and one in the rectum.

It is, of course, possible that the larvae normally leave the respiratory tract of the rat at an earlier date than is the case in the mouse. The larvae from the intestine of Rat F were identical in appearance with those from the intestine of mice.

The second statement, that the larvae probably emigrate in the saliva of the rodent, rested on the presence of larvae in the mouth, and their power of survival on damp articles of food such as bread, and on the apparent exclusion of the other two possible lines of emigration—namely, through the bowel and through the nares. The following observations appeared opposed to emigration through the bowel:

1 and 2. On Rats B and F—given above.

3. Rat A received large doses of ascaris eggs on four days between April 6th and 10th. It suffered from acute ascariasis. Its faeces were examined on April 17th, twelve days after the first and eight days after the last infection, and again on the two following days. No larvae of ascaris were found in them.

4. Rat B was infected on the same days as Rat A, and suffered from acute ascariasis. The faeces were examined on April 17th and no larvae were found.

It will be noticed in the experiments on mice given above that the appearance of larvae in the faeces is extremely irregular.

5. Mouse D, killed eight days after infection; no larvae were found in the caecum or large intestine.

The following observations were opposed to emigration through the nares:

1. Rats B and F: No larvae were found in the nasal cavities between nine and sixteen days after infection.

2. Mice B and D: Killed on the eighth day after infection; no larvae found in the nasal cavities.

Larvae were found in the mouth on the eighth day after infection on three occasions—in mice H, G, and J.

THE USE OF FILTERED X RAYS FOR THE RELIEF OF FIBROUS BANDS AND ADHESIONS RESULTING FROM BULLET WOUNDS.

BY

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EXPERIENCE in the value of x rays in keloid and hypertrophic skin scars suggested to me that, with the addition of a filter, the same treatment might be of use in removing new fibrous tissue giving rise to trouble in deep wounds. I discussed this idea with Colonel Woolbert, I.M.S., in medical charge of York Place Division of the Pavilion Military Hospital, and he kindly placed certain of his cases for me to treat.

The same method was adopted in each case. The tubes chosen for the treatment were of hardness 7 on the Benoist scale. The rays were passed through a thin filter of aluminium 0.5 mm. A weak coil and a mercury dip break set to give 1,000 interruptions a minute, and requiring about 21,000 to 23,000 interruptions to give a Sabouraud dose of filtered rays, were used.

CASE I.—Bullet wound scars, entrance and exit above elbow-joint front and side. The wounds were perfectly healed, but the full extension of the joint was only about two-thirds of the normal. Brachial artery pulsation could be felt to a little below the position of origin of the anastomotic artery, and was then lost; the radial pulse was very weak; there was extensive loss of sensation of forearm and hand, and weakness of grasp. After the first Sabouraud dose of filtered rays rapid improvement followed, the extension of the arm became nearly normal, the sensation returned a little, and the grasp became stronger. A fortnight later a second Sabouraud dose was given, and after a week extension became quite normal, sensation was vastly improved, and the grasp strong. There was no change in the radial pulse. The patient was then discharged fit for active service. The rays in both exposures were discharged in the direction of the bullet track, the tubular speculum of lead glass touching the skin at the scar of entrance.