thus placed on the heart leads to a hypertrophy which can maintain compensation only over a limited period except under the most favourable conditions. The period of childhood and even of adolescence may in cases of chest deformity be occasionally survived without any considerable degree of inconvenience, and the result of excessive cardiac strain may manifest itself first in adult life. Professor Muir has recently shown me the heart of an adult who had a very marked thoracic distortion, and who died with the signs and symptoms of cardiac failure. In this case there was a greatly hypertrophied heart, which exhibited the broad quadrilateral shape described above. Hence, from the examination of cases of pulmonary disease and of distortion of the thoracic wall, there is considerable evidence for the view that healthy lungs and a healthy chest wall are indispensable for the maintenance of an efficient circulation.

HEPATIC DISTOMIASIS (SHEEP ROT) IN MAN.

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TRENTLER, in 1793, recorded a case in which he found a parasite protruding from a ruptured vein in a man's leg. Although he gave it the name of *Hæmathyridium venarum*, it was in all probability a specimen of *Fasciola (distoma) hepatica*, the liver fluke, and his case the first reported of its occurrence in man. Since that time twenty-three other cases have been described, but some of them are almost certainly not authentic, and have been subjected to very damaging criticism; thirteen or fourteen survive as true examples of what must therefore be regarded as a very rare disease; of these at least four were noted in Great Britain.

Four other species of the *Fasciolidae* are found in man, of which the *Distoma sinense* is the most important, occurring extensively in Japan, China, and India. All these affect the liver primarily. A nearly allied pulmonary parasite (*Distoma westermani*), responsible for a disorder characterized by cough and hæmoptysis, is found in the Far East, cases being sometimes imported into the United States of America. Other forms of distomiasis are due to certain intestinal parasites, and occur mostly in India, while in Egypt especially the *Bilharzia hæmatobia* is responsible for much chronic ill health.

LIFE-CYCLE OF PARASITE.

The *Fasciola hepatica* is found in the gall bladder and bile ducts of sheep. More rarely it is parasitic in cattle, but in them the disease does not run so acute or fatal a course. The eggs of the adult fluke are conveyed by the bile into the intestine, and so eventually reach the water or damp grass which is essential to their further development. After a time, varying from three or four weeks to as many months, according to the time of year, a ciliated embryo emerges from each egg by pushing of the lid. These embryos are known as miracidia, and soon enter a small snail—*Limnaeus minutus*—there to form sporocysts. Although sporocysts may be formed in closely-allied species, no further development takes place. In the *Limnaeus minutus* each sporocyst produces a large number of rediae, and from these again are formed cercariae, which leave the snail and lead an independent existence. When living in water, they appear to the naked eye as small whitish bodies. They may then encyst themselves on the stems of grasses, and in that condition are capable of surviving extremes of heat or cold, and even desiccation.

The cercaria is that stage in the life-cycle at which the fluke becomes parasitic in mammals, and may be ingested while actually in the snail, in drinking water, or on vegetable foodstuffs. It is probable that drinking water chosen at the dictates rather of necessity than desire, and watercress, sorrel, etc., are responsible for the transmission of the parasite to man.

The cercariae, having reached the digestive tract of man, obtain access to the bile ducts, where they attain adult size—about 30 mm.—deriving the necessary nourishment

from the blood of the host, which they imbibe after attaching themselves to the mucous membrane. The ova are then deposited in very large numbers; the fluke dies and becomes encysted, or is excreted.

The discovery of this life cycle dates from the observation that the parasite was never found in sheep whose pastures were contiguous to the sea or to salt marshes. Salt has been found an efficient prophylactic, and is used as such in veterinary practice.

INFECTION IN MAN.

It is obvious that there must be many occasions on which this parasite in the cercaria stage reaches the human alimentary canal, for in many parts of the country much of the drinking water is derived from ponds to which sheep have free access. Yet the disease is rare. This points to the fact that man is by no means an ideal host. It is not unreasonable to suppose that the habit of taking salt with vegetable food has something to do with this, or it may be that his digestive secretions either in their nature or concentration are inimical to the parasite.

The presence of only one or two flukes has been frequently recorded. Murchison's case was that of a man who died after attention in hospital for other reasons. A solitary fluke was found in his liver *post mortem*. On the contrary we find records of more than a thousand parasites in one sheep, and of considerable numbers in man. Perhaps those instances of the finding of only one or two are cases in which a more than usually adaptable fluke has survived in an essentially inimical host; those of the finding of large numbers must, then, be held to signify the loss of resistance on the part of the host towards flukes of no especial virulence.

It is by no means improbable that some instances of this disease have been treated as cirrhosis of the liver or cancer; the means of diagnosis are least available in the remoter country practices where the disorder is most likely to occur.

COURSE OF DISEASE IN MAN.

The *Fasciola hepatica* has been so seldom observed in man that it is not easy to judge of the symptomatology from the recorded cases. A consideration of the disease as it affects sheep, and of the effects of the allied parasite *Distoma sinense* will, however, allow one to arrive at a fair idea of the disease process.

The period of invasion may pass unnoticed clinically, and in the early part the host may gain weight and enjoy a good appetite. There may, however, be slight jaundice and moderate enlargement of the liver. In sheep the minimum duration of this stage is six weeks; in man it is probably longer. Secondly, the stage of anaemia is recognized, and this is supposed to coincide with the maturity of the flukes. Anaemia of the mucous membranes is the most obvious sign, but the symptoms are rather those of toxæmia; in the case about to be considered they were certainly quite disproportionate to the anaemia. Appetite fails or may become depraved, the temperature is irregularly elevated, and the frequency of the pulse increased. Gastric pain and intestinal disorders become evident, diarrhoea and vomiting are not unusual, constipation may be present. The eyes are lustreless and the skin dry; experienced veterinary surgeons have been known to base a diagnosis on the condition of the skin alone.

Slight exertion aggravates the dyspnoea which is present, and the third stage soon appears. This is the stage of emaciation and ascites, and is accompanied by increased anaemia and progressive hepatic derangement. Oedema of the eyelids, conjunctivitis, hydrothorax, bronchitis, ascites, and jaundice are among the common symptoms.

The fourth stage seems to correspond in time with the emigration or excretion of the parasites, and in those who have survived the third is one of slow recovery from toxic symptoms. Fibrosis and other changes in the liver remain, and may cause permanent ill health.

An infinite variety of catastrophes may ensue from the presence of flukes in parts of the body other than the liver. These migrations are by no means uncommon; 10 per cent. of all cattle affected are found to have flukes in the lungs, and in the human being they have been seen even in the eye. One case was that of a child, 2 months old, who had an abscess of the scalp, which contained liver flukes; in another they were found in the sole of the

foot. There seems no reason why any intermediate region of the body should not be affected. Abscess of the lung and general peritonitis are among the possible causes of death in this disease.

MORBID ANATOMY OF DISTOMIASIS.

The chief changes are those in the liver, which will therefore be described first. The calibre of the ducts is

increased considerably, and the walls show a proliferation of the epithelium, forming small papillomata. Outside the ducts is an area of inflammation and an accumulation of eosinophile cells, generally very marked, according to Brumpt. In later stages fibrosis is extreme, and calcification may occur. A case is on record of a calcified cyst the size of a hen's egg containing about half a dozen flukes. The inflammation spreads to the surrounding hepatic substance, and in the presence of pyogenic organisms pus formation naturally results. The liver as a whole may show extensive perihepatitis, and cysts may be seen protruding on the surface. Where these have leaked adhesions will be found. The cirrhosis may reach a very marked degree.

In the same way the presence of flukes in other parts of the body is followed by inflammation, eosinophile infiltration, and either fibrosis or abscess formation. The other morbid appearances are those found in cases of severe anaemia, and those proper to wasting diseases. They call for no particular mention; the digestive system may show little abnormal or may be the site of extreme catarrh or even ulceration.

REPORT OF A CASE.

E. D., male, aged 31, was admitted to the Tunbridge Wells General Hospital on November 10th, 1910, under the care of Dr. L. Adeney, by whose kind permission I made the investigations detailed below, and now record the case.

Dr. Adeney had seen the patient before admission in consultation with Dr. Bayfield, of Southborough. A provisional diagnosis of tuberculous peritonitis had been made, but he was admitted for the further investigation. He gave a somewhat indefinite history of about three months' failing health with intermittent "indigestion." There had been no outstanding or striking symptoms of any sort, but an increasing inability to do his work—that of a barber—had caused him to seek medical advice.

At the time of admission the following points were noted. He was a small, slight man of delicate appearance; eyes a little prominent; thyroid normal; skin dry, slight yellow tinge; conjunctivae anaemic, slight but distinct; subcutaneous fat ill developed; heart sounds distant; pulse small volume, increased

frequency, tension poor; abdomen slightly distended; some epigastric tenderness; no pain at time of examination; liver not enlarged; spleen not enlarged; no "doughiness"; no signs of tuberculous foci in lungs or elsewhere. Complaints of weakness, occasional headache, and abdominal pain mostly after meals. Is "very glad to get to bed." Previous illnesses nil. Family history good.

It may be mentioned that he weighed only 6 st. 3 lb. a month later.

A few days' observation showed that his pulse-rate and

temperature were persistently and synchronously elevated towards evening. The temperature fell to normal during the day, but the pulse remained above 100. The respirations averaged 24; this number fell to 20 during convalescence. These variations and the return to normal under treatment are well shown by the accompanying chart.

A blood examination was made in the hope that it might throw some light on the condition. It did—in an unexpected fashion. On November 14th there were 37.2 per cent. of eosinophile leucocytes and 3,757 per cubic centimetre, as against

a normal of 1 to 2 per cent. in adults, with a total of 150 to 250 per cubic millimetre. This increase was so marked that an explanation was immediately sought for. Two frequent causes—namely, asthma and skin diseases—were readily excluded, but it was elicited that the patient had suffered from threadworms. The resulting pruritus ani had, however, been absent for more than nine months; nevertheless it was thought advisable to give him anthelmintics for a time. Santonin and calomel, of each 2 grains, were therefore administered nightly. After fourteen days his blood was again examined, and showed

41.6 per cent. of eosinophiles, with a total of 6,157 per cubic centimetre. The faeces were therefore examined, and the ova of *Distoma hepaticum* found in considerable numbers—four or five in each preparation.

Although the general condition of the patient was improving the haemoglobin was still little above 70 per cent., that is, had not altered since admission. The red blood corpuscles, on the contrary, were above the generally accepted normal of 5,000,000 per c.mm. There can, however, be no doubt that this figure is too low, at least in country districts.

The possibility that tuberculosis might be present as a complication of the distomiasis was considered, but the cuti-reaction was negative and an injection of 100 mg. tuberculin K.R. gave rise to no general reaction. There was a little soreness locally. It did give rise, however, to a remarkable increase in circulating eosinophile leucocytes, which rose to 9,003

per c.mm., a figure not previously nor thereafter recorded in this case. A reference to the table will show the actual differential count. This eosinophile leucocytosis following tuberculin injections has been noted by several authors. Grawitz found as many as 41,000 per c.mm. in one case.

By the second week in December the patient's condition had much improved, and except for some flatulence he appeared to be in normal health. On December 18th and 19th he had sharp pain on the right side at about the level of the ninth rib in the mid-axillary line. It disappeared on December 20th and did

Chart showing variations in pulse-rate, temperature, and respiration, and condition of blood.

Fig. 1.—Ova of *Distoma hepaticum*. a, Showing position of germ area; b, showing asymmetrical form; c, artificially broken, with yolk granules pressed out; d, with lid missing but yolk present; e, typical form. (Drawn by Author from the preparation.)

Table showing Absolute Number of Cells per Cubic Millimetre.

Date.	Red Blood Corpuscles, per c.mm.	Haemoglobin, per Cent.	White Blood Corpuscles, per c.mm.	Polymorpho-nuclears.	Eosinophiles.	Mast Cells.	Transitional Leucocytes.	Lymphocytes.	Large mono-nuclears.
Nov. 14 ...	4,430,000	73	10,100	3,373	3,757	20	606	1,879	465
Nov. 28 ...	4,700,000	72	14,800	5,150	6,157	118	296	2,871	207
Nov. 28* ...									
Nov. 29 ...	5,350,000	72	16,930	3,826	9,007	106	339	3,251	406
Nov. 30 ...	5,650,000	71	11,160	2,902	5,826	112	223	1,964	134
Dec. 4 ...	4,750,000	76	15,000	4,710	6,660	120	600	2,580	330
Dec. 9 ...	5,650,000	74	10,400	3,120	3,578	125	437	2,766	374
Dec. 9† ...									
Dec. 14 ...	5,400,000	82	12,300	3,567	6,199	221	541	1,378	394
Dec. 19 ...	6,512,000	80	12,400	3,745	5,010	149	422	2,579	496
Dec. 31 ...	5,340,000	94	10,900	6,380	2,310	43	286	1,760	110
Jan. 4 ...	5,640,000	90	11,450	2,830	4,876	114	368	3,165	207
Normal ...	5,000,000	90-100	8-10,000	6-8,000	150-250	50	3-500	1,500-2,500	2-400

* Injection of tuberculin.

† Arsenic and iron discontinued. Administration of euonymin commenced.

not recur while he was in hospital, but was present again about fourteen days after his discharge. This may have been due to some localized perihepatitis, but there was no direct proof.

In spite of the improved health the number of ova in the faeces did not diminish, so that we cannot suppose that the flukes were affected. This was hardly to be expected, and it was on the whole gratifying that his health had improved so much.

There are several points of interest about this disease, which is commonly known as distomiasis. This term, however, will include any disease caused by flukes, even that due to bilharzia. Hepatic distomiasis is more accurate, and includes only disorders which much resemble each other, although caused by different species of fluke. The veterinary colloquialism, namely, "sheep rot," is a graphic description of the disease in sheep, but lacks elegance, and can hardly be adapted to meet the case in medical nomenclature.

ETIOLOGY.

This has been in part discussed already. Briefly, there must first be the ingestion of cercariae, and it appears that the encysted form on vegetables is most often eaten. There must, then, be a sufficient concentration of alimentary secretions to dissolve the cyst. Evidence points to the gastric juice as the most important secretion from this point of view. The cyst wall being digested, there must further be an absence of any substance—for example, salt—which is fatal to the parasites. If these conditions are fulfilled the cercariae are probably able to reach the blood vessels and even the bile ducts. Their continued development depends on an adequate food supply of a proper nature. The normal food is the blood of the host. Weinberg has shown that this produces antibodies, presumably toxic to the parasite, but perhaps only of a nature to neutralize its toxin. Previous debilitating conditions or—as in the case recorded—general under development might impair the production of these antibodies, and so favour the growth of the cercariae, or, on the contrary, the parasite may become habituated to them. Probably both processes occur, and decide together the growth or death of the parasites.

DIAGNOSIS.

The diagnosis rests on five points—namely, the discovery of the ova in the faeces, or of the parasites in any situation, the clinical signs, the eosinophile leucocytosis, and the precipitin reaction. The eggs are invariably present in all but the mildest cases and the earliest stages. The discovery of actual flukes is unusual, but has called attention to some of the recorded cases. The clinical signs and symptoms are not likely to lead to a diagnosis in this country owing to the infrequent occurrence of the disease, but are of the first importance in veterinary surgery. The presence of eosinophile leucocytosis has not been recorded in any other case, but the local eosinophile infiltration noted by Brumpt is presumptive evidence of its general appearance. The precipitin reaction, as acknowledged by Weinberg himself, is of theoretical rather than practical

importance. The modern extension of facilities for investigations in clinical pathology may be expected to make the discovery of the ova the most important diagnostic point, as it is now almost the only absolute one.

PROGNOSIS.

The prognosis can hardly be regarded as favourable in view of the migrations in the body of the parasite and the changes produced in the liver. The onset, too, is insidious and much harm may be done before medical advice is sought. Moreover, it is at least possible that only weakly persons are the subject of the disease except in its mildest form. On the other hand, if the infection is not extensive, and if the patient is in a position to procure good food and good hygienic conditions, it should be possible to prevent re-infection and compensate for any damage already done. The subject of this paper had two symptoms which made one particularly cautious in the prognosis, namely, a large number of ova and pain over the liver; the persistence of the latter, even when his general condition was excellent, was considered distinctly unfavourable as probably pointing to some hepatic derangement, of which the ultimate consequences might easily prove fatal.

TREATMENT.

Treatment with anthelmintics is of no value, since they are not excreted in the bile and so cannot reach the parasites; moreover, any drug that was excreted in the bile in sufficient quantities to kill the fluke would probably kill the host as well. As a matter of fact, we do not know of any drug which is secreted in the bile or circulates in the blood in sufficient concentration to act as a vermicide. Quinine is certainly a protoplasmic poison, and exists in the blood as such, and the same is true of arsenic; but these do not seem to have any effect on blood-sucking parasites. It was therefore resolved in this case to endeavour to stimulate the secretion of bile, with the idea that toxic substances should be washed out and their absorption perhaps prevented. For this purpose euonymin was given three times a day in pills containing 1 grain. A marked rise in haemoglobin certainly took place under this treatment, but it cannot be asserted that this was due to the comparatively small doses of euonymin and not to rest and improved hygiene.

Symptomatic treatment follows the usual lines, and even surgical treatment may have to be invoked.

Prophylactic treatment is hardly called for in man, but consists of the administration of common salt with any food likely to be affected.

THE MIGRATION OF THE FLUKES IN THE BODY OF THE HOST.

Adult flukes have been found encysted in the lungs, and even in the subcutaneous tissues. It seems hardly credible that an adult fluke could pass in that state through the heart and large vessels. It must therefore be assumed

that the cercariae reach the blood stream, in which case this parasite has almost as much right to the appellation "haemato-bia" as has the bilharzia, its near relative. If the presence of flukes in unusual situations be due to migrations of their immature forms (the cercariae), it follows that such migrations will take place at a fairly definite period of the disease, at or shortly after the time of invasion. This is important from the prognostic point of view, for in the absence of reinfection we ought to be able to say that after a month or so the damage will be confined to those positions which the flukes then inhabit. It will also appear that the tendency of the organism is to encyst the parasite and thus limit its activities. Virtually sepsis is then the only danger to be feared except in rare instances, and apart from structural changes in the liver. There is the further question as to the way in which the cercariae obtain entry into the vessels. Since they can, it seems, survive in the stomach and duodenum, there is no obvious reason why they should not penetrate the mucosa, and reach the vessels in those organs. They would thus obtain access to the liver. Further, it requires some degree of imagination to suppose that as many as a thousand cercariae may unanimously choose the bile duct as a suitable path in life. It is true that larger worms are not very infrequently found in the duct. Sick collected 64 cases of *Ascaris lumbricoides* in that situation, but the invasion of some hundreds of parasites by this route is a very different matter. On the other hand, we have the undoubted fact that the flukes are found in the bile ducts; this may be explained by supposing that they reach the ultimate ramifications of the canaliculi directly from the smallest portal capillaries—a supposition which appears by no means improbable. It may be mentioned, in support of the view that the gastric mucosa is primarily affected, that there is a case on record of a French army officer who ate some meat infected with an allied parasite (*Distomatos bucco-pharyngée*). The parasites immediately fixed on the walls of the pharynx and mouth in such numbers that he was in danger of suffocation. In the same way, it may be, the *Distoma hepaticum* fixes on the mucosa of stomach and duodenum—probably, indeed, on that of other parts of intestine.

The alternative view has never been proved experimentally, and rests on pure conjecture. It is somewhat difficult to explain, except on the first hypothesis, why the pancreas is not also invaded, since its secretion does not appear to be lethal to the parasites and the road is open. If they do, indeed, traverse the common duct, why should they all take one turning? The presence of larger worms in the gall bladder and not in the pancreas is explained by the lack of space in the latter situation, but surely there is room for a few cercariae.

THE ANAEMIA.

In the case under consideration the anaemia had not reached any great degree, and there are no available records of other cases. It is obvious that it depends on the presence of the parasites in some way, and therefore comes into the great clinical group of parasitic anaemias. It further resembles some of these in the fact that slight loss of blood is an aggravating factor. It is further very probable that sepsis may be a further intensifying agent, but neither this nor the haemorrhage are likely to be sufficient in degree to cause any great disturbance.

The eosinophile leucocytosis, the elevation of temperature, and the increased pulse-rate suggest the presence of some toxic substance absorbed into the general circulation. The fact that antibodies can be demonstrated in the blood is further evidence. The question then arises whether this toxic material is derived from the living or dead fluke, from its secretions or excretions.

It would be convenient to suppose that it arose from dead or decomposing parasite, for this will explain its absence in some cases (which one may suggest from analogy with the case of *Distoma sinense*) and in the early stages. A similar suggestion has been put forward to account for the varying occurrence of anaemia in *Bothriocephalus* infection, but although there is some direct evidence in that case there is none with regard to distomiasis. This view is also negatived by the presence of eosinophilia around living parasites.

In favour of the origin of the toxin from the secretions of

the parasites is the isolation of such substances from various intestinal parasites. The latter, however, were not only certainly dead, but desiccated when the extracts were made, and, moreover, the results of these experiments have not always been verified by subsequent observers. Another suggestion, made in the case of ankylostoma, is that the excreta collect in large masses in the intestine and give rise to the toxæmia. There is a certain amount of inherent probability about this view, and it would apply with more force when the excretions are deposited in the bile capillaries where there is little pressure to remove them.

Whatever the precise source of the toxin there can be little doubt, though its nature is quite unknown, that it is the most important agent in the causation of the anaemia.

The type of anaemia etiologically comes under Hunter's heading of "Septic Anaemia," which presumably includes all anaemias due to the products of organisms foreign to the body. In the absence of a promised volume by the author mentioned, Part II of his "Severest Anaemias," as yet unpublished, the above classification is made with all reserve. Haematologically the anaemia is distinctly of a "chlorotic" type. The following data are derived from ten blood examinations carried out in this case:

1. The number of red blood corpuscles was never below 4,430,000 per c.mm., and under treatment with iron and arsenic rapidly rose to normal. Within fourteen days it had reached 5,350,000, and later a maximum of 6,512,000 was recorded. A fairly constant average of 5,500,000 was established after a fortnight.
2. The haemoglobin remained practically constant at 70 to 75 per cent. for a month, and then rose but slowly, never reaching above 94 per cent. during the period of observation. It did not commence to rise until iron and arsenic had been replaced by euonymin.
3. The size of individual corpuscles was at first below normal—namely, 6.93 μ ; at the last examination it was 7.23 μ . At first 62 per cent. were between 7 μ and 8 μ ; finally as many as 89 per cent. fell between these limits. There was thus a distinct improvement in both respects.
4. Poikilocytosis was never well marked, and varied but little.
5. Polychromasia was present throughout, but was slight.
6. Basophile granulation was also present throughout and was very considerable in degree, even at the last examination.
7. The viscosity of the blood was very obviously diminished at first, and even when the corpuscles were 6,512,000 per c.mm. did not seem to be increased.
8. The leucocytosis, which reached 16,930 per c.mm. on one occasion, was due to an increase of eosinophiles; the other cells were present in normal numbers. The mast cells, however, reached 1.8 per cent. on one occasion—absolute number 221 per c.mm.

The absence of pigmentation, urobilinuria, or of any of those forms of red blood corpuscles which appear after extensive blood destruction, sufficiently attests that this is not a "haemolytic" anaemia. The increased fluidity of the blood, the quick rise of red blood corpuscles and its variable extent, the increase of total blood volume known to occur in ankylostoma anaemia, and the close resemblance to chlorosis shown in the size, etc., of the corpuscles, lead one to suggest that there may be some degree of hydraemia in these cases. This is not, however, a certainly trustworthy deduction; indeed, all deductions drawn from one case only are to be accepted with caution. Moreover, if the anaemia depended solely on dilution of the blood, the haemoglobin percentage ought to rise at the same rate as the percentage of red blood corpuscles. This was not the case, and we may therefore feel certain that there is in this anaemia some perversion of the normal process of forming red cells. Apparently this is shown first by a deficiency of haemoglobin, and in the size and contour of the cells. Later there would be an actual diminution in output of cells, and the qualitative changes would become more marked. It is hard to avoid the conclusion that this is due to some poison circulating in the blood and so reaching the marrow. The polychromasia and stippling may both be due to the same influence, but it is more probable that they have different significances.

Polychromasia is too vexed a subject to discuss here; but if it be regarded as an infallible sign of the extension of blood formation, it is difficult to understand why it persisted in this case long after the blood was normal, and in the absence of any drug which is known to affect the marrow. As regards the basophile granulation, bearing in mind its immediate appearance after small doses of metallic toxins, the author inclines

very much to the view that it represents some adverse effect produced on the red corpuscles while they are actually in the circulation. It will be noted that this granulation showed no signs of diminishing at any time, which accords with the probability that the formation of toxin by the parasites was equally undiminished.

To summarize, the anaemia resembled very closely that ordinarily accompanying chlorosis and showed no signs of being due to haemorrhage. It may be classed as a septic or, better, toxic anaemia, the essential haemolytic lesion being probably aplasia of the marrow.

The eosinophile leucocytosis is interesting and certainly depends on the need for such cells in the immediate vicinity of the parasites. Their presence in such situations has been noted in this and many other allied parasitic diseases, notably trichiniasis.

It has been observed also that when the parasite is well encysted or calcified the eosinophilia disappears. Eosinophile cells have never been regarded as phagocytic in the same way as polymorphs. Is it not very probable that they are chemical phagocytes—that is, neutralize or destroy adverse chemical substances—among which the toxins of parasites may well have a place? It is also at least credible that this power resides in the granules. It has been observed that in the exudate from tick bites in the horse, in which animal the eosinophile granules are particularly large, cells are found in which only one or two granules remain. This may well point to the fact that the rest have been used up and have ceased to be demonstrable by ordinary staining methods.

Now if the output of eosinophile cells was adequate, the toxin would presumably be neutralized before it could do harm, and the anaemia would not occur, and such cases would not seek medical treatment. If at the same time the parasites were speedily so surrounded by impermeable tissue that the toxin did not reach the circulation, there would be no further stimulus to eosinophile leucocytosis. On this hypothesis may be explained the absence of this sign in many cases of infection by parasites which usually give rise to it. On the other hand, the toxæmia might be so intense that eosinophile production was paralysed or exhausted. Eosinophile leucocytosis is, in fact, on a par with polymorphonuclear leucocytosis, which is absent in cases of walled-off abscess and in pneumonias without reaction. The one is excited by the toxins of bacteria, drugs, etc., the other by those of parasites in many cases. The apparent anomalies in the occurrence of eosinophilia are due not to erratic natural laws, but to a lack of information as to the formation and absorption of the toxins which call it into existence.

THE OVA.

The eggs are oval in shape, as a rule; not infrequently the greatest diameter is midway between the two poles, but is, as a rule, towards one end. They are sometimes asymmetrical, as seen under the microscope, and this, perhaps, is due to their lying on one side. The proportion of length to breadth varies very considerably. Measurements of a large number gave an average length of 137μ , with an average breadth of 71.7μ . This is twice as large as the egg of *Bothriocephalus latus*, which in some ways resembles that of *Distoma hepaticum*. The maximum length was 156μ , the minimum 118μ , the maximum breadth 91μ , the minimum 62μ .

The colour of the egg varies from a light yellow to dark brown, they stain very deeply with iodine, but hardly at all with aniline dyes.

At one end of the egg is a cap or lid from $30-40\mu$ in diameter. This is not always very obvious; it may sometimes be demonstrated by tapping the cover-glass, but this usually cracks the egg in some other place. Within the egg are a number of spherical bodies known as the yolk granules. Some of these are $20-30\mu$ in diameter, and possess smaller granules in their interior, the latter arranged in a rosette. The smallest yolk granules measure $4-6\mu$, and appear to consist of two concentric spheres. Both varieties stain slightly with methylene blue, and appear to rest in a nearly homogeneous material, which fills the egg. Towards one end is an area known as the germ area; by the division of its cells the miracidium is formed. The yolk granules disappear, and are said to provide nourishment for the growing embryo. Their comparatively elaborate structure is worthy of note. No stage

beyond that of the faint appearance of a germ area is seen in the faeces except after incubation, which may be carried out in an incubator in winter, or without in summer. It may be months before the miracidia develop. The writer has not been able to see it, although the ova have changed a good deal in colour and even lost their lids.

A résumé of bibliography is given in the following:

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Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

AN EPIDEMIC OF INFECTIOUS JAUNDICE.

An epidemic of jaundice has been under my observation for over three months in a village of 300 inhabitants. A brook running through the place receives some sewage from kitchens, but there are no closets emptying into it. The soil is a mixture of clay and gravel. The first case that came under my notice was in the middle of November last year, and there have been some 25 cases since, and fresh cases still crop up. The ages of patients vary from 2 years to—in one case—60 years, and when one member of a family is affected almost invariably the other children in the house contract the disease in a week. The school-mistress, aged 18 years, was ill in January. The symptoms were much the same in all, though the temperature varied a little; there was yellow staining all over the skin and conjunctiva, the urine was dark with bile, the stools of the usual clay-like appearance, and there was constipation, sickness and general malaise, inability to take food, particularly fatty substances, rise of temperature, constant pain over the liver, headache, and loss of flesh and muscular strength. The treatment consisted in keeping the patients warm in bed, light food, and magnesia and cascara as medicine. So far they have all recovered.

The cases are not entirely limited to the village described, for recently in my own village, which is three miles distant, there has been an outbreak in a family. The first patient was a girl aged 8, who had consorted with children from the village first infected, and subsequently two small children in the same family fell ill. Can all these cases be an instance of an outbreak of the febrile and infective jaundice recorded by Weil of Heidelberg in 1886, and described under the title of Weil's disease in Allbutt and Rolleston's *System of Medicine*?

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CONTINUOUS ANTISEPTIC INHALATION IN PULMONARY CONSUMPTION.

DR. PEREIRA has spoken of giving a patient a respirator saturated with an antiseptic. If 4 or 6 drops are put in the respirator every hour or two, it is not saturated. A woman, 30 years old, has been under my care since February, 1908. From that time till December, 1909, she suffered from cough, profuse expectoration, night sweats, diarrhoea, and three attacks of haemoptysis, one of which nearly proved fatal. The emaciation was extreme. We expected an early termination; as the friends said, "It would be a happy release." During this time (almost two years) I had relieved her symptoms as they occurred with medicine. In December, 1909, I saw a paper by Dr. David Lees in the JOURNAL. I applied the treatment as he recommends it; the effect has been remarkable. She has not required a dose of medicine in 1910. Cough was relieved, sputum diminished, there has been no attack of haemoptysis. General condition so much improved that she can bear to be taken out considerable distances in a chair without fatigue. She has taken sufficient antiseptic solution during these fifteen months to have either poisoned or bled her to death, that is, if the treatment properly used can have such deleterious effects. I have treated several cases in the slums of the town, and they have invariably been much relieved. Sufficient time has not elapsed to pronounce them cured, but they are all in a fair way of recovery.

Rochdale.

W. J. HODGSON, M.D.