daily administration of a pint of the best dry champagne for a week. In connexion with this matter, it is only just to state that the committee of the hospital have never once questioned such expenditure.

I should be sorry to conclude this paper without recognizing the services of my staff. We all formed a machine, each part of which was indispensable to the whole. I often wish I could have the chance of similarly burying my individuality when operating in houses and homes. And in order to show the confidence I have had in my assistants, Drs. Walker and Pringle, I need only state that accident and emergency operations are their perquisite.

THE PRODUCTION OF GENERAL SYMPTOMS IN HAEMOGLOBINAEMIA.

BY

J. O. WAKELIN BARRATT, M.D., D.Sc.,

AND

WARRINGTON YORKE, M.D.

IN the course of a previous investigation upon the effect of intravenous injection of laked red cells,¹ the conclusion was reached that the effective agent in causing an alteration of the general condition of the animal injected was represented by the red cell stromata, and not by the haemoglobin given up by the red cells when laked. As these observations appeared likely to throw some light upon the origin and nature of the general symptoms attending haemoglobinaemia due to the destruction of red blood cells, such as occurs naturally in paroxysmal haemoglobinuria and in blackwater fever, it was determined to repeat on a more extended scale some of the observations already made.

To this end the series of experiments recorded in Table I was made. These experiments fall into three groups. In the experiments of the first group the red cells of rabbit's blood, after twice washing with 0.85 per cent. sodium chloride solution, were laked by the addition of an equal volume of distilled water, solid sodium chloride being added at the end of five to ten minutes in amount equal to 0.85 per cent. of the distilled water employed. A portion of the liquid so obtained was injected intravenously into rabbits. The rest of the liquid was centrifugalized, and thereby separated into a clear supernatant fluid, ruby red in colour, containing haemoglobin, and a viscid jelly-like sediment of stromata. The supernatant fluid was pipetted off and used for intravenous injection of a second group of rabbits; the sediment was mixed with an equal volume of 0.85 per cent. solution of sodium chloride, and the stromata were then distributed uniformly in the liquid by stirring with a glass rod, the liquid so obtained being employed for intravenous injection of a third group of rabbits. In all experiments the injection was made slowly, 3 c.cm. of fluid a minute being delivered into the vein of the ear.

It will thus be seen that three liquids were used for injection. The first contained dissolved haemoglobin together with suspended red cell stromata; the second contained dissolved haemoglobin in approximately the same concentration as the first but was free from stromata; the third contained both stromata and dissolved haemoglobin, the amount of the former being the same as in an equal volume of the first liquid, while the concentration of dissolved haemoglobin was approximately half that present in the first liquid. In the tables the haemoglobin and stromata respectively are expressed in terms of the volume of red cells required to furnish the amounts injected, and, for convenience of comparison of the experiments one with another, these quantities are given per kilogram of body weight of the animal used for injection.

Comparing the three groups in Table I, it is seen that in those experiments in which red cell stromata were injected (the first and the third groups) death or severe symptoms usually followed injection, the amount of stromata employed per kilogram of body weight of the rabbits being represented by amounts of red cells ranging from 4 c.cm. to 20 c.cm. In experiments 4 and 21, in which the amounts of stromata per kilogram of body weight were represented by 12 c.cm. and 5 c.cm. respectively, no ill effect followed injection. The amount injected

TABLE I.—*Intravenous Injection of Laked Red Blood Cells, Dissolved Haemoglobin, and Red Cell Stromata. First Series.*

No. of Experiment.	Weight of Rabbit in Grams.	Haemoglobin injected per Kilogram of Body Weight (in Terms of Red Cells).	Stromata injected per Kilogram of Body Weight (in Terms of Red Cells).	Result.
1	370	20 c.cm.	20 c.cm.	Died six minutes after injection.
2	746	15 "	15 "	Died three and a half hours after injection.
3	373	15 "	15 "	Convulsive attack one and a half hours after injection, followed by recovery.
4	485	12 "	12 "	No marked ill effect followed injection.
5	340	4 "	4 "	Died at end of injection.
6	240	30 c.cm.	Removed by centrifugalization	Not markedly affected by injection.
7	400	30 "	"	Slight lassitude after injection.
8	342	24 "	"	No marked ill effect followed injection.
9	455	20 "	"	Collapse after injection followed by recovery and subsequent relapse. Killed four hours after injection.
10	260	20 "	"	Died at end of injection.
11	400	12.5 "	"	No marked ill effect after injection.
12	300	12.5 "	"	No marked ill effect after injection.
13	260	9.0 "	"	Collapse after injection followed by recovery.
14	370	7 c.cm.	14 c.cm.	Died one minute after injection.
15	556	5 "	10 "	Died one and three-quarter hours after injection.
16	612	5 "	10 "	Died five and a half hours after injection.
17	712	5 "	10 "	Died two and a half hours after injection.
18	330	3.5 "	7 "	Died three hours after injection.
19	520	2.5 "	5 "	Paresis of hind legs after injection followed by recovery.
20	550	2.5 "	5 "	Died one and three-quarter hours after injection.
21	580	2.5 "	5 "	No marked ill effect followed injection.

NOTE.—In experiments 1 to 5 the material injected was laked blood cells; in experiments 6 to 13 haemoglobin solution; and in experiments 14 to 21 red cell stromata (sediment after centrifugalization).

in the latter experiment is in the neighbourhood of the lethal dose, but in the former experiment there is encountered an individual variation in susceptibility to injection which is from time to time met with in rabbits, and is further illustrated in the second group in the same table (Experiments 6 to 13), and also in Table II.

When the solution of haemoglobin free from stromata, prepared as described above, was injected in amount represented by 30 c.cm. to 9 c.cm. of red cells per kilogram of body weight, it was observed that in most experiments no obvious ill effect followed injection. Two animals, however, became collapsed after injection (20 c.cm. and 9 c.cm. respectively per kilogram of body weight), and one died at the end of injection (20 c.cm. per kilogram of body weight). These experiments, which indicate the different degrees of susceptibility exhibited by individual rabbits, illustrate, when taken in conjunction with the preceding and succeeding series, the relatively slight ill effect ordinarily following injection of even large quantities of haemoglobin compared with the lethal character of small amounts of stromata. The experiments further suggest that when symptoms make their appearance after injection of large quantities of solution of haemoglobin, freed as far as possible from stromata by long continued centrifugalization, the ultimate source of the symptoms is perhaps to be found not in the haemoglobin, but in some stromata still remaining in the fluid injected.

A second series of experiments of the same type are recorded in Table II. It will be seen that although some variations are met with in individual experiments (for example, Experiments 3 and 11), nevertheless, on the

TABLE II.—*Intravenous Injection of Laked Red Blood Cells, Dissolved Haemoglobin, and Red Cell Stromata. Second Series.*

No. of Experiment.	Weight of Rabbit in Grams.	Haemoglobin injected per Kilo of Body Weight (in Terms of Red Cells).	Stromata injected per Kilo of Body Weight (in Terms of Red Cells).	Result.
1	228	12 c.cm.	12 c.cm.	Died at end of injection.
2	228	12 "	12 "	Died at end of injection.
3	228	8 "	8 "	No marked ill effect followed injection.
4	256	8 "	8 "	Died three and a half hours after injection.
5	570	8 "	8 "	Died at end of injection.
6	496	7 "	7 "	Died one minute after injection.
7	171	5 "	5 "	No marked ill effect followed injection.
8	300	5 "	5 "	Died eight hours after injection.
9	214	2.5 "	2.5 "	No marked ill effect followed injection.
10	385	27.5 c.cm.	Removed by centrifugalization	No marked ill effect followed injection.
11	385	20 "	"	Collapse at end of injection, followed by complete recovery.
12	313	15 "	"	No marked ill effect followed injection.
13	342	15 "	"	No marked ill effect followed injection.
14	342	10 "	"	No marked ill effect followed injection.
15	385	8 "	"	No marked ill effect followed injection.
16	484	7.5 c.cm.	15 c.cm.	Died thirty-eight minutes after injection.
17	256	7.5 "	15 "	Died one hour and fifty minutes after injection.
18	400	6.5 "	13 "	Died at end of injection.
19	400	5.0 "	10 "	Died twenty minutes after injection.
20	356	5.0 "	10 "	Died eleven hours after injection.
21	313	2.5 "	5 "	No marked ill effect followed injection.
22	400	2.5 "	5 "	No marked ill effect followed injection.
23	228	1.25 "	2.5 "	No marked ill effect followed injection.

NOTE.—In experiments 1 to 9 the material injected was laked red blood cells; in experiments 10 to 15 haemoglobin solution; and in experiments 16 to 23 red cell stromata (sediment after centrifugalization).

whole, fairly concordant results are obtained. The very slight effect produced by the injection of large amounts of haemoglobin and the extremely lethal character of injection of stromata in relatively small amounts are much more effectively exhibited in this table than in Table I.

The character of the symptoms was the same whatever the material injected. The most acute symptoms, appearing towards the end of or shortly after injection, were convulsions of an opisthotonic type, rapidly terminating in death; sometimes collapse preceded or replaced convulsions, and usually the heart continued to beat for about a minute after respiration had ceased. In less severe cases difficulty of respiration was observed, attended with feebleness; if recovery did not occur death was preceded by convulsions. In other cases increased respiratory movements and refusal of food were the only definite indications of illness. After death the lungs generally collapsed normally on opening the chest, though occasionally collapse took place somewhat slowly.

As in the experiments already published, delayed coagulation of the blood was observed after injection. A number of additional observations, which have been collected from a large series of experiments, are grouped together in Table III. They were made with the aid of Dale and Laidlaw's coagulometer.² It will be seen that considerable variation is observable in respect of the time of onset of coagulation at 37° C. In general the onset is delayed, and occurs at the end of one to nine minutes, but

in exceptional cases the coagulability of the blood is increased, clotting taking place at the end of twenty to thirty seconds (the normal time of coagulation of rabbit's blood *in vitro* at 37° C. is generally about sixty seconds, rarely exceeding seventy-five seconds, and being occasionally only about thirty seconds in very young animals). In one experiment the blood remained liquid, and in another clot was found in the heart immediately after death. No clear relation is, however, observed between (1) the alteration of coagulability and (2) the amount of material injected or the length of time clapsing between injection and examination at time of death.

The alteration of coagulability of the blood after injection, taken in conjunction with the circumstance that red cell stromata in presence of calcium chloride act as a coagulant *in vitro* of blood plasma or of a solution of fibrinogen, suggests that the symptoms attending injection may be due to intravascular separation of fibrin.³ To test this point microscopical examination of the viscera was made in all cases of death following injection. Masses of fibrin were found in the blood vessels of the lungs in some experiments, while in others the result of very careful search was negative; thus out of nineteen experiments, in which laked red cells or red cell stromata were injected with fatal result, fibrin masses were found in the blood vessels of the lungs in nine cases. The appearance presented is illustrated in Fig. 1, in which a scanty collection of fibrin fibrils is shown within the lumen of a venule of the lung. In Fig. 2 fibrin has separated out in the form of beaded threads; this form is unusual and is generally associated with the former. Not infrequently collections of leucocytes, usually polymorphonuclear, were seen in the

TABLE III.—*Effect of Injection upon Coagulation of the Blood.*

Material Injected.	Haemoglobin injected per Kilo of Body Weight (in Terms of Red Cells).	Stromata injected per Kilo of Body Weight (in Terms of Red Cells).	Time of Death.	Coagulation Time (37° C.).
Laked red cells	25 c.cm.	25 c.cm.	At end of injection	20 sec.
"	15 "	15 "	8 min. after injection	6 min.
"	8 "	8 "	1½ hrs. after injection	2 min.
"	8 "	8 "	3½ hrs. after injection	Clot in heart at time of death.
"	8 "	8 "	At end of injection	5 min.
"	7 "	7 "	1 min. after injection	5 min.
"	5 "	5 "	4 hrs. after injection	1½ min.
"	5 "	5 "	3½ hrs. after injection	20 sec.
"	5 "	5 "	8 hrs. after injection	Remained liquid.
Haemoglobin solution	20 c.cm.	—	4 hrs. after injection	1½ min.
"	15 "	—	At end of injection	4 min.
Red cell stromata	7.5 c.cm.	15 c.cm.	38 min. after injection	Blood in heart clotted 8 min. after death.
"	7.5 "	15 "	2 hrs. after injection	30 sec.
"	7.0 "	14 "	1 min. after injection	1 min.
"	6.5 "	13 "	At end of injection	9 min.
"	5.0 "	10 "	20 min. after injection	1 min. 10 sec.
"	5.0 "	10 "	1½ hrs. after injection	4 min.
"	5.0 "	10 "	5½ hrs. after injection	3½ min.
"	3.5 "	7 "	3 hrs. after injection	1 min. 10 sec.
"	2.0 "	6 "	1½ min. after injection	Blood collected from heart formed a firm clot at room temperature.
"	1.4 "	4 "	2½ hrs. after injection	Blood collected from heart formed a soft clot at room temperature.
"	1.2 "	3.5 "	3 min. after injection	1½ min.
"	1.0 "	3.0 "	3 min. after injection	1 min.

blood vessels of the lungs (Fig. 3); these possibly represented the sites of intravascular collections of fibrin which have disappeared as the result of phagocytosis. The masses of fibrin when present were, however, in such

Fig. 1.—Section of small vein of lung of Rabbit 20, Table II. The lumen of the vessel contains red cells and red cell stromata (the latter more numerous than represented in the sketch), together with a few leucocytes and fibrils of fibrin, the latter forming a very irregular network. Stained with iron-alum haematoxylin. $\times 500$.

exceedingly sparse numbers that it seemed very improbable that they can be the cause of the symptoms observed after injection. This conclusion is borne out by a circumstance which has not yet been referred to, namely, that when blood obtained from the heart immediately after death clots, the clot, which, as already mentioned, is generally formed slowly, is nevertheless not, as a rule,

Fig. 2.—Section of small vein from lung of Rabbit 15, Table I. The lumen of the vessel is occupied by granular material (not shown in the sketch), representing precipitated protein, retracted from the vessel wall, in which are seen several moniliform fibrin threads of varying length and also a few isolated granules. A small number of leucocytes are visible below, together with three red blood cells, two of which are seen in optical section, the third being decolourized. Stained with iron-alum haematoxylin. $\times 530$.

lacking in firmness, so that no obvious defect of fibrinogen in the blood can be recognized. This is not, however, invariably the case, for, as already noted, the blood occasionally formed a very soft clot or failed to coagulate,

remaining liquid (Table III). Even in the latter cases, however, fibrin masses were very sparse or could not be found in the pulmonary blood vessels, and appeared to be absent in the blood vessels of the other viscera. Further

Fig. 3.—Section of small vein from lung of Rabbit 15, Table I. The lumen of the vessel is occupied by granular material (not shown in the sketch), representing precipitated protein, in places retracted from the vessel wall, together with numerous polynuclear leucocytes, situated towards the right. The leucocytes are more numerous than would appear from the sketch. No red cells or red cell stromata are present in the vessel. Stained with iron-alum haematoxylin. $\times 450$.

light may be expected to be afforded by a determination of the amount of fibrinogen in the blood plasma in those rare cases in which blood collected from the heart immediately after death remains liquid. This occurred in Experiment 9, Table III, but unfortunately an opportunity of estimating the content of fibrinogen was not afforded in this experiment. As already mentioned, this result of injection is an occasional and quite exceptional occurrence, and cannot as yet be produced intentionally.

It thus does not appear possible to attribute the general symptoms attending injection of red cell stromata to a purely mechanical effect of injection brought about by intravascular production of fibrin, for the amounts so produced are very small, and fibrin cannot be detected in every case of death following injection. Nevertheless it must be admitted that the irregular character of the result of injection of laked red cells or red cell stromata strongly suggests a mechanical lesion, for it is to be observed that neither the time of onset nor the severity of symptoms corresponds closely to the amount of stromata injected, and this would appear to be most simply explained on the assumption that a blocking of blood vessels occurs, the incidence of which would be expected to vary in animals of different ages and unequal powers of accommodation to obstruction of the circulation of the blood.

Two facts, not hitherto referred to, suggest the possibility of occurrence of mechanical obstruction of the circulation after injection of liquids containing stromata; one is the circumstance that when such liquids are examined microscopically it is found that the stromata are aggregated into masses of varying size, the exact limits of which are difficult to define; the other is that when a drop of the suspension of red cell stromata employed for injection is allowed to fall into 0.85 per cent. sodium chloride solution or into rabbit's serum, the drop breaks up into flocculent masses of varying size, forming a coarse precipitate. The latter appearance suggests that it would be impossible to inject such a liquid into a vein without the occurrence of blocking of the blood vessels of the lungs, and at first sight appears to furnish a complete explanation of the symptoms following injection. On searching for evidence of such obstruction in sections of the lungs we have not, however, been able to obtain, by any of the staining methods adopted, satisfactory evidence of blocking of pulmonary vessels,* though careful

* The presence of collections of leucocytes in the pulmonary vessels already alluded to is consistent with the hypothesis of an antecedent plug of stromata in these vessels.

observation has been made in all experiments in which injection has been followed by death.

The alternative hypothesis is that the symptoms are toxic in character, due either to substances dissolved out of the stromata before injection or dissolving in part, at any rate, in the blood plasma of the animal after injection. This hypothesis does not appear to us to account so satisfactorily for the variability observed in the time of onset and severity of the symptoms following injection of stromata holding liquids, as does the mechanical explanation above suggested.

Neither of the two hypotheses appears to furnish a satisfactory explanation of the occasional appearance of acute symptoms following upon the injection of solution of haemoglobin free from stromata.

Before concluding this article reference may be made to a series of seven experiments, in which from 7 c.cm. to 2.5 c.cm. per kilogram of body weight of laked rabbit's red cells were injected intravenously into rabbits in which both vagi had been divided before injection. The object of the latter procedure was to avoid the production of reflex arrest of respiration should the stromata injected be capable of exerting any marked local action upon the pulmonary tissue. These experiments, however, afforded no evidence of such action; at the time of death the heart was observed to continue beating after the cessation of respiration, exactly as occurred in those experiments in which division of the vagi had not been practised.

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A CASE OF EOSINOPHILE ASCITES: WITH REMARKS.

By H. D. ROLLESTON, M.D., F.R.C.P.,

SENIOR PHYSICIAN, ST. GEORGE'S HOSPITAL; PHYSICIAN, VICTORIA
HOSPITAL FOR CHILDREN, CHELSEA.

EOSINOPHILE ascites has, so far as I can find out, hardly ever been recorded. This rather contrasts with the attention which has been devoted to the occurrence of eosinophile cells in the exudate of experimental acute peritonitis in its earliest stages, especially by Durham, Opie, Dudgeon and Ross. But the exudate of acute peritonitis in human beings rarely contains as much as 1 per cent. of eosinophile cells. In this case of eosinophile ascites acute peritonitis brought the eosinophile count down from 22 to 0.5 per cent. The explanation of a hæmic eosinophilia of 12 per cent. is not obvious in the absence of animal parasites. Possibly it may be connected with the presence of carcinoma, for Weinberg and U. Mello¹ found 3.33 per cent. of eosinophiles in carcinoma of the colon, and 6.6 in carcinoma of the liver.

History and Course.

A male, aged 52, at the end of September, 1913, had colic and vomiting two days after eating some fish, followed gradually by jaundice and putty-like stools, and about the middle of October by transient pruritus. He was admitted to St. George's Hospital on November 4th with considerable jaundice, some rather vague resistance in the right loin, an enlarged and tender liver, and tenderness near the umbilicus. The gall bladder and the spleen could not be felt. For a time there was some apparent improvement, but on November 21st ascites was obvious, and on December 3rd 7 pints of fluid were removed; Dr. E. L. Hunt found that this contained red blood corpuscles, lymphocytes 46 per cent., endothelial cells 29, eosinophiles 22, polymorphonuclears 2, mast cells 1. The Wassermann reaction in the ascitic fluid and in the blood was negative (Mr. H. W. Bayly). A differential count of the hæmic leucocytes (total count 17,040) showed: Polymorphonuclears, 71 per cent.; lymphocytes, 16.5; mononuclears, 0.5; eosinophiles, 12; mast cells, 0. Examination of the stools did not reveal the presence of any ova of worms. The urine constantly contained bile pigment, but not bile salts, and there was no sugar; in the last week of life there was a trace of albumin on two occasions. Early in December the base of the right lung was found to be dull on percussion, breath sounds to be absent, and skiagraphy showed opacity. On December 12th the patient had severe abdominal pain, and the pulse rose to over 100. The pain gradually increased and necessitated the use of morphine. On December 14th the patient became comatose and died on December 15th at 2 a.m. His temperature, which had always been about normal, rose to 99.6° F. on December 14th.

Necropsy.

The necropsy was performed by Dr. R. S. Trevor. The skin was universally jaundiced. There was acute peritonitis with a copious effusion which, Dr. E. L. Hunt reported, contained polymorphonuclear cells 79 per cent., lymphocytes and endothelial cells 21 per cent.; a count of 400 white cells showing two eosinophiles. There were numerous minute secondary growths on the peritoneum, especially on the diaphragm, which was much thickened. There was a primary "colloid" carcinoma in the ascending colon just above the ileo-caecal valve, which was not narrowed. The caecum was high up in the abdomen on the right side. The liver, 5½ lb., contained numerous secondary growths, some of which were "colloid." There was much matting in the portal fissure, but the portal vein was free from thrombosis. The walls of the gall bladder were white, thickened, and microscopically infiltrated with carcinoma. There were no biliary calculi. The kidneys, 8 oz. each, were bile-stained, but otherwise normal. The spleen, 8 oz., was soft. There was a bilateral bile-stained pleural effusion; there was recent pleurisy on the right side; the fluid was not examined. There was extensive collapse of the lower lobes of both lungs. The apex of the right lung was free from tuberculosis, but near the apex of the lower lobe there was a small calcareous mass. There was no evidence of tuberculosis in the left lung, or of metastases in the lungs or pleurae. The heart, 11 oz., was soft, and the endocardium bile-stained. On the anterior cusp of the mitral valve there was a small serous cyst, the size of a sixpence; as this burst on examining the heart its contents could not be investigated.

Microscopically the cyst was shown to be due to oedematous separation between the superficial and the deep layers of the valve segment, and not to present any evidence of a hydatid cyst. There was no trace of hydatid disease elsewhere in the body. Microscopically the growth in the colon was a columnar celled carcinoma showing extensive myxomatous degeneration. Sections of the primary growth and of the peritoneal metastases did not show eosinophilia.

In this case the discovery of ascitic eosinophilia suggested hydatid infection of the peritoneum, on the ground that hæmic eosinophilia may accompany hydatid disease. But I was somewhat surprised at the apparent absence of any published evidence that rupture of a hydatid cyst into the peritoneum caused a local eosinophilia in that serous sac. I therefore wrote for information to Professor F. Dévé, of Rouen, the authority on hydatid disease, who very kindly replied:

Peritoneal eosinophilia does, indeed, sometimes occur as the result of intraperitoneal rupture of a hydatid cyst—with or without choleperitoneum—but, so far as I know, one case only has been recorded—namely, in Brouqueyre's thesis, "De l'éosinophilie générale et locale dans les kystes hydatiques" (*Thèse de Bordeaux*, 1905, p. 70). This observation was also given *in extenso* (without any fresh cytological details) in Tremblin's thesis, "De l'ouverture spontanée des kystes hydatiques du foie dans le péritoine" (*Thèse de Paris*, 1906, No. 283, p. 45, Obs. I). This is the extract from Brouqueyre's thesis: "M. Leveboullet has told me of an extremely interesting case which he watched when under the care of Professor Gilbert. A hydatid cyst of the liver ruptured, but none of its contents passed into the peritoneum. The usual phenomena due to a secondary ascites of no great size led to paracentesis. The fluid withdrawn was deeply bile-stained, and, on centrifuging, gave a deposit containing a considerable quantity of eosinophile cells, at least 50 per cent."

I have seen a case of the same kind, which, though I intend to publish it eventually, you are at liberty to quote. In February, 1913, I diagnosed a hydatid of the liver in a man of 23 years of age, and advised operation without delay. Four days later it ruptured spontaneously when he was sitting quietly at his desk doing accounts. Laparotomy twenty-seven hours after the rupture revealed rupture of a hydatid cyst on the anterior surface of the left lobe of the liver and 3 or 4 litres of turbid fibrinous fluid the colour of orangeade and containing scolices. Bacteriological cultures proved that the fluid was sterile, and chemical analysis that it contained urea (0.45 per cent.), fibrin, cholesterin, bile pigments, and traces of glucose, but no bile salts. Cytologically I found polymorphonuclear neutrophiles 85 per cent., eosinophiles 8 per cent., small and medium-sized mononuclears 0.5, large mononuclears (macrophages) 0.5, and red blood cells 5.3 per cent. At the time of the operation the blood contained 2.7 per cent. of eosinophiles (before the rupture it was only 0.9 per cent.).

Dr. d'Este Emery has told me that on one occasion he found 10 per cent. of eosinophiles in the ascitic fluid of a man who afterwards died of abdominal malignant disease. I have not obtained any other information from published