oedematous, and the abdominal wall greatly distended with oedema, although there did not seem to be much free fluid in the peritoneal cavity. During this time she was passing an average of 40 to 50 oz. of urine in the twenty-four hours, and was very restless and sleepless. Her blood at this period, as seen in the above table, presented a very interesting feature, namely, a blood crisis—the blood films contained a great number of nucleated red corpuscles. Many of their nuclei were bilobed or trilobed as if dividing. It will be remembered that a somewhat similar condition of the blood was found in Case III where she was most oedematous. The presence of a blood crisis is said to be a favourable sign in prognosis. From this period until the present date she made a gradual improvement, and her oedema almost entirely disappeared. From April 8th to the 19th, while her oedema was passing off, she passed from 130 to 150 oz. of urine in the twenty-four hours.

HAEMORRHAGE.

VII.—The next case is one of haemorrhage that presented the clinical appearance of pernicious anaemia, he had, however, bleeding piles and made a remarkable recovery. On admission to hospital his tissues appeared very flabby, although there was but slight evident oedema. He entirely lost this flabby appearance as he improved.

R. B. Bleeding Haemorrhoids.

	R. C.	W. C.	Hb.	Lymph	Pmn.	N. B.	M. B.	Wt.
Sept. 25	1,680,000	5,000	20	23	76.5	0	0	st. lb. 11 10½
Oct. 27	3,100,000	4,100	55	40.6	58.8	0	0	11 4
Nov. 8	4,350,000	5,900	74	34.8	63.6	0	0	11 6

For the first month in hospital this patient lost steadily in weight, falling from 11 st. 10½ lb. to 11 st. 4 lb. At the same time the haemoglobin rose from 20 per cent. to 55 per cent. From the 27th he began to gain slowly in weight while his haemoglobin continued to rise.

This case was undoubtedly one of severe secondary anaemia due to haemorrhage. It has been often contended that severe long continued haemorrhage may produce a blood picture in all respects similar to pernicious anaemia. In this case and in all the others I have examined it was quite possible from the blood to distinguish the two conditions. It is unfortunate that those authorities who make this contention give no accurate details of the blood examination of such secondary anaemias which present all the features of pernicious anaemia.

CONCLUSIONS.

In conclusion, I may sum up the points of interest suggested by these observations:

1. The absence of loss of weight in anaemic conditions, and the fact that the patient seldom seems emaciated is mainly due to the fact that there is abnormal accumulation of fluid in the blood and tissues. If this excess of fluid were deducted it would probably be found that in these, as in other chronic illnesses, there is a progressive loss of weight in proportion to the severity and duration of the disease.
2. In the cure of such anaemic conditions, especially chlorosis, the first stage seems to be the getting rid from the blood and tissues of this excess of fluid.
3. A gain of weight in a case of pernicious anaemia under treatment, and without any improvement in the haemoglobin, is to be regarded as an unfavourable sign, indicating dilution of the blood and consequent escape of serum into the tissues. It may, however, be a critical phase of the disease, and indicate the first step towards concentration of the blood. Immediately after this sudden increase in oedema there is either a marked improvement or the patient dies.
4. The oedema of anaemic conditions seems to result from a hydraemic plethora of the blood, and is somewhat different in origin and nature from the oedema usually found in Bright's disease.
5. Careful observations in the manner indicated of anaemic conditions, namely, a record of the weight and haemoglobin value of the blood may furnish very interesting results. The points to direct attention to are the occurrence of haemor-

rhages (epistaxes, etc.), oedema, diarrhoea, and profuse sweating. My observations convince me that these symptoms are often the result of dilution of the blood, and may be merely Nature's method of counteracting the excessive and abnormal volume of the blood.

REFERENCES.

- ¹ *Trans. Path. Soc. Lond.*, vol. 11, part 3, 1900. ² See former paper in *BRITISH MEDICAL JOURNAL*, June 15th, 1901.

REPORT LXX.

ABSTRACT OF A REPORT ON THE FORMATION OF LYMPH BY THE LIVER.*

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In 1899 Asher found that the intravenous injection of ox bile, blood serum, and other substances produced an increased flow of lymph from the thoracic duct; he considered that these substances increased the metabolism of the liver, that the lymph came from the liver, and that it was formed as a result of the increased activity of the liver cells.

In the present research pure taurocholate of soda, pure haemoglobin, and certain ammonium salts have been injected intravenously, and the effects of such injections on the lymph flow have been studied.

RESULTS.

I.—*Taurocholate of Soda.*

Slow injections of small amounts invariably lead to an increased flow of lymph. The flow is doubled or trebled, begins ten to twenty minutes after the injection is begun, reaches its maximum in twenty to thirty minutes, and lasts about two hours. The extra lymph is derived entirely from the liver, since after ligature of the portal lymphatics, injections of bile salt cause no increased flow of lymph.

The lymph contains a slightly-increased percentage of solids, and usually also a small amount of free haemoglobin.

Observations on the blood pressures show no change in the arterial or vena cava pressure; there is a slight rise of portal pressure, but it falls within the limits of a physiological rise accompanying the increased flow of bile also produced by injecting bile salt.

When the bile and lymph are collected simultaneously, the variations in the rate of flow are very similar in the two cases; the most important difference is that the increased bile flow sets in earlier than the increased flow of lymph.

II.—*Haemoglobin.*

The injection of haemoglobin in dilute solution leads to results very similar to those obtained with bile salt, except that the bile becomes more concentrated, though its amount is not as a rule increased.

III.—*Ammonium Salts.*

The salts used were the tartrate, carbonate, and chloride. Intravenous injection sometimes leads to an increased lymph flow, but more often a negative result is obtained. The experimental conditions were so unsatisfactory, and toxic symptoms so prominent in the case of ammonium salts that no conclusion could be drawn from them.

COMMENTARY.

The injection of bile salts (and haemoglobin) does not cause blood-pressure changes similar to those produced by bodies either of the peptone class or of the sugar class; in fact, bile salts differ in very many respects from peptone in their mode of action. Consequently they do not belong to either of Heidenhain's classes of lymphagogues.

On the other hand, bile salts do increase the metabolism of the liver and also the formation of lymph; this increased metabolism does not merely accompany, but is the actual cause of the increased flow of lymph. In this respect Asher's view is fully confirmed, and these bodies constitute a "third" class of lymphagogues.

It is further supposed that this suddenly-increased metabolism leads to the setting free in the liver cells of crystalloid metabolites. These enter the surrounding lymph spaces

* The full text is published in the *Journal of Physiology*.

—probably by diffusion—raise the osmotic pressure of the lymph, and attract water from the blood. In this way the increased lymph flow is accounted for. It is, of course, possible that bile salt (and haemoglobin) stimulate the hepatic capillaries to secrete lymph, after the manner suggested by Heidenhain. But the experiments, though not actually opposed to such a view, do not support it.

REMARKS ON A CASE OF HEREDITARY LOCALIZED OEDEMA PROVING FATAL BY LARYNGEAL OBSTRUCTION.*

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DURING the last sixteen years I have had under my observation from time to time some remarkable cases of localized oedema, and I will give the history of one of the cases which was the most striking I have ever seen, and will append some general considerations on the subject.

History of the Case.

The patient was a young woman, aged 18 years, when she first came under my observation. She was admitted into the infirmary under the care of Dr. Eddison on June 21st, 1886, with great oedema of the right arm and hand, and was suffering from dyspnoea. Her voice was husky, and she had some pain on swallowing. Her tongue was coated, but her general health was good and there was no albuminuria, and it was the difficulty in breathing which determined her admission. On laryngoscopic examination there was found very great oedema of the mucous membrane covering the epiglottis and aryteno-epiglottidean folds, so great as almost entirely to conceal the vocal cords. The question of tracheotomy was considered, as was also that of scarification of the swollen mucosa, but it was deemed safe to wait. By the evening the oedema had entirely disappeared from the larynx, the swelling of the arm rapidly subsided, and she was discharged well at the end of five days.

From the patient and her mother the following interesting history was subsequently elicited. Ever since infancy she had been subject to localized swellings on various parts of the surface of the body. Thus shortly after birth one arm swelled to such an extent that the clothing had to be cut to release it. Very often she used to notice a small rounded swelling like a marble under the skin which would enlarge and become diffuse, so that the skin, say on the back of the hand, would swell enormously, and swellings more or less diffused or localized used to develop with great rapidity on her hands, feet, shoulders, chest, or around the vulva. Sometimes these were attended with slight redness and tickling, and when the vulva was affected, micturition was painful but not difficult. These swellings came on with great frequency, but no periodicity had been noticed. Menstruation had been established at the age of 15, and had always been normal, and apparently without influence on the incidence of the swellings. She was unmarried, but had had one child.

A careful examination of the patient failed to reveal any signs of disease or constitutional disturbance of any kind, except that for many years she had been subject to what she termed bilious attacks.

Her father from his earliest infancy had presented swellings of the same kind. On three occasions these seem to have affected the throat. The first attack of this kind occurred at the age of 21, the second a few years subsequently, and the third attack carried him off at the age of 29. He had come home from his work saying his throat was getting swollen, and his medical man who was sent for went to get something for his relief, but during his absence he suddenly got blue in the face and died asphyxiated.

From 1886 the patient was frequently seen by me at intervals until her death within the last few weeks, and I had frequent opportunities of seeing the remarkable swellings. They were, as she described, diffuse or circumscribed. Usually pale, but sometimes slightly pink, they did not pit

on pressure, and I never found that they became vesicular or bullous.

On two occasions the symptoms were rather alarming, and deserve special notice. In July, 1890, she struck her head against the lock of a door, which made her feel sick and caused a small lump on the forehead. Next day the swelling had passed down, affecting the eyelids and the face, and the following morning, when the swelling of the face was beginning to subside, her throat became slightly affected, and felt, she said, as if something were sticking in it. When I saw her, her face was much swollen, the swelling stopped at the margin of the hairy scalp and at the ears, and did not extend on to the neck. In a short time her appearance was again perfectly normal.

In October, 1891, she complained of slight toothache one day, with swelling of the same side of the face. During the night, and especially during the following day, the swelling rapidly increased, and then her voice became affected. At 11 o'clock at night I found her sitting up, in which position the dyspnoea was more tolerable, with her face swollen out of all possible recognition, and presenting the general aspect of a patient with acute renal dropsy of an extreme character. The oedema was practically confined to the face and under aspect of the chin; it was firm, did not pit readily on pressure, and somewhat resembled that of myxoedema, though much more pronounced than anything I have ever seen in that affection. The tongue was swollen, as was also the mucous membrane of the mouth, and the lips were so swollen that the mouth naturally remained slightly open, and the continual escape of the buccal secretion led to a constant necessity for mopping the mouth. The larynx was readily examined, but all that could be seen were three rounded masses, pale, jelly-like, and tense, representing the swollen mucous membrane covering the epiglottis and the two arytenoid cartilages, while the false and true cords were completely concealed. The voice was not much affected. Next day she was much better, the voice was clear and the swelling of the face was manifestly subsiding. The following morning she was perfectly well, nearly all swelling had gone, and the larynx was absolutely normal. A slight feeling of sickness during the night was the only manifestation she had had of general disturbance. The bowels were normal, and there was no alteration in the amount of urine passed.

Some six or seven years ago the patient was married and had three children. Her pregnancies, as on the former occasion, had no influence on the occurrence of the swellings.

The patient was fully alive to the risks attendant on the attacks of swelling in the throat, and the final scene justified her apprehensions. On February 20th, 1902, at 10.30 a.m., she mentioned to her mother that her throat felt swollen, but that the swelling seemed to be passing backwards, and she thought she would soon be all right again. At noon her husband came home from his work, and found her sitting on the floor to all appearance comfortable and free from dyspnoea, and amusing her child, for whom she was just going to spin a humming top. Suddenly she dropped the top, started up, apparently tried to speak, clutched at her own neck, and then at the throat of her husband, which I take to have been a pantomimic action expressive of her distress; got blue in the face, and died almost at once. When I saw her, in about half an hour, the features were livid, the tongue was very slightly protruded between the teeth, and there was some fullness about the neck. Her mother, who had left the room, had again arrived on the scene just at the moment of death, and her first words to me were that her daughter had died in exactly the same way as her father before her.

Necropsy.

The necropsy I made was an incomplete one, but I removed the larynx by cutting widely beyond its limits, and with as little disturbance as possible. The mucous membrane was very oedematous, tense, and pale, and the sides of the laryngeal cavity came in contact with one another a short distance below its superior aperture, and remained so until just below the level of the true cords. A small amount of viscid mucus was found in the cleft between the two opposed mucous surfaces, which may have possibly assisted to a very slight degree in the occlusion, but the oedema was so great that it is not necessary to invoke any such considerations. I

* Read before the Leeds and West Riding Medico-Chirurgical Society.