end of the humerus and femur. It is present in the flat bones, especially in those of the vertebræ and skull, although even in the flat bones yellow marrow takes the place of much of the red late in life. The yellow marrow which fills the long bones of the adult must not be regarded as identical with the fat of connective tissue. It must be compared with the fatty masses which replace the thymus and lymphatic glands, for under certain circumstances the yellow again changes to red. It is also asserted that the administration of yellow marrow has had a beneficial effect in anæmia by promoting the production of red corpuscles, a result which cannot be attributed to simple fat. Marquis [21] has described the marrow in the long bones of the frog, which is red during the active period in spring, becomes mixed with yellow during the summer, but during the autumn and winter rest is entirely yellow. Yellow marrow may, therefore, be regarded as red marrow in a state of quiescence. In chronic wasting diseases such as pulmonary emphysema, tuberculosis, chronic renal nephritis, and also on account of starvation nearly all the fat may be absorbed, causing the yellow marrow to have a jelly-like appearance. The fatty marrow may, on the other hand, increase accompanying eccentric atrophy of the bone until the bone becomes a mere shell and it may even float in water. This occurs especially in cases of senile osteomalacia.

The myeloplaxes or giant cells of the marrow.—The multinucleated myeloplaxes are found in Howship's crypts bordering on the marrow. They show marked powers of movement on the warm stage and also englobe particles. If carmine be injected into the marrow cavity, it is found included in the giant cells near the site of the injection. If an excess of the carmine be injected, the giant cells containing these particles are easily seen in the blood, and carmine is also found in the lungs, liver, spleen, and kidneys, showing that there is an easy exit from bone (Arnold).[22] Multinuclear giant cells are carried into the blood and appear there under a number of circumstances. But their presence in the blood does not indicate any excessive production of them in the marrow.

Lymphoid marrow.—The yellow marrow may be replaced by marrow varying in colour from a pinkish grey up to a deep red. But the lymphoid marrow as met with in disease is by no means the same as the red marrow of the young healthy animal. There is in lymphoid marrow not only a marked increase of the small colourless marrow cells and of the nucleated red cells, but many of the latter are found free in the capillaries, and there is an increase of blood pigment without there being evidence that this pigment can be worked up again as hæmoglobin. The excess of pigment is the result of hæmorrhages due to thrombosis of the blood-vessels, and this thrombosis is a sign that a destruction of leucocytes is in progress. Lymphoid marrow is best marked in pernicious anæmia, in which disease the prevailing factor is the excessive destruction of blood in the spleen and liver. In a like manner chronic septic absorption, especially chronic suppuration of bone, tends to produce lymphoid marrow. In cancerous cachexia a similar condition of the marrow may be also found, but it is necessary to distinguish lymphoid marrow from a diffuse infiltration of the marrow by secondary cancerous growths, which is often difficult. It is difficult to draw a sharp distinction between the lymphoid marrow produced by lymphadenoma accompanied by leucocythæmia and anæmia and new growths of lymphadenoid structure originating in the marrow from the small marrow cells (myeloma or lympho-sarcoma), except that these growths are circumscribed, at any rate in the earlier stages.

The inflammatory diseases of the marrow, as compared with those of the spleen and lymphatic glands, are limited in variety, for the organisms of most infectious diseases are not arrested in the bone. Only a few kinds are so stopped—e.g., tubercle bacilli, the organisms of syphilis, and pyogenic cocci. They infect especially the ends of the long bones of young subjects in which red marrow is still present, and which are especially vascular in the neighbourhood of the epiphyses. These organisms arriving by the circulation settle down and grow, being especially favoured if a blood-clot due to some slight injury be present. In older patients the periosteum, as the most vascular part of bone in the adult, is more likely to be affected than the marrow.

[21] Marquis: Inaugural Dissertation, Dorpat; also Centralblatt für Pathologie, &c., 1893, Band iv., S. 459.
[22] Arnold: Virchow's Archiv, 1895, Band cxli., S. 411.

The Arris and Gale Lectures

ON

SOME POINTS IN THE PATHOLOGY OF HEART DISEASE.

Delivered at the Royal College of Surgeons of England on Feb. 22nd, 24th, and 26th, 1897,

BY ERNEST H. STARLING, M.D., M.R.C.P. LOND.,

JOINT LECTURER ON PHYSIOLOGY AT GUY'S HOSPITAL.

LECTURE II.[1]

Delivered on Feb. 24th.

THE EFFECTS OF HEART FAILURE ON THE CIRCULATION.

MR. PRESIDENT AND GENTLEMEN,—In my previous lecture I discussed the conditions which may lead to a failure of the compensatory mechanisms of the heart. In this lecture I propose to deal with the effects of such failure on the circulation generally, that is to say, on the blood, and the pressure and velocity of this fluid in the different parts of the circulatory system.

I would, in the first place, remind you of some of the elementary facts in the physiology of the circulation. It must be remembered that the vascular system forms a closed circuit of elastic tubes. In one part of this circuit is included a pump—the heart, the contractions of which will, in consequence of the presence of valves, propel the contained fluid in one direction only. In this system there is a resistance to the flow of blood in consequence of the friction between the blood and the vessel wall in the small arteries and to a lesser extent in the capillaries. There is also a difference in the structure of the walls of the tube on the arterial and venous sides, so that a small rise of pressure in the veins will cause a considerable dilatation and increase in capacity of these vessels, whereas on the arterial side the injection of a small quantity of fluid will bring about a large rise of pressure. If the blood is at rest in the system, and assuming that all parts of the system lie in one plane, it is evident that the pressure in all parts of the system must be the same. This pressure was termed by Weber the mean systemic pressure (not to be confused with mean arterial pressure), and amounts in a large dog to about 10 mm. Hg. If the heart now commences to beat, fluid is pumped from the venous side into the arterial side, where it accumulates to a certain extent in consequence of the resistance in the smaller arteries and capillaries, so raising the pressure on the arterial side and lowering the pressure on the venous side. The fall, however, on the venous side is not directly proportional to the rise on the arterial side, since, as I have pointed out above, a large change in the capacity of the veins is only attended by a small variation of the pressure within them, whereas the blood removed from the veins cannot be pumped into the much less distensible arteries without causing in them a considerable rise of pressure. Somewhere in the circulation there must be a point where the pressure is neither raised nor lowered and where, therefore, the pressure is independent of the cardiac activity. At first sight it might be thought that this neutral point must lie in the situation of the peripheral resistance—viz., between the arterioles and capillaries. It must be remembered, however, that even when the blood is circulating there is not necessarily, although there may happen to be generally, a fall of pressure all the way round the system. What does fall from the heart through the arteries, capillaries, and veins back to the heart is the energy in the fluid at any given point. The total energy of a mass of blood at any point in the system is represented, not only by the hydrostatic pressure at that point, but also by the kinetic energy of the moving mass of fluid ($= \frac{1}{2} mv^2$). In the capillaries, in consequence of the great area of the bed, the velocity is very small and rapidly increases in the veins. Hence the proportion of the total energy represented by $\frac{1}{2} mv^2$ is relatively small in the capillaries and relatively large in the veins. The other

[1] Lecture I. appeared in THE LANCET of Feb. 27th, 1897.

factor—the pressure—must be therefore relatively large in the capillaries and small in the veins. It thus follows that the neutral point in the vascular system, where the mean systemic pressure is neither raised nor lowered by the inauguration of the circulation, lies considerably on the venous side of the capillaries—at any rate, in most parts of the body.

Now in such a system it is evident that a failure of the heart pump, however induced, whether by vagus-inhibition, by damaged valves, aortic stenosis, or cardiac fatigue, can only cause a return of the pressures in the various parts of the system to one level—the mean systemic pressure. We shall, therefore, have a rise of pressure on the venous side of the neutral point and a fall of pressure on the arterial side. By actual experiment we may ascertain the extent and direction of the change of pressure in the various parts of the circulation. To this end we may register the pressure in the arterial system, in the vena cava, and in the portal vein. We may deal in the first place with the case in which the vessels are removed from the interference of the vaso-motor centre by section of the cord or splanchnic nerves, so that we may study the pure effects of cessation of the circulation. In such an experiment the pressures before cardiac inhibition were: Femoral artery, 58 mm. Hg; portal vein, 73 mm. H_2O; and femoral vein, 62 mm. H_2O. On exciting the peripheral end of the right vagus the heart stopped, and ten seconds later the pressures were: Femoral artery, 20 mm. Hg; portal vein, 72 mm. H_2O; and femoral vein, 68 mm. H_2O. After three minutes the pressures were: Femoral artery, 6½ mm. Hg = 84 mm. H_2O; portal vein, 83 mm. H_2O; and femoral vein, 82 mm. H_2O; so that complete equilibrium was established between the pressures at all parts of the system. Here the total failure of the heart pump caused a considerable fall in arterial pressure, a considerable rise in the vena cava pressure, and practically no alteration in the portal vein. The last represents, therefore, a neutral point of the system; in all parts distal to the portal vein there must have been a fall of pressure. The only part of the body, therefore, where heart failure can by itself cause a rise of pressure is in the large veins of the trunk and possibly in the capillaries of the liver.

In the living body, however, we never get a reproduction of so uncomplicated conditions. In a closed circuit of tubes, as described by Weber, the mean systemic pressure can only be increased by injecting a further amount of fluid into the system or by diminishing the capacity of the system. In the body neither the amount of circulating fluid nor the calibre and capacity of the vessels forming the system are constant quantities, and it is possible that changes in the heart leading to its failure may have as an immediate consequence an alteration either of the capacity of the system or of the amount of circulating fluid. We might therefore have, as the remote effect of heart-failure, an increase in the pressures, not only near the heart, but also in the peripheral capillaries. Thus, if we repeat the experiment of vagus inhibition just described, but carry it out on a normal animal, we find that at first the results of stoppage of the heart are exactly similar to those already described. After the inhibition has lasted from twenty to forty seconds the anæmia of the brain causes a strong excitation of the vaso-motor centre. We therefore get extreme vascular constriction, with diminution in the capacity of the system, as is evidenced by a considerable rise in portal, vena cava, and arterial pressures. We may, in fact, in consequence of this constriction, obtain a doubling of the mean systemic pressure. Another point which we must bear in mind is that the quantity of circulating fluid is intimately dependent on the pressures at various parts of the vascular system. Almost every fall of arterial pressure gives rise to a diminished concentration of the blood due to the taking up of fluid by the blood-vessels from the tissues, whereas a rise of blood pressure augments the concentration by causing increased transudation. With these varying factors to take into account it is impossible to predict in our study chairs what will be the effect of failure of the heart pump on the pressure and distribution of the blood throughout the different parts of the body, but we must in each case have recourse to experiment.

In attempting to deal with this subject experimentally one is met at the outset by the difficulty of reproducing clinical phenomena in the laboratory. We have to choose a method of interfering with the action of the heart-pump which resembles in some degree the chronic processes of nature; artificial stenosis or valvular lesions are useless for this purpose, owing to the wonderful powers of compensation possessed by the normal heart, so that the margin between complete compensation and absolute failure becomes too narrow for experiment. The best method is perhaps that adopted by Cohnheim—viz., the interference with the action of the heart by the injection of oil into the pericardium. By this means we may effect an impairment of the heart's action of considerable duration without running the risk of stopping its action altogether. In the experiments of this nature carried out by Cohnheim, this observer measured the pressures in the carotid artery and in the jugular vein, and noticed that as the diastolic expansion of the ventricle began to be affected by the injection of oil into the pericardium there was a fall of arterial, accompanied by a rise of venous, pressure. The jugular vein is, however, so near the heart and so influenced by the diastolic suction of this organ that one cannot deduce a general rise of venous pressures from the occurrence of a rise in this vessel. I have, therefore, repeated Cohnheim's experiment with two modifications. In the first case I have measured simultaneously the pressures in the inferior vena cava, in the portal vein, and arterial system, while at the same time oil was being injected into the pericardium. In the second series of experiments I have observed the influence of the pericardial injection on the circulation in the limbs, using as my guide the volume of the limbs.

We will consider, in the first place, the effects of injecting oil into the pericardium on the pressures in the portal vein, vena cava, and in the arterial system. In these experiments the animal having been anæsthetised with a large dose of morphia and A.C.E. mixture, cannulæ were placed in the central end of the splenic vein, all other vessels of the spleen having been ligatured, in the central end of the iliac vein, and in the central end of the carotid or femoral artery. The venous cannulæ were connected with manometers filled with coloured $MgSO_4$ solution, so that the height of the fluid could be easily read off on the graduated scales behind the tubes. The arterial cannula was connected with an ordinary mercurial manometer, the excursions of the meniscus being registered on the blackened surface of a kymograph. At the end of the experiment, the abscissæ having been taken, the readings were plotted out as curves. After the attachment of the manometers the chest was opened in the middle line and a cannula tied into an opening in the pericardium for the injection of oil, which was allowed to run in from a graduated burette. In one experiment of this sort the pressures at the beginning of the experiment were: arterial, 90 mm. Hg; portal vein, 128 mm. $MgSO_4$; and vena cava, 36 mm. $MgSO_4$. 20 c.c. of oil were then allowed to flow into the pericardium. This had no influence on the pressures in any of the vessels. Injection of another 20 c.c. then caused a slight rise in the vena cava to 40 mm. $MgSO_4$, but no distinct change in the other vessels. Injection of another 20 c.c. caused a rise—the vena cava to 58 mm. $MgSO_4$, a slight but temporary fall in the arteries, and no change in the portal vein. Another 10 c.c. injected gave a large rise to 76 mm. in the vena cava, a slight rise to 134 in the portal vein, and practically no effect on the arterial pressure. I had now arrived, after the injection of 70 c.c. of oil, at the limits of compensation possessed by the heart and vascular system for the hindered diastolic expansion. Injection of another 20 c.c. of oil now caused a very marked change in all the pressures. The arterial pressure sank to 56 mm. Hg, while the portal pressure rose to 160 mm., $MgSO_4$, and the vena cava pressure to 124 mm. $MgSO_4$. The injection of a further 10 c.c. put a stop to the circulation altogether. The arterial pressure sank to 26 mm., and then as the heart stopped to 18 mm., while the portal vein and vena cava rose first to 180 and 170 mm., and then with the stoppage of the heart, to 215 mm., the pressures in both vessels being practically the same. The oil was now suddenly let out of the pericardium, and the heart, after a short pause, re-commenced beating. The first effects of the re-establishment of the circulation were striking. With the very first heart-beat the pressure in the vena cava sank with extreme rapidity to its normal pressure of 36 mm., and then rose slightly to about 40 mm. $MgSO_4$. The arterial pressure rose with equal rapidity to a point far above its previous height—viz., 146 mm. Hg., to fall rapidly to 84 and then rise gradually towards the end of the experiment. The pressure in the portal vein, instead of falling, like that in the vena cava, continued to rise rapidly

on the re-commencement of the heart-beats until it attained the enormous height of 322 mm. $MgSO_4$, whence it fell in the course of the next six minutes to 148 mm. $MgSO_4$—i.e., somewhat above its height at the beginning of the experiment.

How are we to explain these results? It is evident that we have here at least two factors at work—viz., first, the gradual impairment and ultimate failure of the heart pump; and, secondly, a diminution of the capacity of the vascular system and consequent rise of the mean systemic pressure, brought about by the activity of the vaso-motor centre. We have already seen that the effect of a pure stoppage of the heart is to cause a rise of pressure in the vena cava and a fall in the arteries, while the pressure in the portal vein, which represents the neutral point of the system, remains practically unchanged. In this case, however, the vena cava pressure not only rises to the height of the normal portal pressure, but attains nearly double this amount, this rise being accompanied by a parallel rise in the portal vein and a large fall in the arteries. Now we know that any anæmia of the vaso-motor centre, however caused, excites its activity, and therefore active constriction of the vessels all over the body, but especially in the splanchnic area. As soon, therefore, as the hindrance to the diastolic expansion becomes sufficient to diminish the normal outflow of blood from the heart in a given time there is a fall of arterial pressure, and the vaso-motor centre is excited and endeavours to restore the arterial pressure to its normal height by an active constriction of all the arteries. That at the moment in the experiment when the heart stops the bloodvessels are constricted is shown by the enormous rise in pressure which occurs directly the heart re-commences to beat. The heart, which is richly supplied with blood from the distended venæ cavæ, propels this into the arteries, fills them up, and then has to overcome the resistance of the constricted arterioles, so that the pressure rises high above its normal amount, to sink again as this raised pressure furnishes a sufficient supply of blood to the vaso-motor centres of the brain, which, it seems, from the work of Bayliss and Hill and other observers, do not participate in any general vascular contraction. The rise of pressure in the portal vein is analogous to that which occurs in the arteries, and is due to the constriction of the branches of the portal vein in the liver, so that the trunk of this vessel is supplied with blood more rapidly than can be passed through the liver into the vena cava. This experiment brings out two facts which are of importance for our knowledge of the conditions in heart disease. In the first place, any heart failure will tend to be accompanied by active vascular constriction and a rise of mean systemic pressure, so that the pressure at the neutral point of the system, as well as on the venous side of this point, must be raised considerably above normal. In all cases, therefore, of heart failure there will be a large rise of pressure in the capillaries of the liver. In the second place, the existence of this vascular constriction shows that the heart, although inadequate to perform its functions, cannot relieve itself by calling on the depressor mechanisms when by so doing it would diminish below what is necessary the blood-supply to the vaso-motor centre.

We have now the important question to decide as to how far back this rise of venous pressure goes. Is the diminution in the capacity of the vascular system consequent on the vaso-constriction ever so great as to raise the pressure in the small veins and capillaries in the peripheral parts of the body such as the limbs? To determine this question one must carry out an experiment similar to the above, but at the same time enclose one limb in a plethysmograph and record changes in its volume by means of a Marey's tambour or a piston-recorder connected with the plethysmograph. If an experiment be arranged in this way and oil injected into the pericardium, it is found that, while the arterial pressure remains constant, practically no change occurs in the volume of the limb, although there may be a rise of pressure both in the portal vein and vena cava. As soon, however, as the compensatory mechanisms become inadequate and the arterial pressure begins to sink, the volume of the limb also diminishes. Thus the volume of the limb, and presumably therefore the pressure in its smaller vessels, including its capillaries and veins, is directly proportional to the arterial pressure, and is not altered by considerable changes in the venous pressures consequent on heart failure. We may conclude, therefore, that, even with coincident vascular constriction, the effect of heart failure must be a fall and not a rise of blood pressure in the capillaries and smaller veins of the limbs. Since this conclusion is opposed to the ordinarily accepted view, which we owe largely to Cohnheim, that failure of compensation in heart disease leads to a general rise of venous and capillary pressures in all parts of the body, we must inquire whether there may not be other factors at work by which a rise of pressure might be produced. Especially we can elevate the mean systemic pressure, not only by vascular constriction, but also by increasing the amount of fluid contained in the system—i.e., by causing a plethora. Are there any conditions in heart failure which might lead to a plethora sufficient to raise the venous and capillary pressures in the limbs in the absence of any arterial rise?

I pointed out last year that a slight degree of plethora must be the invariable consequence of any heart failure, since a fall of capillary pressure increased the fluid taken up from the tissues and diminished the transudation into the tissues. We therefore get a fall of specific gravity of the blood whenever the arterial pressure is lowered by heart failure or by bleeding. It will be noticed, however, that such an increased absorption of fluid from the tissues must come to an end as soon as the cause of the absorption is removed—i.e., as soon as the capillary pressure is restored to its normal amount, so that a plethora brought about in this way could never be made responsible for a rise of capillary pressure in the limbs. Of course, even when the pressure in the limb capillaries has attained its ordinary amount there is one factor left tending to produce a hydræmia or hydræmic plethora — viz, the diminished urinary flow in consequence of lowered arterial pressure,— and last year I thought that possibly this factor would be sufficient to cause a condition of hydræmic plethora in heart disease and raised capillary pressure in the limbs. Whether this is or is not the case can only be decided by direct clinical observation. If a condition of hydræmic plethora be present in uncompensated heart lesions we should expect to find—(1) a diminution in specific gravity of the whole blood; and (2) a diminution in the amount of hæmoglobin and number of corpuscles present in the blood.

With regard to the first point, it has been shown fairly conclusively by Lloyd-Jones that in failure of compensation there is a marked diminution in the specific gravity of the whole blood. Now this diminution might be due to a hyperplasmia of the blood, possibly brought about by a condition of plethora or to a decrease in the solids of the plasma or corpuscles or of both—i.e., either to hydræmic plethora or to hydræmia. In order to decide which of these two conditions is responsible for the lowered specific gravity we must have recourse to the determination of the hæmoglobin and corpuscles in the blood of such persons. On referring to the various researches on the subject we find a considerable discrepancy. According to the majority of writers, the relative number of corpuscles in the blood of patients suffering from heart disease is increased, whereas according to others, such as Stintzing and Gumprecht, the exact opposite is the case. In face of this discrepancy Dr. Fawcett has undertaken a series of observations on the amount of hæmoglobin and the number of corpuscles in the blood of patients suffering from heart disease, whether accompanied or not by signs of so-called "backward pressure." Although these researches are not completed, he has kindly allowed me to quote some of his results in this lecture.

The first case investigated seemed at first to favour the hypothesis of the existence of hydræmic plethora.

CASE 1. *Mitral disease.*—This patient, aged thirty-three years, was admitted to hospital on Oct. 30th, 1896, with œdema and ascites of a month's duration. On Nov 3rd the hæmoglobin amounted to 48 per cent.; on Nov. 14th it was 50 per cent.; on Nov. 24th, 54 per cent.; on Nov. 30th, 65 per cent.; and on Jan. 10th, 1897, 85 per cent. Here there was a marked increase in the percentage amount of hæmoglobin during the patient's stay in the hospital. It must be remarked, however, that the œdema and ascites had disappeared entirely by Nov. 11th, when the hæmoglobin was only 50 per cent., and that the condition of the blood only commenced to improve markedly with the administration of iron, which began on Dec. 7th. In all the other cases investigated the amount of hæmoglobin, even when marked ascites was present, was either above normal or very little below.

CASE 2 *Mitral stenosis and regurgitation, with tricuspid*

regurgitation.—This was a patient aged twenty-eight years. On Nov. 11th the hæmoglobin amounted to 100 per cent. and there were œdema of the legs and ascites. On Dec. 12th the hæmoglobin was 97 per cent., and the ascites and œdema had disappeared.

CASE 3. *Mitral stenosis (ulcerative endocarditis)*.—This was a patient aged forty-one years. On Nov. 11th the hæmoglobin was 105 per cent., and there was much œdema. On Nov. 24th the hæmoglobin had risen to 110 per cent.; there was still much œdema, and the patient died on Nov. 27th.

CASE 4. *Bronchitis with heart failure*.—This was a patient aged twenty-three years. On Nov. 18th the hæmoglobin amounted to 97 per cent., and there were œdema and ascites. On Dec. 7th the hæmoglobin was 90 per cent., and the patient was in the same condition and died on Dec. 8th.

In nearly all cases it was found that the number of corpuscles was increased to a greater extent than the hæmoglobin, so that the corpuscles themselves contained less hæmoglobin than normal.

It will be seen that these results lend no support to the view that failure of compensation is attended by a condition of plethora. They tend rather to show that the amount of blood in the circulation is subnormal, unless we adopt the unlikely supposition that the defective aeration of the blood in this disease acts like mountain air, in stimulating the production of red corpuscles and hæmoglobin. We must, therefore, conclude that there is no condition of the plethora, hydræmic or otherwise, in heart disease, and that the lowering of the specific gravity of the whole blood is due to a diminution of solids of the plasma, and possibly also of the corpuscles—i.e., there is hydræmia, but no plethora. Since the volume of the fluid contained within the vascular system is not increased, the only factor which can contribute to the raising of the mean systemic pressure, and a consequent production of a rise of pressure in the peripheral veins and capillaries by failure of the heart-pump, is the active vascular constriction caused by the anæmia of the vaso-motor centre. We have already studied the limits of a rise of systemic pressure so produced, and have seen that it is inadequate to bring about a rise of capillary pressure in the limbs when the heart fails. We must, therefore, conclude that the ordinarily accepted notion is erroneous which assumes that the pressure in the capillaries and veins all over the body in uncompensated heart disease is raised. The pressure must in these situations follow the arterial pressure, and thus be lowered. The only parts of the body where there can be a rise of pressure in heart disease are the capillaries of the liver (whence the well-known "nutmeg" liver of this disorder) the great veins near the heart, and possibly in the system of capillaries which surround the tubules of the kidney. In the limbs, and probably in the abdominal viscera other than those just mentioned, the capillary pressure will be lower than in the normal individual.

For many years, and, indeed, since the time of Richard Lower, we have been wont to pride ourselves on our exact understanding of the factors responsible for the production of anasarca and ascites in heart disease, and have confidently ascribed the increased serous exudation to the rise of pressure in the capillaries due to the transmitted backward pressure from the incompetent heart, and the dropsy of heart disease is generally regarded as affording striking support to Ludwig's filtration hypothesis of lymph formation. The whole argument is, however, based on the false assumption that there is a rise of pressure in the smaller veins and capillaries in heart disease; whereas, as I have just shown, the reverse is the case. We must, therefore, seek for some other explanation of the dropsy in this disease.

VICTORIA HOSPITAL, SWINDON.—The ninth annual meeting of the Victoria Hospital, Swindon, was held on Feb. 13th. The report of the general committee showed the total expenditure for the past year to have been £419, against receipts amounting to £500; the sum of £107 was due to the treasurer on account of the building and furnishing fund. There were 153 patients admitted in 1896, against 131 in 1895, the average cost of each being 2*s*. 11¾*d*. daily. The committee suggested that the celebration to be held in Swindon on account of the Queen's long reign should take the form of raising funds for the benefit of the hospital.

A SUCCESSFUL CASE OF INTRAVENOUS INJECTION OF SALT SOLUTION AFTER LAPAROTOMY IN A CASE OF RUPTURED TUBAL GESTATION.

BY ARTHUR H. N. LEWERS, M.D. LOND.,
OBSTETRIC PHYSICIAN TO THE LONDON HOSPITAL.

ON April 20th, 1896, I was called in by Dr. Fennell of Dalston to see a married woman, aged thirty years, when the following history was obtained. She had been married nine years and had had four children, the last nineteen months previously. There had been no miscarriages. Instruments were used at the first confinement, but not at the others. She generally got up ten days after her confinements, but after the last confinement she was in bed nearly two months on account of what the medical attendant told her was peritonitis. Menstruation was quite regular up to Feb. 26th, 1896, but she "saw nothing" in March. She took some pills on April 4th with a view to bringing on the period; these gave her great pain. On April 6th a vaginal discharge containing blood began, and she had been losing blood more or less ever since up to the day when I first saw her (April 20th). The patient said that a solid substance came away from the vagina on April 11th when passing urine. She had had some pain in passing urine and had suffered from constipation since April 6th. She said that she had had pain at the lower part of the abdomen, especially over the left iliac region, for some days before I saw her, and there was an especially sharp attack of pain on the early morning of April 20th. On examination of the abdomen there was some resistance in the left iliac region and some tenderness there. The breasts did not appear active. On vaginal examination there was a little blood to be seen about the external parts. The uterus was anteverted and slightly flexed and distinctly larger than normal, although the sound only passed three inches. A swelling was felt behind and to the left of the uterus occupying the left posterior quarter of the pelvis. It was distinctly elastic and could be displaced a little by upward pressure. The case appeared to me in all probability to be one of extra-uterine pregnancy. The question arose as to whether the substance passed by the patient a few days before I saw her might not have been an ovum, and the case one of ordinary miscarriage, with possibly some peri-uterine inflammation; but, on the whole, it seemed to me much more probable that the case was really one of tubal pregnancy, and that the substance in question said to have been passed, if anything more than a clot, was probably a decidual cast of the uterus. The arrangements of the patient's home were not very convenient for performing abdominal section, and accordingly I had her removed to the London Hospital, where she was admitted under my care on April 21st.

Operation.—On April 23rd she was anæsthetised with the A.C.E. mixture; the abdomen was opened in the usual way and was found to contain blood and masses of clot. The left Fallopian tube was considerably enlarged and had ruptured. The left ovary contained a large corpus luteum with a well-marked yellow convoluted border. The uterine appendages on the right side were also slightly enlarged, and the uterus was considerably enlarged. The appendages on both sides were removed in the usual way and the peritoneum well washed out with saline fluid. A Keith's tube was inserted and the rest of the abdominal incision was closed. When this had been done it was observed before the dressing was put on that the Keith's tube had become full of dark blood. This was withdrawn once or twice, but the tube very soon became again full of blood. The abdomen was therefore re-opened to discover the source of the bleeding; the stumps of the broad ligaments were pulled up and inspected, but no blood was coming from them. On looking at the back of the uterus there were some quite superficial tears in the peritoneal coat near the level of the internal os, from which blood was coming rather freely. Several attempts were made to control this bleeding by passing silk ligatures on a curved needle under the tissues at the bleeding point and tying, and also by applying Wells's forceps; but the tissues were so friable that the more manipulation took place in the neighbourhood of the tears