Observations

ON

THE PATHOLOGY OF CARDIAC DROPSY.

BEING THE OLIVER-SHARPEY LECTURES DELIVERED
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BY

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(Abridged.)

LECTURE I.

[AFTER expressing his sense of the honour conferred upon him by the College, Dr. Bolton referred to the work of Lower, Cohnheim, and Starling, explained that the object of his researches was to produce experimentally uncompensated heart disease in order to study the resulting pathological processes, and proceeded as follows:]

Interference with the Filling of the Heart.

Experiment had shown that the heart, with its muscle in all respects intact, can so perfectly accommodate itself to increased work, that it will overcome an increasing resistance opposed to it up to a certain point, and thus maintain the arterial and venous pressures at their normal heights; beyond this point, however, when the heart cannot completely overcome the resistance, it suddenly fails and the circulation comes to an end. One cannot, therefore, experimentally interfere with the heart in its capacity as a force-pump so as to produce a gradual heart failure.

The alternative method of preventing the free diastolic filling of the heart proved successful. The means which I adopted to interfere with this function consists in constricting the pericardium by means of a row of sutures placed in the right side of that structure. This proceeding exercises a certain amount of pressure on the right auricle and roots of the great veins, and interferes with the free expansion of the ventricles. The animals, which in all the experiments to be described were cats, were anaesthetized with ether, and artificial respiration was maintained through an intubation tube during the operation, which was performed under strict antiseptic precautions. On the following day the animal has become dropsical, and this condition gradually increases in degree. The maximum amount of venous engorgement is found towards the centre of the body, the superior and inferior venae cavae and the liver, whilst the periphery appears practically unaffected. The distribution of the dropsy corresponds with that of the venous engorgement; there is ascites and double hydrothorax with marked oedema of the mediastinum, and to a less extent of the retroperitoneal tissue. This oedema may spread along the great veins, more particularly the superior vena cava, so that it extends out of the chest, giving rise to subcutaneous oedema of the front of the neck and chest, and even of the axillae. This distribution of the subcutaneous oedema is evidently influenced by gravity.

The immediate effects upon the circulation of the blood, as measured by arterial and venous manometers, I found to be the same as in Cohnheim's experiment. The arterial pressure at once fell to varying degrees in proportion to the tightness of constriction, and the venous pressure in all parts of the body, head, feet, and portal vein, at once rose also to varying degrees. On observing the experiment for an hour or more I was, however, surprised to find that the venous pressure in all parts of the body gradually fell until it returned to the normal level, whilst the arterial pressure still remained at the low level to which it had fallen. On the following day, when dropsy was present, I was again surprised to find that the venous pressures were still normal or a little raised, and that the arterial pressure had now usually regained its normal height.

When I made these observations we had no idea what the arterial pressure was in cardiac dropsy, but three years later H. J. Starling published a series of clinical observations in which he showed that in uncompensated heart disease with dropsy the arterial pressure was normal, except in cases associated with arterial or renal disease

when it was raised. The distribution of the subcutaneous oedema in the dependent parts of the body in cardiac dropsy is due to the upright position of the patient, so that the venous engorgement of the liver and veins of the central parts of the trunk is relieved by gravity at the expense of the legs. When it is necessary to tap the subcutaneous tissues, it is a common practice to let the patient sit up in a chair so as to drain the fluid into the legs. The changes are essentially the same as in these animal experiments, and the differences are entirely due to differences of position.

I concluded that the venous pressure fell owing to dilatation of the large veins of the trunk and of the liver, so that blood accumulated in the central parts, whilst it was abstracted from the periphery. In support of this supposition was the visible distension of these veins and the liver, and also the fact that, when the venous pressure had fallen to the normal level after its initial rise, relaxation of the pericardium brought about a further fall below the normal. The dilatation was due to the raised venous pressure and to a nutritional loss of tone in the vessels of the congested area. The dropsy was produced in this central area of the body whilst the capillary pressure was about the normal, and therefore I concluded that the oedema was primarily due to some alteration in the capillary wall.

From these observations it was quite evident that certain adaptive changes had occurred which were worthy of closer study, but the method of pericardial constriction was not a satisfactory one to employ for a large series of experiments, because so many animals died from the operation, as it was quite impossible to gauge the amount of constriction.

Obstruction of Both Venae Cavae.

I therefore employed another method for producing the same result. This consisted of simultaneous obstruction of the superior and inferior venae cavae. The constrictions were applied immediately above and below the right auricle, the azygos vein being ligatured. The average diameters of the veins were obtained by washing out the blood and distending them with gelatine at the normal venous pressure. After the gelatine had set the vessels were cut out and measured. The veins were constricted to varying degrees by tying round them short pieces of split rubber catheter of known diameters. I found that constriction of both veins led to a condition of dropsy precisely similar in its distribution to that produced by pericardial constriction, and that the effects upon the venous and arterial pressures were also the same.

Obstruction of Inferior Vena Alone.

An incomplete obstruction of the inferior vena cava alone produces no effect whatever unless the vein is narrowed down to three-fifths of its normal diameter; the animal survives, and develops dropsy. Obstructions greater than this produce similar results, but narrowing down below two-fifths of the diameter of the vein kills the animal as in complete ligature. One is thus able to guarantee the survival of any animal by using a rubber tube of 2.5 to 3 mm. in diameter, and also that the animal will develop dropsy. The ascites is rapidly produced, measurable amounts being present after two hours, and large amounts during the first few days; it then slowly increases up to the third or fifth week, as a rule, but sometimes for a longer period; it then slowly subsides with the establishment of anastomoses, and has disappeared in from two to six months. Now and then it may persist longer than six months. In addition to ascites there is commonly a little oedema round the inferior vena cava behind the peritoneum, and exceptionally it may extend from this locality down the iliac veins to the groin; here it forms a bag of fluid, which further extends along the anterior abdominal subcutaneous tissue, and down the thigh to the knee. This extension is due to gravity, the position assumed by the animal favouring this distribution of the subcutaneous fluid.

The appearance of oedema at a certain point in the subcutaneous tissue does not necessarily imply that it is formed in this position, because fluid is able to diffuse itself in the areolar tissues much in the same way as subcutaneous emphysema; in this form of diffusion gravity plays an important part.

Consequent Pressure Changes.

We have now to consider the pressure changes in the various parts of the circulatory system as a result of this obstruction of the inferior vena cava.

Immediate Changes.

A gradually increasing constriction produces no effect whatever on the pressures until the vein has been narrowed to three-fifths of its diameter, when the pressure at the lower end of the inferior vena cava rises by about 30 or 40 mm. MgSO_4 solution. The normal pressure in this situation is about 90 to 100 mm. MgSO_4 , so that the rise is about one-third of the normal. The solution was made of a specific gravity of 1.046, so that it is one-thirteenth the weight of mercury. A rise of 30 to 40 mm. therefore represents about 2 to 3 mm. Hg. At exactly the same point the arterial pressure falls by 20 mm. Hg or more owing to deficient filling of the heart. Constrictions greater than 3 mm. produce somewhat greater depressions in the arterial pressure curve and greater rises in the venous, and complete ligation of the vein causes the arterial pressure to sink to about 30 mm. Hg above zero, whilst the venous pressure behind the obstruction runs up rapidly to over 200 mm. MgSO_4 . To cause a high degree of venous pressure, therefore, the vein must be completely occluded. The rise of venous pressure is not limited to any portion of the venous area drained by the inferior vena cava, but extends back to the venous radicles at all points. Thus a cannula placed in a vein of the foot shows precisely the same increase of pressure in the manometer, and the rise of pressure is also propagated through the liver into the splenic vein to the same degree. On the other hand, the pressure in the superior vena cava and its tributaries falls, the fall amounting to about 10 or 15 mm. MgSO_4 solution.

We see, thus, that there is a redistribution of the amounts of blood in the different parts of the body; by its accumulation in the inferior vena cava and its tributaries it has been abstracted from the rest of the venous system and the arteries. The capillary pressure in all the parts drained by the inferior vena cava rises, whilst the velocity of the blood is diminished. This rise we have seen to be on the average about 2 to 3 mm. Hg, so that, if the normal capillary pressure were 30 mm. Hg, after obstruction of the vein the pressure would stand at 32 to 33 mm. Hg. This is a comparatively insignificant rise. We must, however, remember that the height of the capillary pressure depends not only upon the rate at which blood is leaving the capillaries, but also upon the rate at which it is being supplied to them. If the arterial pressure remained normal and these vessels contained the normal amount of blood, the capillary pressure after obstruction would stand at the height indicated above, but blood is abstracted from the arteries and the pressure in them falls; less blood than normal is therefore supplied to the capillaries, so that for this reason the pressure within the latter is not raised to the level indicated by the height of the venous pressure. Exactly what lessening of effect is produced by the fall of arterial pressure it is impossible to say; we can only conclude that the capillary pressure is raised, but to a smaller extent than one would expect from the height of the venous pressure, and in any case the rise is trifling. The fall of venous pressure in the superior vena cava and its tributaries, together with the fall of general arterial blood pressure, naturally results in a fall of capillary pressure in these parts.

These are the immediate effects of narrowing of the inferior vena cava, and they do not last for very long, because the venous pressures in all the parts of the body behind the obstruction commence to fall gradually, and, if the animal be kept under observation for an hour or more, it will be found that the venous pressures in the inferior vena cava itself, in the portal vein, and in the foot eventually reach the normal level or a point very near it. The venous pressure in the head, however, at the end of this time still remains at the low level it had immediately assumed on obstruction of the vein. The arterial pressure in all cases remains at the level to which it had fallen on the occurrence of obstruction, or it may be lower still. The higher the pressure in the inferior vena cava the quicker it falls to the normal level.

This fall of venous pressure is due to precisely the same cause as we have seen to be operative in constriction of the pericardium—namely, dilatation of the veins and capillaries of the liver more especially, but also of the

portal system, and of the inferior vena cava and its immediate tributaries.

This distensibility of the abdominal vessels is responsible for a further depletion of the fore part of the body, and the hind limbs and peripheral portions of the abdomen now share in this depletion. Owing to the increased capacity of a part of the circulatory system, the pressures in all parts of the system naturally fall, the blood volume remaining normal. The venous pressures in all parts behind the obstruction being normal or thereabouts, and the arterial pressure low, it naturally follows that the capillary pressure is subnormal in these parts, except in the liver, where it is about normal. The capillary pressure in the fore part of the body falls lower than it was before.

This is the condition of the circulation when dropsy commences; an increased output of lymph occurs in the plethoric areas giving rise to ascites and some degree of retroperitoneal oedema. The ascitic fluid comes from the viscera, especially the liver, and the oedema from the capillaries of the short lumbar veins directly opening into the inferior vena cava. The veins of the abdominal wall and the hind limbs actually contain less blood than normal. Increased absorption of fluid occurs in these regions and also in the fore part of the body owing to the deficient filling of the vessels and the resulting low capillary pressure.

The output of lymph in the plethoric areas is thus balanced by the absorption of fluid in these depleted areas, the blood volume remaining normal. It is well known that such an absorption always occurs, because Lazarus-Barlow has shown that, when a part is rendered plethoric and oedematous, the specific gravity of the tissues in the anæmic areas of the body rises, showing that fluid has been absorbed from the tissues into the blood.

The capillary pressure being normal or thereabouts, the increased output of lymph is entirely conditioned by the state of the vessel wall. Exactly what this state is one can merely surmise, but it can hardly be doubted that the normal nutritional exchanges of a part in which there is such a high degree of stasis of blood are seriously interfered with. So far as we can see at present the fault is primarily a nutritional change in the capillary wall, and what part is played by filtration at the normal capillary pressure, or by osmosis resulting from altered tissue metabolism, must at the present moment be left an open question.

All the changes which I have hitherto described occur during the first few hours after the vein is obstructed, and we have now to consider what later alterations in the pressures take place. For this purpose a series of dropsical animals was examined at various intervals after the operation until the dropsy had disappeared.

Later Changes.

The later alterations occur in two stages, and they are due to two distinct causes:

1. The first stage is characterized by a rise of venous pressure, approximately to the height from which it had fallen, and a rise of arterial pressure to about the normal level.
2. During the second stage the venous pressure rises to a high degree, the pressures which I have recorded being comparable to those following complete ligation of the vein. The arterial pressure still remains at the normal level.

First Stage: Restoration of Venous and Arterial Pressure.

The alterations in the circulation at the first stage are usually completed in a few hours, nearly always within twenty-four. In one case on the fifth day, however, I have found a normal venous and subnormal arterial pressure, although the animal had 100 c.cm. of fluid in its abdomen. At this stage the venous pressures in the portal vein and inferior vena cava gradually rise until they attain the heights at which they stood immediately after the operation. The pressure, however, in the veins of the legs always remains relatively lower than that in the central part of the trunk. This pressure does not rise above the normal, and unless the pressure in the trunk is very definitely raised it is subnormal. There is, therefore, a relative alteration in these two pressures; the venous pressure in the trunk is always relatively raised, and that in the legs relatively lowered. The arterial pressure rises to the normal, varying somewhat with the venous pressures

in the trunk. The venous pressure in the territory of the superior vena cava still remains at the low level it immediately assumed when the operation was performed. This again shows, as we saw before, a relative plethora of the central parts of the trunk, and depletion of the peripheral parts of the circulation.

The Cause of this Alteration in the Venous and Arterial Pressures.

In the normal animal the arterial pressure is kept at a constant level in order that the capillaries may be supplied with an adequate flow of blood. This especially applies to the brain, so that the medulla may receive its normal supply of oxygen. Whenever the arterial pressure falls, so that the vasomotor centre does not receive its proper supply of blood, it is excited to action and the blood pressure rises. One would expect, therefore, that the low arterial pressure in the dropsical animals would excite the vasomotor centre to action so as to produce a general arterial constriction, with its resulting rise of arterial pressure and a greater flow of blood to the brain. It might, however, be argued that in these cases absorption of fluid had overbalanced output and brought about hydraemic plethora, and that this increase in the total blood volume was the cause of the raised vascular pressures. I have, however, shown that at this stage of the disease there is no increase in the blood volume. It must therefore be by vasomotor constriction, which automatically occurs by excitation of the vasomotor centre as the result of anaemia of the medulla, that the arterial pressure becomes raised to the normal. Naturally, this result varies in different animals, according to their power of recovery after a severe operation. In some cases it rapidly occurs, so that the arterial pressure rises to its originally normal level in three or four hours, whilst in others the pressure still remains low on the following day or even later. It was shown by Bayliss and Starling that stimulation of the splanchnic nerves not only raises arterial blood pressure, but also the pressure in the inferior vena cava by 20 to 30 mm. $MgSO_4$ solution.

Vascular constriction, therefore, fully accounts for the rise of arterial and venous pressures. It is an adaptive mechanism enabling the animal to compensate as far as possible for its changed circulatory conditions. The rise of arterial pressure ensures a normal supply of blood to the brain, and the raised venous pressure increases the velocity of blood through the constriction, so that the maximum amount possible may reach the heart and be distributed to the tissues.

These alterations in venous and arterial pressures in their turn affect the capillary pressure. The rise of venous pressure results in a rise of capillary pressure in the central parts of the trunk, but this rise is minimized by the arterial constriction which cuts down the supply of blood to the capillaries, so that it is doubtful whether the capillary pressure rises above its normal level except in the liver, where it must go up, because both portal and inferior cava pressures are raised. The capillary pressure in the legs, abdominal wall, and fore part of the body is subnormal, and increased absorption continues in these regions as before. The alteration in pressures does not obviously affect the output of lymph, because there is no distinct relation between the height of the venous pressure in the trunk and the amount of ascites present.

Second Stage: Rise of Venous Pressures to a Great Height.

In these animals the venous pressures are very high after the first week and occasionally during this period, but it is not until the end of the second week that they attain their maximum heights. The maximum pressures occurred from the second to about the eighth week. The pressure in the portal vein followed that of the inferior cava, and was raised to about the same proportionate extent. The venous pressure in the hind limbs was always raised above the normal after the second week, but to a less relative extent than that of the inferior cava. Although all the venous pressures have gone up, there is still the same alteration in the pressure levels as described before, the venous pressure in the trunk being relatively more raised than that in the hind limbs. This occurrence is, therefore, a constant feature at all stages of the disease. Another constant feature refers to the pressure in the superior vena cava, which in all cases was still below its

normal level, as immediately after the vein was constricted. The same disproportion in the distribution of the blood as we saw to occur in the early stages is, therefore, still present. There is a relative plethora of the central portion of the trunk, and relative anaemia in the peripheral parts. The arterial pressure was normal, although in four cases it was a trifle higher than usual. The capillary pressures are raised in all parts behind the obstruction, but to a less relative extent in the hind limbs than in the trunk; those of the fore part of the body are below normal.

With regard to the influence of this high venous pressure upon the production of dropsy, it may be definitely stated that there is no constant relation between the height of the venous pressure and the amount of ascitic fluid present, and that, moreover, during the subsidence of the ascites and after it has disappeared the venous pressure still remains high. These observations upon the venous pressures in the later stages of the disease, therefore, agree with those made in the earlier stages in showing that a high capillary pressure is not the primary cause of the dropsy and is not necessary for its production. Neither is the presence of a high pressure capable of preventing the subsidence of the dropsy. It is true that the period of highest pressure corresponds with some increase in the ascitic fluid, although an examination of individual cases does not bring out this point, because the ascites begins to subside at different times in different animals whilst the pressure is still high. So that when once oedema is being produced a high capillary pressure will increase it, although the two conditions may be dependent upon some other factor.

It seems quite clear that some new factor must have been introduced to cause this additional rise of venous pressure, which in half the cases becomes more than double its original height. Such a factor can only be an increase in the total quantity of the blood, and this leads us to inquire whether absorption in the anaemic parts of the body may not have eventually overbalanced output in the plethoric parts and led to a condition of general plethora; and, further, if the total blood quantum is increased, what is the nature of the increase, and what is its effect upon the production of lymph?

Establishment of Plethora.

That plethora occurred in certain forms of dropsy had been suggested by clinicians for many years before Cohnheim definitely opposed the idea. He considered it as proved that a lasting pathological increase in the blood quantum could not occur, because it had been shown that, when the blood volume of the dog was increased by transfusion of large amounts of blood, the excess was disposed of in a relatively short space of time.

I am aware of one case only in which the blood volume of a patient with uncompensated heart disease was actually estimated. This was an observation of Lorrain Smith, in which he found an increase in the blood volume. I have estimated the blood volume of twenty-five dropsical cats at all stages of the disease. The volume of the blood was obtained by the washing out method. The blood volume of a cat weighing 2,500 grams is about 100 c.cm. Ten estimations were made on cats during the first week. In five cases the blood volume was normal, in three it was diminished by 6, 9, and 16 c.cm., in two it was increased by 4 and 7 c.cm., so that during the first week the blood volume is normal or decreased; an increase may occur at this early date, but it is very slight and unable to affect the venous pressure. This period, it will be remembered, is that during which the venous pressure is raised by vasoconstriction alone. Four estimations were made during the second week. The blood volume of the animal on the eighth day was diminished by 10 c.cm. and the other three increased by 6, 15, and 28 c.cm. Towards the end of the second week, it will be remembered, the venous pressures tend to rise and afterwards remain uniformly high. Between the second and eighteenth week eleven estimations were made, and all showed an increase. The increases at this period were on the whole much greater than those of the second week. The lowest figures were from 7 to 18 and the highest from 23 to 47 c.cm. Considerable increases in the blood volume thus occurred during the period when the venous pressures were uniformly very high. It seems justifiable to conclude that about the second week absorption of fluid overbalances

output and leads to an increase in the total blood volume. This causes a rise of venous pressure in the parts of the body behind the obstruction, but the quantity of blood in the previously anaemic area is not made up to the normal, and the pressure in this region is subnormal. Absorption, therefore, continues and keeps the blood volume above the normal. This implies a fairly rapid circulation of fluid into and out of the blood, and that this actually happens we shall see later in speaking of the extravascular circulation of the lymph.

This rapid and constant intake of fluid, therefore, seems to be the actual cause of the plethora, otherwise, as in Cohnheim's experiment, the excess of blood would be rapidly disposed of and the blood volume return to the normal. The condition of plethora in these experiments is not analogous to the artificial production of plethora by injection of fluid, because its maintenance depends upon a constant intake of fluid. Fluid is also rapidly leaving the vessels as ascites, but, as I shall show later, absorption of the ascites is constantly occurring into the blood so that by this rapid and constant circulation of fluid the volume of blood is permanently increased. During the first two weeks the blood volume bears no relation to the amount of ascitic fluid. The formation of ascites is very active from the first day and the fluid accumulates in large quantities during the first week. During this period the volume of blood is usually normal or even diminished, and as a fact the amount of ascites is commonly large when the blood volume is small. The increase in blood volume can in no way, therefore, be said to be the cause of the dropsy, because the essential cause or causes are at work before the blood increases in volume. The increase in blood volume must, therefore, be looked upon as a secondary phenomenon and due to an effort of the animal to make up its normal quantity of blood in the anaemic areas, and to replace the fluid lost as dropsy. At the same time it may be a contributing factor and assist in increasing an already existing dropsy. This appears to be the case, for the dropsy tends to increase about the time the volume of the blood increases, and after the second week is passed the amount of ascitic fluid varies more or less directly with the blood volume. However this may be, after the fifth week, when the dropsy is subsiding, the blood volume still keeps high. So that an increase in the blood quantum is unable to prevent the disappearance of the dropsy.

A further effect of this increase in the volume of the blood and the resulting high venous pressure is seen in the liver. In the early stages we have seen that the pressure in it is very little above the normal, and the capillaries are dilated moderately; in the stage of plethora, however, the pressure in this organ is considerably raised, the capillaries are dilated into sinuses, and the liver tissue atrophies.

It is obvious that if the total amount of fluid in the body is increased water must have been retained. The sources from which the blood derives fluid in order to increase its volume are the alimentary canal and the extravascular tissues. The daily volume of urine is diminished to about one half during the first week, after which it becomes normal, with a tendency towards an increase in the later stages. Less fluid is, however, consumed during the first few days, and, as a fact, the amount of fluid taken in and that excreted run parallel throughout the disease. Taking into account all sources of error involved in the method employed, it appears that there is retention of fluid in the body during the early period of oedema formation and increase of the blood volume, and a slight loss of fluid during the subsidence of the dropsy. That the excretory capacity of the kidneys in these animals is not materially interfered with is proved by the readiness with which they respond to an increase in the available amount of fluid presented to them to excrete. The retention of water appears, therefore, to depend primarily upon the circulatory processes at work, which are causing the dropsy.

The fluid which is absorbed directly into the capillaries from the extravascular spaces, so far as is known at present, consists of water and salts only. There are no direct observations to prove that protein is absorbed by the capillary wall.

Changes in Haemoglobin Content.

During the past twenty years or more the condition of the blood in uncompensated heart disease as regards its content of water has been investigated from time to time. The methods employed have been either that of ascertain-

ing the total solids of the blood, of measuring its specific gravity, or of ascertaining the percentages of haemoglobin and red corpuscles which it contained. Many of these clinical observations agree that in cardiac dropsy hydraemia is present, but others differ from them in finding the haemoglobin content of the blood normal or even increased.

I have made 182 haemoglobin observations on 52 dropsical cats, and have estimated the total solids of the blood, serum, lymph, and ascitic fluid of 11 cats at various stages of the disease. These observations have shown that on some day during the first week the percentage of haemoglobin in the blood begins to decrease. This never occurs on the first day after the operation, every case examined at the end of twenty-four hours showing a normal or slightly increased percentage. It may commence on the second day, but only in about one out of six cases. Two-thirds of the cases show a commencing fall of the haemoglobin percentage on the third day, and the remainder on the fourth, fifth, and sixth days. One case showed a normal percentage on the eighth day. With this last exception every case out of seventy-six estimations, conducted from the seventh day to the end of the fifth week, showed a decrease in the haemoglobin percentage. After this period, although the blood volume remains increased, the haemoglobin percentage rises, becoming normal or slightly above this level in most of the cases during the next few weeks.

On comparing the haemoglobin percentage with the blood volume it appears that the period of continuous increase in the blood volume corresponds with the period of continuous decrease in the haemoglobin percentage, and that after this, although the blood volume remains higher than normal, the haemoglobin percentage commences to rise. It is therefore evident that the decrease in the haemoglobin content of the blood is due to a dilution of the blood, and not to a decrease in the total amount of haemoglobin. The rise in haemoglobin percentage whilst the blood is increased in volume represents an effort on the part of the animal to restore this percentage to the normal. This it succeeds in doing sometimes whilst the ascites is present, and sometimes after it has disappeared.

Here is to be found the explanation of the discrepancies which have occurred in clinical observations on the haemoglobin content of the blood. The alterations in the haemoglobin percentage and the corpuscular content of the blood pass through very definite stages, so that a normal, an increased, or a diminished percentage in either of these is found at different times in the same individual. The formation of new blood corpuscles is probably due to the diminished oxygen tension of the blood. This is brought about in uncompensated heart disease by the diminished velocity of the blood and the cyanosis.

Changes in Blood Plasma.

We have now to discuss the changes which occur in the blood plasma. The total solids in the ascitic fluid amount to about 5 or 6 per cent., and occasionally 7, being slightly less than those of the lymph in any given animal. A considerable amount of protein is thus rapidly lost by the blood, the diminution in volume of the latter being made up by the absorption of water. There results, therefore, in the earliest stages a simple hydraemia, in which the total volume of the blood is normal, but the percentage of water in the plasma is increased. The total solids of the blood and serum are diminished, but the haemoglobin percentage is normal, showing that the total blood volume is normal.

We know very little about the formation of the blood proteins beyond the fact that they are formed fairly rapidly. My observations on the total solids of the blood and the serum fully bear out this point, because I have found that after the first day, whilst the total solids of the blood are uniformly and considerably diminished, together with the percentage of haemoglobin, the solids of the serum are only a little less than normal. This means that the animal rapidly makes up the proteins of its plasma, so that the preliminary hydraemia tends to diminish. After the fifth week has passed, and the excessive lymph production is falling off, the total solids of the serum become absolutely normal.

We must therefore distinguish several phases through which the blood passes: A simple hydraemia, the total

volume of the blood being normal or even diminished; a plethora in which the plasma is to some extent hydraemic and the percentage of corpuscles diminished; a true plethora in which the plasma is normal in composition and the percentage of corpuscles has also reached the normal. The proteins of the blood are thus made up much more rapidly than the corpuscles, so that one cannot say that hydraemic plethora exists merely because the corpuscles are diminished in percentage. Were it not for the constant absorption of water and output of protein, tending to keep the blood hydraemic, the proteins of the blood would, no doubt, be found normal in quantity from an early date. We have already seen how the organism attempts to compensate for the anaemic condition of certain portions of its vascular system by vaso-constriction, whereby it ensures that the maximum amount of blood possible reaches the tissues. It has only a certain quantity of available blood at its disposal, and with this it is unable to supply the tissues with their normal amount. A further series of events is therefore initiated whereby the blood is first increased in quantity, and then becomes normal in quality.

SUMMARY. *

1.

The increased output of lymph giving rise to cardiac dropsy is one of a very definite series of events following the occurrence of failure of the heart in its capacity as a pump. These events may be grouped into the following stages:

Stage I.—Accumulation of blood in the veins, with a moderate rise of venous and capillary pressure in all parts. Abstraction of blood from the arterial system with a fall of arterial blood pressure.

Stage II.—Dilatation of the veins and capillaries of the central parts of the body, with a fall of venous and capillary pressure to normal or thereabouts. This leads to increased congestion of the central parts and anaemia of the peripheral parts of the body, in which the venous and capillary pressures fall below normal. Increased output of lymph in the congested area giving rise to dropsy. Increased absorption of water in the anaemic areas, which balances the loss of fluid by the blood. Simple hydraemia results.

Stage III.—Vasomotor constriction resulting from anaemia of the medulla causing:

1. A rise of arterial pressure to the normal.
2. A moderate rise of venous and capillary pressure in the central parts of the trunk.

The venous and capillary pressures of the peripheral parts remain subnormal, or may become normal.

Stage IV.—Hydraemic plethora is established and raises the venous and capillary pressures in the central portions of the body to a great height. There is still a relative anaemia of the peripheral portions, although to an increasingly limited extent, as the congested area spreads peripherally.

Stage V.—The ascites and hydrothorax increase in degree and the oedema spreads in the areolar tissues peripherally, as the congested area encroaches upon the anaemic regions. True plethora is established.

2.

The abnormal output of lymph leading to dropsy is primarily conditioned by the nutritive state of the capillary wall, and is increased by the general plethora and consequent high capillary pressure.

3.

In uncompensated heart disease in man the central portions of the trunk are relieved by gravity at the expense of the legs, which become included in the congested area. The dropsical area spreads up the legs to the abdominal wall, thus encroaching upon the anaemic area from below upwards.

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APPARATUS FOR TREATMENT OF FRACTURES OF THE FEMUR AND OF THE LEG.

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THE apparatus which I am now describing permits the treatment of fractures of the femur and of the leg in a position of extension as well as of flexion of the hip and knee-joint. In the flexed position the action of certain muscles often becomes less disturbing, while the relaxation of these muscles decreases the force required for successful traction. Especially for certain cases of high and low fractures of the femur, the treatment by flexion offers real advantages. I have therefore devised an apparatus for the treatment

FIG. 1.

of fractures of the femur in extension or in flexion, which permits a change of position when required, without the necessity of first removing or displacing the apparatus, which, moreover, allows the simultaneous treatment of fractures of the femur and of the leg. It is made of metal, and consists of two lateral splints, united above by a posterior arc, which has, in its internal part, a special

FIG. 2.

curve similar to that in M. Delbet's apparatus. This special curve provides a good leaning on the ischium, a point well adapted to sustain a certain amount of pressure. At the further end the two splints are united by a transverse bar, in the middle of which is applied a dynamometer, which regulates the strength of the traction exercised by means of diachylon, network, plasters

FIG. 3.

Finochietto's stirrup, etc. The dynamometer can be replaced by elastic tubes or other springs, etc. The apparatus consists of two parts, articulating in a special manner corresponding to the knee-joint; the upper part supporting the femur, and the lower part the leg. In order to give the appliance more rigidity, two transverse arcs unite the two splints, and a large support at each end gives additional stability to the apparatus, which should be placed upon a firm base. Linen or metal straps form the ham-mock which carries the limb.

A number of holes in the splints facilitates the suspension of the apparatus, the femoral part of which can be