

Capillary Pressure in Right Heart Failure.

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This investigation was undertaken to determine whether increased hydrostatic pressure within the capillaries was sufficient to explain the formation of edema in right heart failure without recourse to other factors such as anoxemia and increased permeability of the capillary membrane. Landis¹ has determined the gradient of intra-capillary pressure in the capillary loops of the human skin by introducing a very fine glass cannula directly into the blood capillary. He determined the pressure in the arteriolar limb, at the venous end and near the summit of the loop. Landis' method, being a direct pressure measurement, is the most accurate method for measuring capillary blood pressure. The average pressure of 125 observations in the arteriolar limb was 32 mm. Hg. and the average pressure of 99 observations in the venous limb was 12 mm. Hg., according to Landis. The average pressure from 19 observations in the summit of the loop was 20 mm. Hg. We have made determinations of the capillary blood pressure on 6 normal adults, making use of Landis' method. The values for the arteriolar limb were 30-30-30-32-35-40, or an average of 33 mm. Hg. It was only possible to make observations of the blood pressure in the summit of the loop in 2 cases. Both of these gave values of 18 mm. Hg. Measurements of the hydrostatic pressure in the venous limb of the capillaries of 6 adults gave values of 15-12-12-12-12-10 or an average of 12 mm. Hg. These values agree extremely well with the average values obtained by Landis.

The venous pressure as well as the capillary blood pressure in the venous end of the capillary loops were measured on 5 cases of right heart failure. Edema was present at the time the measurements were made. Table I contains the values observed. In a case of mitral stenosis with right heart failure and edema (Case 3, Table I) the venous pressure was 12 mm. Hg. and the capillary blood pressure in the venous limb was 25 mm. Hg. The patient became compensated, edema disappeared and another measurement showed venous pressure of 5 mm. Hg., capillary blood pressure in the venous limb 12 mm. Hg., in the middle of the loop 18 mm. Hg., and in the

¹ Landis, E. M., *Heart*, 1929-31, 15, 209.

arteriolar limb of the capillary loop 30 mm. Hg. In all of these cases the hydrostatic pressure within the venous limb of the capillary loop was considerably higher than the mean colloid osmotic pressure of the blood, which is about 21 or 22 mm. Hg.² In one case it was found that when the patient became compensated and edema disappeared, the hydrostatic pressure within the capillary loops was normal and of such value that the passage of fluid from the capillary to the outside in the arteriolar limb would be exactly compensated by the movement of fluid into the capillary loop from the outside in the venous end of the loop. These figures are the first measurements of capillary pressure in right heart failure by the direct method. They show that it is not necessary to assume any other mechanism for the formation of edema in these 5 cases of right heart failure than an increase in the mean hydrostatic pressure along the capillaries definitely above the colloid osmotic pressure of the blood.

TABLE I.

	Venous Pressure*	Capillary Pressure*		
		Arteriolar Limb	Summit of Loop	Venous Limb
Landis' Aver. Normal Adults		32	20	12
Ershler's Aver. 6 Normal Adults		33	18	12
Case 1	10			35
" 2	24			41
" 3 A	12			25
" B	5	30	18	12
" 4	10			25
" 5	27			35

A = edema present.

B = " absent.

* = all measurements given in mm. of Hg.

It has been assumed that dilatation of the capillaries possibly in consequence of anoxemia leads to increased permeability of the capillary wall and thus to edema formation. We have 3 observations which tend to show that dilatation of the capillary alone is not sufficient for edema formation if the intracapillary pressure is not raised. In one case of congenital heart disease the capillaries had a diameter of twice the normal size but the capillary pressure in the venous limb was 12 mm. Hg., in the middle of the loop 20 mm. Hg., and in the arteriolar limb 40 mm. Hg. No edema was present. In 2 cases of polycythemia the diameter of the capillaries was twice the normal. In one of these cases the pressure in the arteriolar limb was 35 mm. Hg. and in the venous limb 15 mm. Hg. In the other

² Kerkhof, A. C., *Ann. Int. Med.*, December, 1937, in press.

case the pressure in the arteriolar limb was 30 and the venous limb was 10. In neither of these cases was any edema present.

In one case of glomerulonephritis with a strong nephrotic tendency with edema, ascites and hydrothorax, without any heart failure but with a colloid osmotic pressure of approximately 9 mm. Hg., the capillary pressures were 30 mm. Hg. in the arterial limb and 22 mm. Hg. close to the middle of the loop. The pressure in the venous end of the capillary loop was not measured but the pressure in the median basilic vein was 3.7 mm. Hg. This shows that with normal hydrostatic pressures in capillary loops but with a lowered colloid osmotic pressure, it is possible to get edema, despite the fact that the capillaries in this case were extremely small. A diminution in the diameter of the capillary did not prevent edema formation when the colloid osmotic pressure was lower than the mean pressure along the capillary.

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Venous Pressure as a Factor in Determining Collateral Circulation in the Heart.

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In tying off the left descending coronary artery of the dog, it was noted by LaBree¹ that the apex was rarely infarcted if the veins were tied at the same time as the artery. Gross² showed that there was a markedly increased opening up of collateral circulation if the coronary artery of a dog was ligated after first ligating the coronary sinus. Many clinicians have noted that angina pectoris symptoms and the symptoms of coronary thrombosis are apt to be less severe in cases with severe right heart failure, *i. e.*, with a high venous pressure. With these observations in mind, we decided to ligate the left circumflex artery both with and without previous ligation of the accompanying veins and also after the production of a high pressure in the coronary sinus, one approximating the venous pressure in the coronary sinus during severe heart failure.

¹ Personal communication.

² Gross, Louis S., Blum, Lester, *PROC. SOC. EXP. BIOL. AND MED.*, 1935, **32**, 1578.