ROLE OF VENOUS CONGESTION IN COMPENSATION OF TRICUSPID INSUFFICIENCY.

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The possibility of the establishment of compensation in the lesions of the tricuspid valve was doubted by Romberg (Cf. Franke[2]) and hesitatingly admitted by Krehl (Cf. Franke[2]) on account of the weakness of the right auricle, which is the only cavity of the heart left to perform the task. MacKenzie[1] and others have, however, described cases of tricuspid insufficiency lasting over long periods. Recently, Franke[2] has cited several cases where, in the light of the literature, he believes that dilatation and hypertrophy of the right auricle and ventricle, venous congestion, and especially congestion of the liver were factors in maintaining compensation.

The clearness with which the rôle of venous congestion as an element in compensation is demonstrated makes the following case worthy of record:

History.—The patient, an adult white female with a long-standing heart lesion, developed ascites. There was mitral insufficiency, secondary tricuspid insufficiency, and enlargement of the heart which extended beyond the anterior axillary line. The usual murmurs were present. The jugulars were greatly distended. Shortness of breath had been pronounced for many months. Edema of the lower extremities, varying in extent, had been present for a long time. There had been periods of broken compensation.

With the onset of swelling of the abdomen, these signs were not materially changed. The patient had not been seen for a number of weeks when, on account of urgent dyspnea and rapid action of the heart, the physician was called.

Examination.—The heart was slightly displaced upward. The action was so tumultuous that, on auscultation, nothing could be determined with certainty. No radial pulse could be found. The jugulars were collapsed in diastole, and in systole presented a very large wave. The face was pale and the lips showed but a little color. There were no signs of congestion about the face, neck or upper extremities. Dyspnea was distressing. The abdomen was greatly distended and tense, the subcutaneous edema extensive. The lower extremities were greatly swollen and shapeless.

Treatment.—The abdomen was tapped and immediately after the pressure was reduced the following conditions were noted: The rate of the heart's action became regular and slower; the radial pulse returned. On auscultation, half an hour later, the murmurs had returned. The jugulars were greatly distended and the systolic pulsation reduced in extent. The face was at first congested, with the

1. Am. Jour. Med. Sc., 1907, cxxxiv, 12.
2. Wien. klin. Wchnschr., 1906, xix, 956.

lips blue. Within a few minutes, however, the lips became red and the respirations were slower, deeper and easier. After the first reaction the patient became more comfortable.

The points of especial interest in this case appear to be the following:

1. (a) The congestion of the superior cava, caused by the cardiac lesion, continued until the abdominal pressure obstructed the inferior cava, after which the branches of the superior cava were freely drained by the heart. (b) The congestion of the superior cava reappeared as the obstruction of the inferior cava was removed by reducing the abdominal pressure. (c) The diminution of the congestion of the superior cava was followed by a large systolic venous pulsation which diminished in extent as the congestion returned.

2. The heart's action was phenomenally tumultuous and the quantity of blood delivered to the arteries was very small; there was no palpable radial pulse.

3. Withdrawal of fluid from the abdomen was followed by congestion of the veins and lips, a retardation of the heart's action, a very great improvement in the character of the radial pulse, and a relief of the dyspnea.

4. The previous history pointed to the existence of tricuspid insufficiency.

Physiologic data show that the capillary pressure may vary from zero to 50 mm. of mercury, ranging under normal conditions from 10 to 30 mm. If a ligature be placed about a finger, the capillary equals the static arterial pressure. It is, moreover, fallacious to argue that a fall of arterial pressure necessarily produces a fall of pressure in all of the capillary areas of the body.[3] The capillary pressure is normally from one-sixth to one-third of the arterial pressure.

The venous pressure is also influenced by the arterial pressure. Schäfer[4] says: "When all the venous exits from any part are ligated, then the pressure rises, peripherally to the seat of ligation, to the arterial pressure."

If the pressure in an obstructed vein from any part may rise to the arterial pressure, the same principle should apply to the entire venous system. This has been shown to be true by Cohnheim's[5] experiments on pericardial pressures. Cohnheim inserted canulæ in the pericardium, femoral artery and jugular vein. Oil was injected into the pericardium and pressures were measured in millimeters of oil for the pericardium,

3. Schäfer, Text-Book of Physiology, ii, 116.
4. Vol. i, p. 121.
5. Allgemeine Pathologie, 1882, Bd. 1, 22.

in millimeters of mercury for the femoral artery, and in millimeters of soda solution for the jugular vein. The results were as follows:

1. Thirty mm. of oil pressure was taken up by the elasticity of the pericardium without change of pressure in either the femoral artery or jugular vein.

2. Sixty to 70 mm. of oil pressure caused a drop of 20 to 30 mm. of mercury in the femoral artery and a rise of 60 mm. of soda solution in the jugular vein.

3. One hundred to 120 mm. of oil pressure produced a fall in the femoral pressure to half its original height and a rise to 100 mm. of soda solution in the jugular vein.

4. Two hundred and forty to 320 mm. of oil pressure caused a cessation of pulsation in the pulmonary artery, but the femoral continued to pulsate and registered a pressure of from 10 to 15 mm. of mercury; the venous pressure was 220 to 240 mm. of soda solution.

On removing the pericardial pressure the circulation returned to its original condition, save for a few minutes when the arterial pressure was above its original point. If in (4) the femoral pressure is converted into terms of soda solution, 133 to 200 mm., it is apparent that the venous pressure equals the arterial when the flow from the veins is blocked and the system becomes a closed pipe. In other words, the circulation in the systemic arteries, capillaries and veins is really one and is controlled by the same principles as those governing the action of fluids in an open or closed system of pipes.

In a closed system the pressure is the same throughout, as illustrated in Cohnheim's experiment when the venæ cavæ were blocked, as in (4). Here the pressure in the cavæ and arteries was equal. In a partially closed system the pressure in the end distal to the pressure head is equal to that of the pressure head minus friction and flow. In Cohnheim's experiment the venæ cavæ were partially closed as in (3). The decreased flow of blood caused the arterial pressure to fall and the venous pressure to rise. The system being partially closed, the pressure throughout was proportionately equalized. In the normal condition the open ends of the system, the venæ cavæ, register zero or less. Here all of the pressure head is consumed in friction and flow.

The storage of blood in the veins is due to the arterial flow, and the pressure under which the blood is maintained in the veins is the arterial pressure minus friction and flow. The venous congestion may be compared to a closed system in which, while the blood is stationary, the pressure represents potential energy; while flowing, kinetic energy. During life, venous congestion is associated with and to a degree equal to the

active force obstructing the flow of blood in the vein. It represents the potential energy of the arterial pressure or left ventricle, transferred from source of power of a partially closed tubular system to the distal end, to be used, as kinetic energy, in overcoming or compensating the force obstructing the flow of blood in the vein. The left ventricle compensates venous obstruction.

Supported as it is, this theory as to the nature of venous congestion should form a safe basis for a consideration of some of the mechanics of pathologic circulatory changes. It is generally acknowledged that mitral insufficiency is usually associated with hypertrophy and dilatation of the left auricle and ventricle, pulmonary congestion and varying degrees of hypertrophy of the right ventricle (compare Mority[6]). In tricuspid insufficiency, hypertrophy and dilatation of the right auricle and ventricle may be compared with the similar changes in the left heart in mitral insufficiency; while the general venous congestion may be regarded as analogous to the pulmonic congestion. The rôle of the right ventricle in mitral lesions has, however, no counterpart in disease of the tricuspid valve. Both Stadler[7] and Franke[2] assert that, because of atrophy, it is impossible for the left ventricle to play a part in the compensation of tricuspid insufficiency.

In the case cited, the course of events was probably somewhat as follows: There was hypertrophy and dilatation of the heart, tricuspid insufficiency and intense venous congestion. The development of a terminal ascites gradually obstructed the inferior cava. As this obstruction developed, the flow of blood to the heart diminished until the superior cava and its branches were relieved of their former congestion, thereby removing all traces of congestion from the head and upper extremities. The jugular vein was collapsed in diastole, and in systole a large pulsation, due to backflow from the right ventricle, was present. The presence of the backflow into the superior cava which was not congested shows that, when the tricuspid lesion had developed, the backflow takes no part in the causation of congestion.

In this case the superior cava was an open system in which the pressure at the extremity distal to the power should be low; while the power was much reduced, thereby diminishing the amount of blood which could, under low pressure, be forced through the capillaries. The low arterial pressure was indicated by the absence of radial pulse. The right ventricle was competent to move forward all the blood delivered by the venæ cavæ, as was shown by the condition of the superior cava. This

6. Deutsch. Arch. f. klin. Med., 1899, lxvi, 421.
7. Deutsch. Arch. f. klin. Med., 1905, lxxxiii, 71.

competency of the right ventricle was due to rapid and forcible action of the heart. The low pressure in the superior cava developed when the amount of blood delivered to the systemic veins was less than that delivered to the pulmonic artery. The inferior cava, obstructed by the peritoneal pressure, was congested by a pressure which, undoubtedly, was equal to the low arterial pressure; and the circulation in the lower portion of the body was almost *nil,* as indicated by the extreme edema.

The absence of congestion of the superior cava, its presence in the inferior cava as a result of ascites, and the existence of the backflow which had no appreciable effect on the circulation in the superior cava, suggest that, in tricuspid insufficiency, the activity of the right ventricle may be considered under two heads: 1. The effort expended in pumping blood into the pulmonic artery. This factor is deficient as compared with the normal and is the immediate cause of venous congestion. 2. The effort expended in propelling the regurgitant blood into the auricle and veins. The production of this backflow acts mechanically by increasing, first, the auricular and, later, the auricular and venous pressure so as to build, at the tricuspid opening, a wall, so to speak, of blood under high pressure, which, in part, takes the place of the tricuspid valve. This may be regarded as a utilization of a portion of the congested blood.

The large systolic venous pulsations, the absence of radial pulse and the forcible rapid heart action show that only a small amount of blood was delivered to the arterial system and that a large portion of the blood was returned during each systole to the veins and right auricle in which the pressure was low. Hypertrophy and dilatation of the right ventricle and, as MacKenzie[1] suggests, the right auricle, were the only active compensatory factors. They were unequal to the task, leaving the heart to exhaust itself by rapid action which moved a minimum amount of blood and delivered a minimum amount of nutrition to the heart.

When the abdominal pressure was relieved, congestion in the venæ cavæ and distention of the right auricle were immediately re-established, thus furnishing a pressure of a degree sufficient to diminish the tricuspid regurgitation and to raise the pressure in the right ventricle to such an extent that a larger amount of blood was delivered to the pulmonic circulation and left heart. The dyspnea was then diminished; the heart's action was reduced in rate and regular, the radial pulse returned, and the large systolic venous pulsation was diminished in size.

It should be noted that, after the abdominal pressure was removed, the right ventricle had sufficient strength to perform the required work; it must consequently be inferred that the ventricle had the power necessary to pump the usual amount of blood while the inferior cava was ob-

structed. But during this period the low pressure in the auricle and superior cava offered the path of least resistance for the greater portion of the small amount of blood delivered by the superior cava. Venous congestion is the resistance on which the hypertrophied right ventricle must depend for support.

The right ventricle would thus appear to depend on venous congestion of a degree sufficient to maintain a pressure which, with the addition of that contributed by the backflow, may offer resistance enough to act as a substitute for the insufficient tricuspid valve. Now, since the venous congestion depends on, is caused by and represents the arterial pressure, minus friction and flow, or, in other words, the activity of the left ventricle, it follows that in tricuspid insufficiency the left ventricle bears the brunt of that part of compensation which is played by venous congestion.

As the flow of blood to the left heart is diminished, the work of the left ventricle is progressively lessened. The left ventricle may, therefore, remain of normal thickness or it may become atrophied. The large normal excess of arterial pressure over that in the capillaries renders it possible that even with diminished activity of the left ventricle and a lowered arterial pressure there may yet be a rise in venous pressure, a portion of which is utilized for compensation, the balance being consumed through friction and flow of blood.

The factors in tricuspid compensation usually considered[8] are: 1. Hypertrophy and dilatation of the right auricle. 2. Venous congestion. 3. Hypertrophy and dilatation of the right ventricle. 4. Aspirating effect of the thorax with inspiration.

1. As the tricuspid valve begins to leak, the right auricle is distended and hypertrophies to some extent. But when the lesion becomes extensive, as in this case, the auricle plays a minor rôle, if, indeed, it plays an active part at all; it is, however, probably inactive, as shown in one case by Franke,[2] and especially noted by MacKenzie.[1] If inactive, it virtually becomes a portion of the venous system.

2. The venous congestion, compensatory venous congestion, may be considered under two heads: (a) the constant or accumulated (diastolic) pressure from the arterial system, and (b) the pressure added by the backflow from the right ventricle.

These pressures serve two purposes: First, the constant pressure maintains the venous distention, so that, with the backflow of blood under a relatively high pressure, the venous pressure rapidly rises to a

8. Sahli: Diagnostic Methods. Transl. by Kinnicutt and Potter from fourth German edition, 1905, 335.

maximum, after which the greatest possible amount of blood enters the pulmonary artery. Second, the maximum venous pressure aids in filling the ventricle during diastole. Through its elasticity, experimentally shown by Salamon,[9] the liver in the pulsating venous system serves the same purpose as an air chamber in a force pump; it also supplies storage for blood.

3. Hypertrophy of the right ventricle is required to develop the added systolic venous pressure and to perform the extra amount of work due to the size of the ventricular cavity when there is dilatation. Here, to propel the usual amount of blood, the muscle or power moves over a shorter distance, consequently must do more work. A certain amount of dilatation is necessary for compensation, supplying space for the blood which is returned in systole to the auricle and veins. Dilatation beyond this point furnishes storage room for blood.

4. Aspiration with inspiration is a minor factor.

5. To this test should be added the work of the left ventricle which is represented by the venous congestion.

Of these factors, the venous congestion and the hypertrophy and dilatation of the right ventricle are the most important. Impairment of either factor leads to heart failure, each in its own way.

In the one event the heart, deprived of the venous pressure against which it works, exhausts itself by increased activity and an ever-diminishing amount of nutrition until it can no longer contract. At autopsy the heart and cavæ should contain little blood. In the other event, if the right ventricle fail, the right heart and veins are so distended with blood that the weakened heart muscle is unable to contract. At autopsy the right heart and veins are distended with blood, while the left heart contains little or no blood.

CONCLUSIONS.

In tricuspid insufficiency the left ventricle and venous congestion play the same rôle as do the right ventricle and the pulmonary congestion in mitral insufficiency. In the latter condition the congestion of the auricle and the pulmonary circulation furnishes the resistance against which the hypertrophied left ventricle works; in the former the congestion of the right auricle and cavæ favor the action of the hypertrophied right ventricle.

In the one, the right ventricle causes the pulmonic congestion; in the other, the left ventricle, the caval congestion. This is as it should be, for in each circulation the ventricle is best fitted to develop the neces-

9. Salamon: Lancet, London, 1907, l. 4.

sary venous pressure. The remaining factors have a temporary or minor influence on the compensation of insufficient mitral and tricuspid valves.

SUMMARY.

Mitral Insufficiency.

L. auricle. { Dilatation. / Hypertrophy.

L. ventricle. { Dilatation. / Hypertrophy.

Pulmonic veins, congestion.

Pulmonic artery, congestion.

R. ventricle, hypertrophy.

Increased resistance.

Tricuspid Insufficiency.

R. auricle. { Dilatation. / Hypertrophy.

R. ventricle. { Dilatation. / Hypertrophy.

Systemic veins, congestion.

Systemic arteries, low pressure.

L. ventricle, normal or atrophied.

The normal margin of arterial pressure above that in the capillaries makes possible a decrease in the arterial pressure (l. ventricle either normal or atrophied) in the presence of a rise in the capillary pressure, until the venous, capillary and arterial pressures are relatively the same, or until the flow of blood through the capillaries is insufficient to sustain life.