The Pulmonary Vessels in Incipient Left Ventricular Decompensation

Radiologic Observations

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The appearance of clinical symptoms in a patient with an overloaded or weakened left ventricle generally provides the earliest evidence that the compensating cardiac mechanisms are no longer able to cope with normal physiologic demands. If, however, clinical signs and symptoms appear only after a certain level or duration of left ventricular decompensation, then the asymptomatic, apparently compensated patients must include some in what might be termed "incipient" left ventricular decompensation. It might be important to recognize such patients as an intermediate group; they may readily regress into the symptomatic group, or alternatively the symptoms might be anticipated or even prevented by appropriate therapy.

The radiologist may contribute in this area by detecting objective evidence of left ventricular decompensation before clinical symptoms have appeared. Characteristic changes in the pulmonary vascular pattern are seen on the plain chest roentgenogram.

Radiologic Findings

Normal

Before consideration of these changes, it is helpful to review some of the normal radiologic features of the pulmonary vasculature (fig. 1). The pulmonary arteries diverge from the hilus well above the point at which the veins converge on the left atrium, at least 3 or 4 cm. in the adult. Thus, in the mid and lower zones of the lungs the direction of a vessel helps identify it as artery or vein. In the upper zones, the arteries and veins run in the same direction but the vein lies lateral to its corresponding artery. The main upper-lobe veins may be seen to cross the main pulmonary artery at an angle inconsistent with entry into it. In the normal person the upper-lobe vessels, both veins and arteries, are substantially smaller than those in the lower lobe. This normal disparity is due to the conical shape of the lung with the lower-lobe vessels subserving a greater volume of tissue.

This normal vascular pattern is encountered in some patients with left ventricular disease, but almost invariably without symptoms of left ventricular decompensation (fig. 2). They are thus well compensated by both clinical and radiologic criteria.

Pattern of Overt Left Ventricular Decompensation

Practically all our patients with clinical evidence of left ventricular decompensation observed in the last 5 years have shown, with greater or lesser clarity, a characteristic change in the pulmonary vascular pattern (fig. 3). In the upper zones the pulmonary veins undergo marked dilatation and the pulmonary arteries also dilate slightly. In the lower zones, paradoxically, the veins and arteries do not dilate and in many cases show clear-cut evidence of narrowing. They may be difficult to identify, contrasting markedly with their normal ready visibility and good caliber. An absolute change of caliber of single vessels is usually clearly detectable, but the disparity between the dilated upper vessels and relatively narrowed lower-zone vessels is more striking. It should not be forgotten that normally the upper-zone vessels
are actually smaller than those in the lower zones.

In the more severely decompensated cases, vascular detail may be partly obscured by pulmonary edema. This may be predominantly interstitial, causing a diffuse faint haziness at the lung bases occasionally with the appearance of septal lines of Kerley, or may be true alveolar edema with scattered irregular areas of consolidation progressing to the classic "butterfly" pattern of consolidation. Pleural effusions may occur with accentuation of the interlobar fissures. The cardiac silhouette may show a rapid enlargement. Such patients are invariably symptomatic, however, and the diagnosis of left ventricular failure is seldom in doubt (fig. 4).

**Pattern of Incipient Left Ventricular Failure**

Many patients without symptoms of left ventricular decompensation, though frequently with a history, previous clinical documentation, or radiologic evidence of left ventricular disease, have the typical vascular pattern of left ventricular decompensation. The roentgen examination shows dilated upper-lobe vessels and narrowed lower-lobe vessels.

Are these changes due to "incipient" left ventricular decompensation? Several findings obtained favor this possibility: 1. A prolonged circulation time has been demonstrated in some cases, a few returning to normal values after treatment. 2. Rapid and significant weight loss in response to diuretic therapy has occurred in a number of such symptomless patients. 3. In a few patients effectively treated by diuretics, the pulmonary vascular pattern has returned to normal. 4. The concept has sometimes been tested in less fortunate manner. The typical observations on pre-operative films have been ignored on occasion, and two such cases developed pulmonary edema during surgery. Occasionally, patients presenting with pneumonia or pulmonary embolism may have an unsuspected component of left ventricular failure, possibly precipitated by the infection or embolism. In these cases the radiologist may observe the dilatation of the upper-lobe vessels and constriction of the lower-lobe vessels, in addition to the focal lesion, and is thus able to amplify the diagnosis, leading to better therapy. In one case with lobar pneumonia, the consolidation responded poorly for approximately 10 days until the decompensation was treated, after which resolution was dramatic (fig. 5).
PULMONARY VESSELS IN DECOMPENSATION

The vascular pattern of left ventricular decompensation, whether clinically overt or incipient, is sometimes a transient phenomenon, returning rapidly to normal with effective treatment. More commonly, however, the vascular changes are persistent. It is possible that such individuals may be incapable of regaining complete physiologic compensation and remain in a chronic state of "incipient" decompensation.

The widely held concept that the venous overdistention in left ventricular failure is a passive manifestation of damming back of blood, or elevation of the pulmonary venous pressure, fails to account for the decreased caliber of the vessel in the lower zones. In 1956, the writer proposed an alternative hypothesis to explain a similar pulmonary vascular pattern in patients with mitral stenosis and suggested its applicability in left ventricular failure (fig. 6):

1. In the erect position of man, since the veins of the upper and lower lobes communicate freely via the left atrium, the pressure in the lower-lobe veins is normally substantially greater than in the upper-lobe veins because of the difference of hydrostatic level between them.

2. A significant rise in pulmonary venous pressure, almost invariable in mitral stenosis, is probably also an early effect of left ventricular decompensation.

3. There is reasonable evidence to postulate a localized segmental reflex, initiated by a rise of pulmonary venous pressure above a critical level, that results in constriction of the veins and arteries in that particular lung segment.

4. In left ventricular failure the rising venous pressure reaches this critical level in the lower zones long before it does in the upper zones. Vasoconstriction thus occurs initially in the lower zones, reducing the circulating volume through them.

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5. Cardiac output is maintained by diversion of blood through the upper zones. Thus, the vascular dilatation in the upper lobes represents increased blood flow and is not due to “damming back” or “congestion” of the blood.

It is tempting to speculate on the mechanism of orthopnea on the basis of this hypothesis. In the upright position the vasoconstrictive phenomena are confined to the lower zones and cardiac output is maintained by increased flow through the upper zones. In the horizontal position, the pressures in the veins of the upper and lower zones are equalized at intermediate values. In the less severe cases, the pressures might still be generally below the critical level and the horizontal position would thus be tolerated. In more severe cases the critical level would be widely exceeded and generalized reflex vasoconstriction would occur with rapid restriction of blood flow through the lungs. The resulting oxygen lack would in turn stimulate respiratory efforts.

Much of the evidence in support of the vasoconstrictive reflex hypothesis is derived from prior studies on cases of mitral stenosis in which the elevation of pulmonary venous pressure is slowly progressive and catheterization data were available. From these studies it would appear that the critical venous pressure at which vasoconstriction occurs is of the order of 10 to 15 mm. of mercury (normal, 0 to 5 mm.)

Angiograms (fig. 7) in a 7-year-old boy in congestive cardiac failure due to primary myocardial disease, with elevated wedge pressure at 25 mm. of mercury, provide convincing support for the hypothesis. In the early film (fig. 7, left) there is slight dilatation of the upper-zone arteries and marked constriction of the lower-zone arteries. In the later film (fig. 7, right) there is gross dilatation of the upper-zone veins whereas the lower-zone veins are barely visualized and are undoubtedly constricted.

Flow through the lower zone is greatly retarded while the relative rate of flow through the upper zone is greatly increased. Thus, the contrast medium outlines the great upper-lobe veins and passes into the left atrium via the superior orifice, long before it has even negotiated the constricted lower-zone arteries. This supports the concept that the vascular dilatation in the upper zone is due to diversion of blood from the lower zone,
PULMONARY VESSELS IN DECOMPENSATION

probably with locally increased flow, and not due directly to a damming back of blood.

An exception to the typical vessel pattern in left ventricular failure occurs in patients with severe pulmonary emphysema or chronic bronchitis (fig. 8). In these cases both the lower-lobe vessels and the upper-lobe vessels appear to become dilated, simulating the generalized dilatation of pulmonary vessels seen in cases of left-to-right shunt with increased flow through the lungs. The explanation is uncertain and may be related to shunting of blood through overdeveloped bronchial collateral vessels or failure of the postulated reflex vasoconstriction mechanism.

The pattern of vascular dilatation in the upper zones and constriction in the lower zones characterizes other causes of pulmonary venous hypertension, e.g., mitral stenosis, left atrial tumors, and some cases of pericardial effusion.4, 14–18

It may also sometimes be seen in cases of basal bronchiectasis or basal emphysema without pulmonary venous hypertension in which pulmonary circulation in the lower lobes is reduced by the local parenchymal disease, and for this reason blood becomes diverted through the upper lobe vessels.4

Conclusion

An awareness of the typical pulmonary vascular pattern associated with left ventricular decompensation may facilitate early diagnosis of this condition, not infrequently before symptoms have appeared. In such cases the symptoms may be anticipated or prevented by appropriate treatment.

Summary

Dilatation of the pulmonary vessels of the upper zones and constriction of the vessels of the lower zones characterizes left ventricular decompensation.

These changes may precede the onset of clinical symptoms providing a useful warning sign of impending danger.

A hypothesis is suggested to account for the non-uniform vessel pattern.

An exception is noted in patients with severe emphysema or bronchitis in whom generalized dilatation may occur.

References

10. Short, D. S.: Radiology of the lung in severe

Figure 8
Severe emphysema without failure (left) and complicated by left ventricular failure (right). The lower-zone vessels have dilated as well as the upper-zone vessels, representing an exception to the general rule.
The Idea of Chemical Transmission of Nervous Impulse

Now I have to turn to the best known of my scientific achievements, the establishment in 1921 of the chemical theory of the transmission of the nervous impulse. Until 1921 it was generally assumed that transmission was due to the direct spreading of the electrical wave accompanying the propagated nervous impulse from the nerve terminal to the effector organ. Since the character of that potential is everywhere the same, such an assumption would not explain the well-known fact that the stimulation of certain nerves increases the function of one organ and decreases the function of another. A different mode of transmission had, therefore, to be considered.

As far back as 1903, I discussed with Walter M. Fletcher from Cambridge, England, then an associate in Marburg, the fact that certain drugs mimic the augmentary as well as the inhibitory effects of the stimulation of sympathetic and/or parasympathetic nerves on their effector organs. During this discussion, the idea occurred to me that the terminals of those nerves might contain chemicals, that stimulation might liberate them from the nerve terminals, and that these chemicals might in turn transmit the nervous impulse to their respective effector organs. At that time I did not see a way to prove the correctness of this hunch, and it entirely slipped my conscious memory until it emerged again in 1920.

The night before Easter Sunday of that year I awoke, turned on the light, and jotted down a few notes on a tiny slip of thin paper. Then I fell asleep again. It occurred to me at six o'clock in the morning that during the night I had written down something most important, but I was unable to decipher the scrawl. The next night at three o'clock, the idea returned. It was the design of an experiment to determine whether or not the hypothesis of chemical transmission that I had uttered seventeen years ago was correct. I got up immediately, went to the laboratory, and performed a simple experiment on a frog heart according to the nocturnal design. I have to describe briefly this experiment since its results became the foundation of the theory of chemical transmission of the nervous impulse.

The hearts of two frogs were isolated, the first with its nerves, the second without. Both hearts were attached to Straub cannulas filled with a little Ringer solution. The vagus nerve of the first heart was stimulated for a few minutes. Then the Ringer solution that had been in the first heart during the stimulation of the vagus was transferred to the second heart. It slowed and its beats diminished just as if its vagus had been stimulated. Similarly, when the accelerated nerve was stimulated and the Ringer from this period transferred, the second heart speeded up and its beats increased. These results unequivocally proved that the nerves do not influence the heart directly but liberate from their terminals specific chemical substances which, in their turn, cause the well-known modifications of the function of the heart characteristic of the stimulation of its nerves.—OTTLO LOEWI. An Autobiographic Sketch. Perspectives in Biology and Medicine 4:16, 1960.