Regulation of Pulmonary Capillary Blood Volume by Pulmonary Arterial and Left Atrial Pressures

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ABSTRACT

We used single-breath CO diffusing capacity to study the effect of changes in pulmonary arterial and left atrial pressures on pulmonary capillary blood volume in 15 supine dogs whose lungs were perfused with a nonpulsatile pump. Effect of pulmonary arterial pressure: When left atrial pressure measured relative to the bottom of the lungs ≤ alveolar pressure, increasing pulmonary arterial pressure increased diffusing capacity markedly (mean, 0.71 ml/min x mm Hg per mm Hg change in pulmonary arterial pressure). When left atrial pressure > alveolar pressure plus the height of the lungs, increasing pulmonary arterial pressure had less effect on diffusing capacity, although blood flow was increased through a wide range (mean, 0.22 ml/min x mm Hg CO per mm Hg increase in pulmonary arterial pressure). Effect of left atrial pressure: At zero flow, increasing left atrial pressure increased diffusing capacity markedly; when pulmonary arterial pressure was high, increasing left atrial pressure had no significant effect on diffusing capacity. Increasing bronchial arterial pressure from 0 to 150 mm Hg or inhaling 10.5% CO2 had no effect on diffusing capacity. Injection into the pulmonary artery of glass microspheres decreased diffusing capacity only when left atrial pressure was low; their effect was exaggerated when pulmonary arterial pressure was high in the control state.

ADDITIONAL KEY WORDS
pulmonary intravascular pressures microemboli dogs single-breath CO diffusing capacity pulmonary capillary blood volume
circulation was isolated and perfused so that we could control pulmonary arterial and left atrial pressures separately and thereby examine how these two pressures interact to determine the volume of blood in pulmonary capillaries.

**Materials and Methods**

We anesthetized 15 mongrel dogs (20-25 kg) with sodium pentobarbital (30 mg/kg iv). We performed tracheostomies and ventilated the dogs with room air, using a Harvard respirator at a constant tidal volume (500-600 ml). We measured tracheal pressure via a catheter which was connected to a strain gauge (Statham PM 131); when there was no airflow in this closed system (during measurement of diffusing capacity), pressure at the trachea was assumed to equal alveolar pressure. End-expiratory tracheal pressure was maintained at 2 to 3 mm Hg to prevent collapse of the lungs when the chest was open. In the supine dog, we opened the chest widely and placed purse-string sutures in the left atrial appendage and the right ventricular outflow tract. An umbilical tape was placed around the pulmonary artery, heparin sodium was injected (3 mg/kg iv) and perfusion cannulas were placed in the main pulmonary artery and in the left atrium. The main pulmonary artery was never occluded for more than 30 seconds during cannulation. Catheters were placed inside these cannulas so that the catheters protruded 0.5 cm beyond the tip of the perfusion cannulas; the other end of each catheter was connected to a strain gauge (Statham P23Db) to record intravascular pressure. These pressures were referred to the deepest point in the posterior thorax with the animal in the supine position (this point, the lowermost portion of the lungs, is referred to as the bottom of the lung in this paper). The height of the lungs at full lung inflation was also measured from this point. Figure 1 illustrates the relation between intravascular pressures and al-

![Diagram illustrating influence of left atrial pressure (P_{LA}) and alveolar pressure (P_{ALV}) on the volume of pulmonary capillaries in different portions of lungs during zero pulmonary blood flow, i.e., when P_{LA} = pulmonary arterial pressure (P_{PA}). Stippled areas represent blood vessels; those on left represent arterial circulation, those in center pulmonary capillaries, and those on right pulmonary venous circulation. Dark areas represent vessels on arterial side with critical opening pressures. Zero levels for intravascular pressures are at bottom of lungs, and heights of columns indicate intravascular pressure at the bottom of lungs. In these experiments bottom and top of the lungs are defined as the lowest and highest points of the lungs in the thorax with the dogs in the supine position. Scale of pressures is located at left of diagram. Three rounded areas represent alveoli at different levels of lungs (P_{ALV} = 10 mm Hg); these are connected to tubes representing conducting airways. Pulmonary capillaries are shown under two conditions: (A) When P_{LA} at bottom of lungs is approximately equal to P_{ALV}, alveolar capillaries are shown compressed at all levels of lungs. (B) When P_{LA} at top of lungs is approximately equal to P_{ALV}, almost all alveolar capillaries are distended.](http://circres.ahajournals.org/lookup/doi/10.1161/01.RES.22.1.1)
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FIGURE 2
Arrangement of apparatus for perfusing lungs in situ. Arrows with solid shafts indicate direction of blood flow. Those with dashed shafts indicate that the horizontally placed tubing leading from the left atrium can be moved vertically to change left atrial (LA) pressure. PA = pulmonary artery.

Veolar pressure at different levels of the lungs.

During perfusion, blood flowed from the left atrium through wide-bore tubing into a plastic reservoir, thence via a roller type of pump, debub-bler and depulsating unit (to achieve a constant, nonpulsatile blood flow through the lungs), and a heat exchanger (which kept the blood temperature at 35°C) into the pulmonary artery (Fig. 2). Eight hundred ml of blood from a donor dog and 400 ml of 0.9% sodium chloride solution were re-quired to prime the system. We regulated the pressure in the left atrium by adjusting the height of the outflow tubing, which was placed horizon-tally; we regulated pulmonary arterial pressure by changing flow. We calibrated the perfusion pump at the end of each experiment using a stopwatch and allowing the blood leaving the pump to enter a calibrated cylinder. In two dogs, plasma hemoglobin levels measured at the beginning of the perfusion were 9 and 7 mg/100 ml of plasma; at the end of 3 hours of perfusion, the levels were 24 and 37 mg/100 ml of plasma. Blood hemoglobin concentration varied from 7 to 18.5 g/100 ml. All determinations of diffusing capacity were corrected to a hemoglobin concentration of 15 g/100 ml.

We measured diffusing capacity by a modified single-breath method (5) as follows: After adjusting pulmonary arterial and left atrial pressures, we stopped ventilation at end-expiration and inflated the lungs rapidly from a calibrated syringe filled with 600 to 800 ml of a gas mixture which contained 0.5% neon and 0.3% CO. (This inflation volume was chosen to ensure that an adequate sample was obtained.) Ten seconds later, an electrically timed solenoid opened, dead space gas was evacuated into a trap and an alveolar sample was obtained and analyzed in a gas chromatograph. Alveolar volume was calculated using the neon concentration in the expired gas sample. During the measurement of diffusing capacity, we recorded pulmonary arterial, left atrial and alveolar pressures on a multitrae recorder (Electronics for Medicine), using the pressures measured 5 seconds after the start of lung inflation as the mean pressure during the 10-second period. Pulmonary vascular resistance calculated at 2 liters/minute of blood flow ranged from 4.9-8.2 mm Hg per liter/minute (mean, 6.3) when left atrial pressure measured relative to the bottom of the lungs equaled alveolar pressure, and ranged from 2.7 to 7.1 mm Hg per liter/minute (mean, 3.7) when left atrial pressure relative to the top of the lungs equaled alveolar pressure. Between determinations of diffusing capacity we perfused the lungs at a flow of 1 liter/minute, and left atrial pressure was less than 5 mm Hg. We waited at least 3 minutes between measurements of diffusing capacity so that no significant back pressures for CO existed during our experiments. We held alveolar pressure as constant as possible in each experiment whenever we measured diffusing capacity (range, 8 to 12 mm Hg).

To examine the relative effects of pulmonary arterial and left atrial pressure on diffusing capacity we used the following procedure: We made a series of measurements of diffusing capacity with either (1) left atrial pressure held constant and pulmonary arterial pressure increased by increasing blood flow, or (2) pulmonary arterial pressure held constant and left atrial pressure increased by adjusting the level of the outflow tubing (decreasing flow as left atrial pressure was increased). Measurements of diffusing capacity were thus obtained for increments of pulmonary arterial pressure at constant left atrial pressure and for increments of left atrial pressure at both zero flow (when there should be no additional contribution of inflow pressure to the size of the pulmonary capillary bed) and when pulmonary arterial pressure was high (and therefore the influence of left atrial pressure on pulmonary capillary blood volume should be modified by inflow pressure). In each experiment we progressed from low to high intravascular pressures. When pressures were changed we waited for 2 minutes before measuring diffusing
capacity, to allow for intravascular adjustments. Our criteria for stability of the preparations were: (1) diffusing capacity at similar intravascular pressures was unchanged with time; (2) pulmonary arterial pressure at given alveolar pressure, left atrial pressure, and blood flow was unchanged; (3) alveolar pressure during the measurement of diffusing capacity was unchanged. Using these criteria, we were able to obtain reproducible data for periods of 2 to 4 hours.

In 3 dogs, we obtained control studies and then injected a sufficient number of glass microspheres (100 μm diam) (B.F. Drakenfeld & Co., New York) into the pulmonary artery to increase pulmonary arterial pressure by 4 to 9 mm Hg when there was a blood flow of 1.5 liters/min and a control pulmonary arterial pressure of 24 mm Hg. In 2 dogs, we tested the effect of increasing bronchial arterial pressure on diffusing capacity by isolating and perfusing the aortic segment giving rise to the bronchial arteries (via the intercostal arteries). At the end of these experiments, we injected radiopaque dye and confirmed radiologically that the bronchial arteries were included in the isolated segment. These experiments were conducted when left atrial pressure was zero and when there was zero pulmonary blood flow.

We obtained samples from the catheter in the pulmonary artery and measured O₂ and CO₂ tensions with electrodes (6) and pH with a micro-radiometer-capillary electrode. Except when CO₂ was added to the inspired gas, pH was 7.65 to 7.75 units; Pco₂ < 10 mm Hg; and Po₂ was 110 to 130 mm Hg. We calculated the slopes of change in diffusing capacity/change in intravascular pressures in the linear portions of the curves using the method of least squares.

Results

A. EFFECT OF PULMONARY ARTERIAL PRESSURE ON DIFFUSING CAPACITY

If pulmonary arterial pressure has a significant effect on the size of the pulmonary capillary bed, its effect should be most marked when left atrial pressure is low (i.e., when the capillaries are not already distended) and least when left atrial pressure is high. Therefore, we examined the effect on diffusing capacity of increasing pulmonary arterial pressure (by increasing flow) at different levels of left atrial pressure.

1. Low left atrial pressure.—When left atrial pressure relative to the bottom of the lungs ≤ alveolar pressure (11 dogs), increasing pulmonary arterial pressure increased diffusing capacity.

![Figure 3](http://circres.ahajournals.org/Downloadedfrom)

**FIGURE 3**

Effect of pulmonary arterial pressure (P_PA) on single-breath CO diffusing capacity (D_LCO) under two conditions: (A) When left atrial pressure (P_LA) was low, i.e., when P_LA ≤ P_ALV at the bottom of the lungs. (B) When left atrial pressure was high, i.e., when P_LA ≥ P_ALV at the top of the lungs. Each line represents an experiment in a single dog and each point represents a single observation.
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capacity by a mean of 0.71 ml/min × mm Hg CO per mm Hg change in pulmonary arterial pressure (range, 0.32 to 1.12) (Fig. 3A).

2. High left atrial pressure.—When left atrial pressure relative to the bottom of the lungs ≥ alveolar pressure plus the height of the lungs (7 dogs), increasing pulmonary arterial pressure increased diffusing capacity only 0.22 ml/min × mm Hg CO per mm Hg change in pulmonary arterial pressure (range, 0.01 to 0.40) (Fig. 3B). In the 7 dogs in which the effect of changing pulmonary arterial pressure was studied under the two conditions, the increase in diffusing capacity was greater in every animal when left atrial pressure was low than when it was high.

In 5 dogs, we compared the effect of changing pulmonary arterial pressure on diffusing capacity at three or more levels of left atrial pressures. The increase in diffusing capacity with increasing pulmonary arterial pressure was greatest when left atrial pressure was low; when left atrial pressure was maintained at higher levels, diffusing capacity was higher at every level of pulmonary arterial pressure, but the change in diffusing capacity with changing pulmonary arterial pressure was less. Finally, when left atrial pressure relative to the bottom of the lungs ≥ alveolar pressure plus the height of the lungs, pulmonary arterial pressure had no significant effect on diffusing capacity (Fig. 4).

B. EFFECT OF LEFT ATRIAL PRESSURE ON DIFFUSING CAPACITY

We could not study the effect of left atrial pressure alone, but we compared the effect of left atrial pressure on diffusing capacity in 3 dogs under two different conditions: (1) during zero flow, when only a static distending force existed at the capillary level, and (2) when pulmonary arterial pressure was high (and should have a marked influence on pulmonary capillary blood volume).

1. Zero blood flow.—When there was no flow through the lungs, diffusing capacity was low when left atrial pressure was low; increasing left atrial pressure increased diffusing capacity in each dog (0.72, 0.3, and 0.24 ml/min × mm Hg CO per mm Hg increase in left atrial pressure) (Fig. 5A).

2. High pulmonary arterial pressure.—When pulmonary arterial pressure exceeded alveolar pressure plus the height of the lungs (by 10, 18 and 34 mm Hg) diffusing capacity was as high or higher than diffusing capacity measured during zero flow when left atrial pressure ≥ alveolar pressure plus the height of the lungs; increasing left atrial pressure under these conditions had no significant effect on diffusing capacity (0.02, 0.03 and −0.01 ml/min × mm Hg CO per mm Hg change in left atrial pressure) (Fig. 5B).

C. EFFECT OF INJECTION OF GLASS MICROSPHERES INTO THE PULMONARY ARTERIAL ON DIFFUSING CAPACITY

If most of the pulmonary arterial bed is partially or totally occluded by emboli, increasing pulmonary arterial pressure should have a relatively small effect on the size of the pulmonary capillary bed, but the effect of
Effect of left atrial pressure ($P_{LA}$) on single-breath CO diffusing capacity ($D_{LCO}$) in 3 dogs (1,2,3) under two conditions: (A) Zero flow—pulmonary arterial pressure ($P_{PA}$) = $P_{LA}$. (B) $P_{PA}$ high and constant. Values of $P_{PA}$ are in parentheses.

A. Constant $P_{LA} = 17$ mm Hg

B. Constant $P_{PA} = 32$ mm Hg

Effect of microemboli in pulmonary artery on single-breath CO diffusing capacity ($D_{LCO}$) in 1 dog under two conditions: (A) Increasing pulmonary arterial pressure ($P_{PA}$) when left atrial pressure ($P_{LA}$) was held constant = 17 mm Hg. (B) Increasing $P_{LA}$ when $P_{PA}$ was held constant = 32 mm Hg. Alveolar pressure during measurement of $D_{LCO}$ = 10 mm Hg; height of lungs = 15 cm.

Increasing left atrial pressure should be unaffected. Furthermore, the effect of embolization on diffusing capacity should depend on the level of pulmonary arterial pressure in the control state. Therefore, we studied the effect of pulmonary emboli on diffusing capacity in 3 dogs under the following conditions.

1. Constant left atrial pressure; increasing pulmonary arterial pressure (2 dogs).—When left atrial pressure was held constant (zero mm Hg in one experiment, 17 mm Hg in the
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other), increasing pulmonary arterial pressure increased diffusing capacity in the control state; after embolization, increasing pulmonary arterial pressure had no effect on diffusing capacity (Fig. 6A).

2. Constant high pulmonary arterial pressure; increasing left atrial pressure (1 dog).

-When pulmonary arterial pressure was high, increasing left atrial pressure had no significant effect on the diffusing capacity in the control state. After embolization, at low left atrial pressure, diffusing capacity was decreased compared to the control state, but increasing left atrial pressure still increased diffusing capacity (Fig. 6B).

D. EFFECT OF BRONCHIAL ARTERIAL PRESSURE ON DIFFUSING CAPACITY

To determine whether our experiments were affected significantly by the absence of bronchial arterial pressure, we isolated and controlled the blood flow in the bronchial artery separately in 2 dogs and studied the effect of increasing bronchial arterial pressure on diffusing capacity when there was zero pulmonary blood flow and when left atrial pressure was zero (i.e., when the bronchial blood flow should have a maximum effect on the size of the pulmonary capillary bed). Under these conditions, increasing bronchial arterial pressure from 0 to 150 mm Hg did not change diffusing capacity significantly.

E. EFFECT ON DIFFUSING CAPACITY OF VENTILATING THE LUNGS WITH CO₂

Since we perfused only the pulmonary circulation in most of our experiments, the blood perfusing the pulmonary circulation was hypoxemic and alkalotic (see Methods). Therefore, we studied the effect (on diffusing capacity) of ventilating the lungs of one dog with 10.5% CO₂. Although the Pco₂ of pulmonary arterial blood increased from 8 to 70 mm Hg during ventilation with CO₂, neither the slope nor the absolute values for diffusing capacity plotted against change in pulmonary arterial pressure varied significantly from values obtained when the lungs were ventilated with ambient air.

Discussion

These studies suggest that pulmonary arterial pressure is an important determinant of the volume of blood contained in pulmonary capillaries under certain conditions. When left atrial pressure was low relative to alveolar pressure, diffusing capacity was small when pulmonary arterial pressure was low and increased markedly with increasing pulmonary arterial pressure, presumably because inflow pressure was the sole force distending capillaries at every level of the lungs. When left atrial pressure was maintained at higher levels, pulmonary arterial pressure had less effect, presumably because capillaries at the bottom of the lungs were already distended by the outflow pressure. When left atrial pressure was very high, diffusing capacity was maximal, and increasing pulmonary arterial pressure had no effect, presumably because capillaries at all levels of the lungs were fully distended. Thus the present study is at variance with that of Lawson et al. (4) who showed that inflow pressure in the same range as in the present study had an insignificant effect on diffusing capacity in isolated lungs of cats. We believe that the high pulmonary vascular resistance encountered by these authors may account for the absence of effect of inflow pressure on pulmonary capillary blood volume; their findings indicate that their animals had a high arteriolar resistance to blood flow. Under those circumstances, inflow pressure was probably largely dissipated before reaching the pulmonary capillaries. Our present concept of the influence of pulmonary arterial pressure on the pulmonary capillary bed is shown diagrammatically in Figure 1. Increasing inflow pressure above alveolar pressure distends arterial vessels first at the bottom, then middle, then top of the lungs; these increasing pressures may be required to open vessels that previously had no blood flow or to distend vessels that already had some blood flow (or both).

Our studies also indicate that left atrial pressure is also an important determinant of pulmonary capillary blood volume, but only under certain circumstances: When pulmonary arterial pressure was high, increasing left atrial

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pressure had an insignificant effect on pulmonary capillary blood volume, since the capillaries were already distended by the high inflow pressure. To study the effect of outflow pressure alone on pulmonary capillary blood volume, we accepted previous measurements indicating that pulmonary veins had little impedance to blood flow (7); to the extent that pulmonary venous impedance is small, the effect of left atrial pressure on pulmonary capillaries at zero flow should reflect its influence on the pulmonary capillaries during flow. In our experiments, during zero flow, when pulmonary arterial pressure had no additional influence on the capillary bed, increasing left atrial pressure increased the pulmonary capillary blood volume markedly. Figure 7 shows diagrammatically our concept of the influence of outflow pressure on the pulmonary capillary bed: The postcapillary vessels have little resistance to flow (7) and are easily distensible. Thus, any increase in left atrial pressure is transmitted with little change to the capillary level, and a smaller increase in left atrial pressure (compared to pulmonary arterial pressure) is required to distend capillaries at various levels of the lungs. It should be noted in Figures 1 and 7 that capillaries in our model may be recruited from the venous side, even though arterial pressure is not high enough to produce filling from the arterial side (or when no blood flow is occurring). This concept may be especially important when emboli lodge in the pulmonary arteries. When inflow pressure is decreased to a portion of the pulmonary capillary bed, left atrial pressure may be the sole distending force keeping capillaries in this area open. Thus, in our experiments, when the pulmonary arterial pressure was high and left atrial pressure was low in the control state, injection of glass beads into the pulmonary artery decreased diffusing capacity, but injection of these arterial emboli had an insignificant effect on diffusing ca-

FIGURE 7
Influence of pulmonary arterial pressure (P_{PA}) and alveolar pressure (P_{ALV}) on the size of pulmonary capillaries in different portions of lungs when left atrial pressure (P_{LA}) at bottom of lungs is approximately equal to P_{ALV} (i.e., when P_{PA} is the sole intraoesuhtr force distending pulmonary capillaries). Diagram shows pulmonary capillaries under two conditions: (A) When P_{PA} is approximately equal to P_{ALV}, plus the height of the lungs, many pulmonary capillaries remain undistended. (B) When P_{PA} is much greater than P_{ALV}, plus the height of the lungs, more capillaries distend.
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When left atrial pressure was high in the control state.

In interpreting the present experiments, certain limitations and criticisms must be considered.

I. Since pulmonary capillary blood volume cannot be measured directly, we used the single-breath CO diffusing capacity as an index of the size of the pulmonary capillary bed (including capillaries containing static blood). Several objections may be raised about this measurement: (a) We have not measured the absolute values for membrane diffusing capacity and pulmonary capillary blood volume separately, so we are using the values of diffusing capacity as an index of pulmonary blood volume. (b) We chose the single-breath method of measuring diffusing capacity to minimize the effects of changing blood flow on the measurements (3). Since blood flow and inflow pressure usually change together, it is possible that blood flow rather than pulmonary arterial pressure was the critical factor in changing diffusing capacity. However, the following results suggest strongly that blood flow had no significant influence on the measurement. When left atrial pressure was low, increasing pulmonary arterial flow increased diffusing capacity, but when left atrial pressure was high, increasing blood flow through a wide range had no significant effect on diffusing capacity. If blood flow per se had a significant effect on diffusing capacity, it should do so regardless of left atrial pressure. Further evidence that blood flow per se had no influence on diffusing capacity was obtained after injection of pulmonary emboli: increasing blood flow when much of the pulmonary arterial bed was obstructed and the remainder probably already distended had no significant effect on diffusing capacity, while increasing left atrial pressure still increased diffusing capacity. Therefore, all of our results are compatible with the theory that the single-breath measurement of diffusing capacity is influenced markedly by changes in pulmonary capillary blood volume and uninfluenced by blood flow. (c) Pulmonary capillaries in the bottom, middle, and top of the lungs would be expected to be distended to different degrees, depending on the levels of inflow and outflow pressures. Thus, except at high left atrial or pulmonary arterial pressure, inhomogeneity of diffusing capacity would also be predicted at different levels of the lungs. This further limits the use of diffusing capacity as a quantitative measure of pulmonary capillary blood volume. (d) Diffusing capacity was measured by inflating the lungs with the test gas mixture. Thus the measurements were not made at resting lung volume, and some differences may occur in the relation between alveolar pressure and intravascular pressures at different lung volumes.

II. We isolated the pulmonary circulation and perfused the lungs separately, but we did not perfuse the rest of the animal's body. The blood flow was not pulsatile and there was no reflex activity. The pulmonary vascular resistance in our studies was similar to those reported in dogs whose pulmonary circulation was not interrupted (8, 9), and the diffusing capacity and pulmonary arterial pressure at controlled left atrial and alveolar pressures did not change for several hours in our experiments. The absence of perfusion of the body resulted in respiratory alkalosis, and this could have influenced our results, especially by changing smooth muscle tone in the pulmonary vascular bed. However, increasing CO2 concentration in the inspired gas in 1 dog had no significant influence on either pulmonary arterial pressure or on diffusing capacity. This is not surprising, since it is extremely difficult to obtain changes in pulmonary vascular tone in lungs that are perfused separately. Furthermore, the absence of effect of CO2 on either diffusing capacity or on pulmonary vascular pressures suggests that the effects of CO2 on diffusing capacity reported previously were due to changes in intravascular pressures rather than to the speed of uptake of CO by erythrocytes (10). Thus the reason for using our preparation was its low pulmonary vascular resistance, stability over a long period, and our ability to control many of the variables.
III. Our results could have been influenced by the lack of perfusion of the bronchial circulation in most of our experiments. However, when we perfused the bronchial artery separately, we found that increasing bronchial arterial pressure from 0 to 150 mm Hg had no significant effect on diffusing capacity. Thus, the small blood flow which normally exists in the bronchial circulation does not appear to influence diffusing capacity, but in conditions where bronchial collateral blood flow increases markedly, e.g., after pulmonary artery occlusion, this circulation may be an important determinant of diffusing capacity.

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