STUDIES IN INFARCTION.

III. The Circulation in Experimental Pulmonary Embolism.*

By HOWARD T. KARSNER, M.D., and ALBERT A. GHOREYEB, M.D.

(From the Laboratory of Pathology (Phillips Fund) of the Harvard Medical School, Boston.)

In an earlier paper[1] it was stated that "simple bland embolism of the pulmonary artery produces definite circulatory changes in the lung area supplied" (consisting chiefly of congestion) "provided this area extends along the sharp edge of the lobe and the embolus is of sufficient size, but no evidence of true infarction is to be found. So far as technical limitations permit, occlusion of the bronchial arteries makes no change in the circulatory alterations following simple embolism" of the pulmonary artery. This lack of participation of the bronchial artery was in apparent contradiction of an old experiment of Virchow's[2] in which he found that occlusion of the main trunk of the pulmonary was not followed by destruction of the lobe because the bronchial artery enlarged and supplied sufficient nourishment for the lobe. This experiment has been applied by many writers so as to explain the absence of true infarction following embolism of a smaller branch of the pulmonary artery; *i. e.*, by stating that the nutrition of the area supplied normally by the embolized pulmonary artery is taken over by the bronchial artery. In another paper[3] we have pointed out that in simultaneous injections of the two arterial systems there is almost no mixture of the bloods unless the pressure is zero in one or other of the two systems, and that when embolism of a smaller branch of the pulmonary artery occurs the pressure in the area supplied does not sink to zero because, as was shown in the first paper,[4] a circulation, independent of the bronchial circulation, is maintained, even though such circulation is considerably less than normal.

In order more thoroughly to understand what part the two circulations play in the case of embolism of the pulmonary artery the

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[1] Karsner, H. T., and Ash, J. E., Studies in Infarction. II. Experimental Bland Infarction of the Lung, *Jour. Med. Research,* 1912, xxvii, 205.

[2] Virchow, R., Gesammelte Abhandlungen zur wissenschaftlichen Medicin, Frankfurt a. M., 1856, 295, experiment XX.

[3] Ghoreyeb, A. A., and Karsner, H. T., A Study of the Relation of Pulmonary and Bronchial Circulation, *Jour. Exper. Med.,* 1913, xviii, 500.

[4] Karsner, H. T., and Ash, J. E., *loc. cit.*

following study was carried on in conjunction with the general study of the relationship of the two systems.[5]

Dogs were anesthetized and four turnip radish seeds deposited in the right heart by means of a glass cannula inserted through the right jugular vein. The thorax was opened under artificial respiration and the injection of the two arterial systems carried out as in the general study,[6] the same animals being used.

EXPERIMENTS.

Series 1.—Constant pressure of 140 mm. in the aorta, and pressures of 0, 10, 20, 30, 40, 60, and 80 mm. in the pulmonary artery. The type experiment was made with a pressure of 140 mm. in the aorta and 40 mm. in the pulmonary artery. With these pressures the embolic area showed a poor and somewhat irregular injection of the pulmonary vessels and capillaries in contrast to the excellent injection of the normal lung. The bronchial vessels were well injected with the red aortic mass and there was extension to the surrounding alveolar and infundibular capillaries somewhat greater in the embolic area than in the normal lung. With decreases in pressure in the pulmonary artery, the injection of the pulmonary vessels in the embolic areas became less complete. In those cases with pulmonary pressures as low as 10 mm. the extension of the bronchial vessel injection masses in the embolic areas to the capillaries near the bronchi was not more marked than in the normal lung. When, however, the pressure became zero in the pulmonary artery, the extension of the bronchial injection masses to the pulmonary capillaries in the embolic areas became more marked, but never so marked as in the normal lung. With increases in the pulmonary pressures the injection of the embolic areas through the pulmonary artery became more marked and was practically complete at 80 mm. At 60 mm. there was still considerable extension of the bronchial mass to the surrounding capillaries in the embolic area, greater than in the normal lung, but at 80 mm. this extension was not found. Throughout this series the mixture of bronchial mass and pulmonary mass in the bronchial capillaries in the embolic areas remained practically the same.

Series 2.—Constant pressure of 40 mm. in the pulmonary artery and pressures of 0, 40, 60, 100, 140, 160, 180, and 200 mm. in the aorta. The same experiment is regarded as the type in this series as in series 1. With decreases in aortic pressure down to 40 mm. there was progressively less tendency for the aortic injection mass to pass from the bronchial vessels to the alveolar and infundibular areas in the embolic area, and with a pressure of only 40 mm. in the aorta the aortic injection mass was strictly limited to the bronchial vessels. On the other hand, increases in aortic pressure insured greater penetration of the aortic injection mass into the alveolar and infundibular capillaries, so that the embolic

[5] Ghoreyeb, A. A., and Karsner, H. T., *loc. cit.*

[6] Ghoreyeb, A. A., and Karsner, H. T., *loc. cit.*

area showed somewhat more aortic injection mass than pulmonary mass. This extension is more marked in the pulmonary capillaries near the bronchi than in those a little distance removed. Throughout the series the degree of injection through the pulmonary vessels remained the same, not diminishing in extent with the increasing extension from the bronchial vessels. The mixture of the two masses in the bronchial vessels remained practically the same until pressures of 180 and 200 mm. were reached in the aorta, when the amount of pulmonary mass was considerably diminished as compared with that from the aorta.

Series 3.—Constant pressure of zero in the pulmonary artery and pressures of 60 and 140 mm. in the aorta. In all these experiments the embolic area was clearly but not sharply defined from the normal lung. With increases in pressure the embolic area showed progressively better injection, that with a pressure of 180 mm. being almost as complete as in the normal lung injected through the bronchial artery. A striking difference in the injections is the fact that whereas the use of the low pressure left many of the capillaries uninjected, the use of the highest pressure brought about injection of practically all the vessels in the embolic area. In the latter case, however, the injection mass appeared as smaller strands. This condition is due in all probability to low pressure in the embolic area rather than to constriction of the capillaries, because, as has been stated in an earlier publication,[7] the embolic area at the end of one half hour is congested rather than pale, as it would be if vasoconstriction prevailed.

Series 4.—Constant pressure of zero in the aorta and pressures of 40 and 80 mm. in the pulmonary artery. The lower pressure produced very imperfect injection of the embolic area and also of the bronchial vessels. With the higher pressure, however, the injection was almost complete, and was apparently favored by the absence of pressure in the bronchial artery. In the embolic area the bronchial vessels did not show as complete injection as in the normal lung, due to the fact that although the diminished pressure in the embolic area was sufficient for the injection of the pulmonary vessels, it was not sufficiently high to provide for good injection of the bronchials.

SUMMARY.

From series 1 it is concluded that increases in pulmonary pressure improve the circulation in the embolic areas and that decreases in pulmonary pressure limit the circulation in the same areas. In this series the bronchial vessels showed no tendency to take up the circulation in the area of pulmonary embolism until the pulmonary pressure was as low as zero, and then only to a limited degree. It has been shown, however, that in the circulation of an entire lobe the fall to zero pressure in the pulmonary circuit is followed by almost complete taking over of the circulation by the bronchial arterial supply and the most reasonable explanation of why this does not

[7] Karsner, H. T., and Ash, J. E., *loc. cit.*

occur when embolism is present in a smaller branch of the pulmonary is the inference that the physiological anastomosis between the two systems takes place in part before the pulmonary artery breaks up into branches small enough to be occluded by seeds of about three millimeters in diameter.

From series 2 it is concluded that although increases in pressure in the bronchial arteries cause somewhat greater inflow of bronchial injection mass into the embolic area, extremely high pressures are not sufficient to provide for complete circulation in the same district. This fact would tend to support further the belief that the anastomosis between the two vascular systems occurs before the smaller divisions of the pulmonary artery are reached.

From series 3 it is concluded that with a zero pressure in the pulmonary artery there is improved injection of the embolic area through the bronchial artery which is to be expected when it is remembered that zero pressure in the pulmonary artery favors a taking over of the circulation by the bronchial system. What pressure is found in the smaller pulmonary vessels is supplied by the bronchials. It has been shown that the anastomosis between the two systems probably occurs before the branches are reached which would provide lodgment for the turnip radish seeds. The pressure supplied by the bronchials would naturally be less beyond this point and hence the injection would be less complete in the embolic area than in the normal lung. This is shown to be the case by the results of the experiment.

From series 4 it is concluded that the absence of pressure in the bronchial circulation favors a better injection of the embolic area through the pulmonary vessels, which is to be expected when it is recalled that not until zero pressure is reached in the bronchial circuit does the pulmonary artery supply the bronchial vessels with blood.

Throughout the study it was found that whenever the two vascular systems were injected simultaneously the pleural vessels over the embolic area, as well as those over the normal lung, received their supply from the pulmonary vessels. When only one system was used for injection the pleural vessels over the embolic area

showed about the same degree of injection as those of the embolic area itself.

CONCLUSIONS.

The same laws for mixture of the blood of bronchial and pulmonary vessels laid down in connection with the circulation in the lung in general, apply also to the circulation in an area of embolism; *i. e.,* there is no notable mixture of the bloods until the pressure in the one system or the other sinks to zero. Simple embolism of the pulmonary artery results in lowered pressure in the embolic area, the pressure, however, not sinking to zero unless the blood supply of an entire lobe is cut off.

The circulation in the embolic area may be improved by increasing the pressure in either the pulmonary or bronchial arteries, but so long as the two circulations are going on, extremely high pressures are not sufficient to restore the circulation to normal in the area of embolic congestion.

With normal pressures in the two vascular systems, an area of pulmonary embolism involving less than the entire lobe receives its blood supply almost entirely from the rich anastomosis of the pulmonary artery between its own branches, and only when the bronchial pressure is raised to an extremely high point does the blood from this vessel play a notable part in the circulation in the area.