General Practice

Radiological Features of Normal and Abnormal Pulmonary Blood Flow

J. A. BEYERS

SUMMARY

A short review is given of the anatomy of the pulmonary arterial system and of the bronchial arterial system. The normal vascular pattern of the lung as well as the altered vascular pattern under a number of abnormal circumstances is described.

S. Afr. med. J., **55**, 881 (1979).

Pulmonary blood flow comprises two systems, namely a big volume, low pressure, low oxygen content system composed of pulmonary arteries, pulmonary capillaries and pulmonary veins, and a small volume, high pressure, high oxygen content system composed of bronchial arteries, bronchial capillaries and bronchial veins. These two systems communicate at different levels and have a certain amount of reciprocity. If the pulmonary arterial blood flow is diminished there is a tendency for the bronchial arterial blood flow to increase. If the pulmonary venous pressure is raised there is a tendency for blood to be shunted into the bronchial veins.

The pulmonary arteries and their branches accompany the bronchi and their branches and are situated centrally in the bronchopulmonary segments and pulmonary lobules. At the level of the respiratory bronchiole, the pulmonary arterioles break up in an extensive intercommunicating capillary network around the alveoli. These capillaries unite to form pulmonary venules and veins which run in the interlobular and intersegmental septa towards the hili, and two pulmonary veins from each lung drain into the left atrium.

Bronchial arteries, usually two to the left lung and one to the right lung, arise from the aorta, intercostal arteries and/or internal mammary arteries. They supply the mediastinal pleura and the vasa vasorum of the pulmonary arteries and veins. They follow the bronchi, divide with the bronchi and supply the bronchial tree with oxygenated blood down to the level of the terminal bronchiole, where they break up into a capillary network which anastomoses with the capillary network of the pulmonary arteries.

Bronchial arterial supply to the mediastinal pleura, pulmonary arteries, pulmonary veins and the bronchial tree down to the level of the terminal bronchiole is drained via bronchial veins to the azygos and hemiazygos veins and so to the right atrium.

At various levels there are communications between the pulmonary and bronchial systems. There is dispute about which of these communications are present in the normal lung and which develop only during abnormal conditions. It is suggested that the following communications are present normally but enlarge under pathological conditions:

Precapillary anastomoses between pulmonary and bronchial arteries. These are very important in pulmonary embolism where they prevent or limit the extent of pulmonary infarction, and in pulmonary atresia where they may be the only way to maintain life.

Precapillary anastomoses between pulmonary and bronchial veins. These are important in chronic pulmonary venous hypertension, as in mitral stenosis, where the anastomotic channels may become varicose and may give rise to severe haemorrhage with copious haemoptysis.

Capillary anastomoses between pulmonary and bronchial circulations peripheral to the terminal bronchiole. These form an alternate route for oxygenation of blood in congenital cyanotic cardiac disease.

Normal Markings

On a radiograph of the normal lung the lung markings are composed of pulmonary arteries and pulmonary veins only. Under normal conditions bronchi (except when seen end-on), bronchioles, alveoli, pulmonary interstitial tissue, bronchial arteries, bronchial veins, lymph vessels and capillaries are not radiologically demonstrable.

In the hilar regions the right and left main pulmonary arteries divide into branches which fan out into the lung fields. The pulmonary arteries branch at regular intervals, and smoothly and gradually taper towards the periphery; they can be followed to within 1 centimetre or so of the lung periphery.

Pulmonary veins can sometimes, but not always, be identified separately from pulmonary arteries.

In the upper lung zones the main vein draining the upper lobe lies lateral to the main artery supplying the upper lobe. In the right lung this vein is joined by a vein from the right middle lobe; the vein so formed crosses the lower part of the right hilum obliquely to drain into the left atrium. On the left side this vein is joined by a vein from the lingula and drains into the left atrium. At the right lung base, two veins draining the right lower lobe course more or less horizontally towards the left atrium and cross the pulmonary arteries to the right lower

Department of Radiology, Tygerberg Hospital, Parowvallei, CP
J. A. BEYERS, M.B. CH.B., M.MED. (RAD.), M.D. (CLIN.), *Part-time Consultant Radiologist*

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lobe more or less at right angles before they join to enter the left atrium. This crossing of vessels gives a rather vascular pattern to the right costophrenic region which must not be considered as abnormal. The right lower lobe veins can normally be seen in most patients. The venous pattern of the left lower lobe is similar to that of the right lower lobe, but these veins, contrary to those on the right side, can only rarely be identified on routine films.

Gravity has a radiologically demonstrable effect on perfusion of the lungs. In the upright position there is very little or even no perfusion at the extreme lung apex and perfusion increases progressively towards the lung base (Fig. 1). Radiologically this is reflected in small blood vessels, pulmonary arteries and pulmonary veins at the lung apex, which gradually and progressively increase in size towards the lung base. At the same distance from the hilum, pulmonary blood vessels in the upper lung zone are about one-half to one-third as big as those in the lower lung zone.

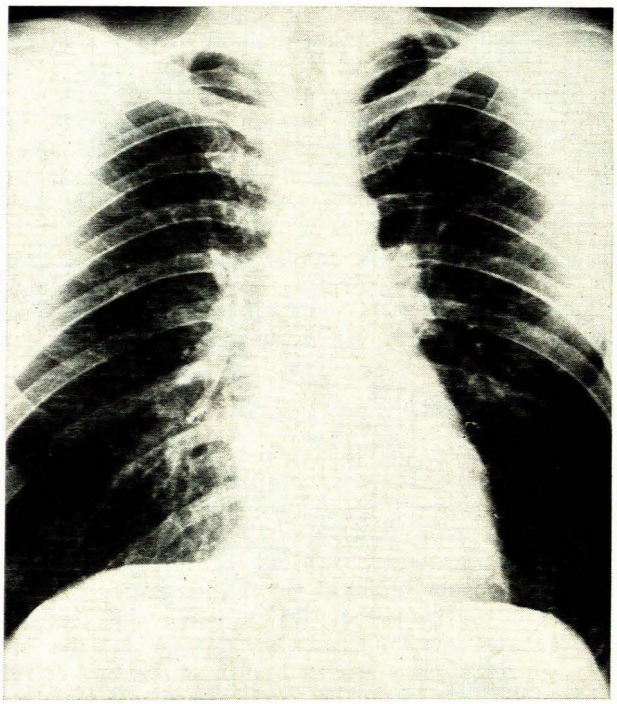

Fig. 1. Radiograph demonstrating the normal gradient of small blood vessels in the upper lung zones and big blood vessels in the lower lung zones in an erect healthy athlete.

Pathological Changes

This normal gradient in the size of the blood vessels is altered by numerous conditions, the most important of which are the following:

Position. If the radiograph is taken with the subject supine or in the Trendelenburg position the normal gradient is obliterated or even reversed (Fig. 2).

Pulmonary venous hypertension. As pulmonary venous pressure rises the pulmonary capillary pressure rises likewise and more fluid is filtered into the interstitial tissue spaces. In the erect position more fluid collects at the lung base than at the lung apex. The increased fluid interferes with passage of oxygen from alveolus to pulmonary capillary and this relative anoxia causes pulmonary vasoconstriction. Because this is more marked at the lung base, blood is shunted from base to apex and this is seen radiologically as a diminution in the calibre of blood vessels at the lung base and an increase in the size of blood vessels at the lung apex, the so-called upper lobe blood diversion, reversed pulmonary blood flow or dilated upper lobe vessels. This is the earliest radiologically detectable sign of increased pulmonary venous pressure and can often be seen before there is any clinical evidence of left ventricular failure.

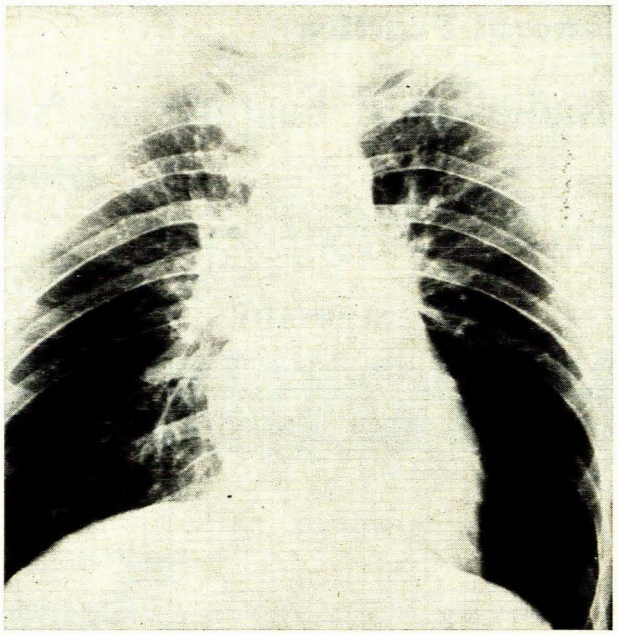

Fig. 2. Radiograph taken with the athlete standing on his head, showing complete reversal of the normal gradient of the blood vessels.

Basal emphysema and/or bronchiectasis. Because of diminution of the capillary vascular bed and the increased peripheral vascular resistance in the lung bases in these circumstances, blood is shunted to the upper lung zones and this causes a change in the normal gradient of pulmonary blood vessels (Fig. 3).

Increased pulmonary blood flow, especially in big left-to-right shunts. To accommodate this increased pulmonary blood flow the reserve vascular capacity in the upper lung zones is utilized and the difference in size between upper and lower lung vessels becomes less obvious or even obliterated.

Normally the pulmonary arteries gradually and progressively decrease in size from the hilum to the periphery without any sudden change in size or direction. In pulmonary arterial hypertension the main pulmonary arteries and their main branches are dilated and within a short distance from the hilum they suddenly decrease in size

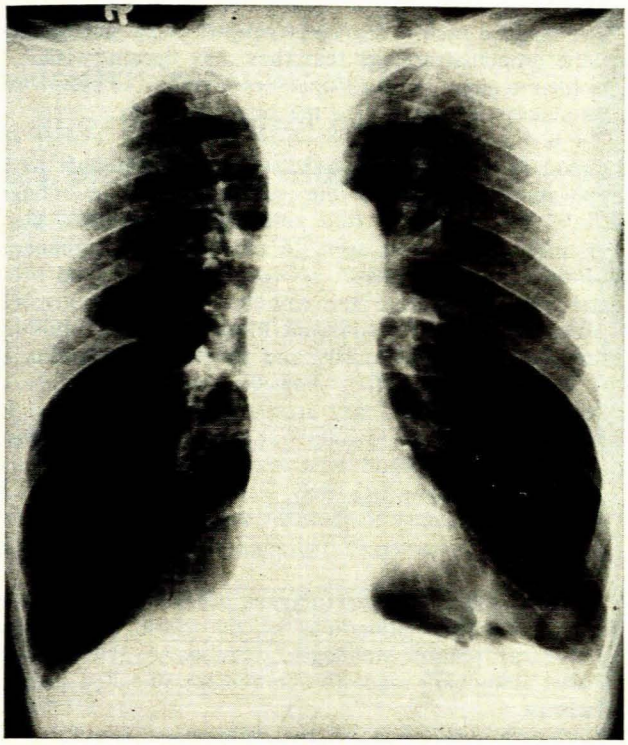

Fig. 3. Upper lobe blood diversion in basal emphysema. Note the diminutive blood vessels in the lower lung zones and the dilated vessels in the upper lung zones.

to continue as small tortuous arteries. This gives an oligaemic appearance to the peripheral two-thirds of the lung fields. The arterial pattern under these circumstances is reminiscent of a Pierneef etching of a baobab tree (Fig. 4).

The normal gradual change in size of pulmonary arteries is also affected in emphysema, where the pulmonary arteries in the emphysematous zones are abnormally thin and have few branches.

When pulmonary blood flow is increased the pulmonary arteries and veins dilate to accommodate the bigger blood flow. The reserve vascular capacity in the upper lung zones is utilized and relatively more blood flows through the upper than the lower lung zones. The main pulmonary arteries and their branches up to the periphery of the lung fields are uniformly and evenly increased in size and the lung fields appear hypervascular or pleonaemic (Fig. 5). Screening may show abnormally vigorous pulsations in the main pulmonary arteries.

The flow of fluid through a tube varies with the 4th power of the radius of the tube. If, all other things being equal, the radius of the tube is doubled, the flow will be increased 16-fold. The pulmonary blood vessels therefore need only dilate slightly to accommodate a considerably increased blood flow. Therefore pulmonary blood flow has to increase about 3-fold before it becomes radiologically detectable. As the pulmonary vessels dilate to accommodate the increased blood flow the pulmonary vascular resistance is lowered and the pulmonary arterial

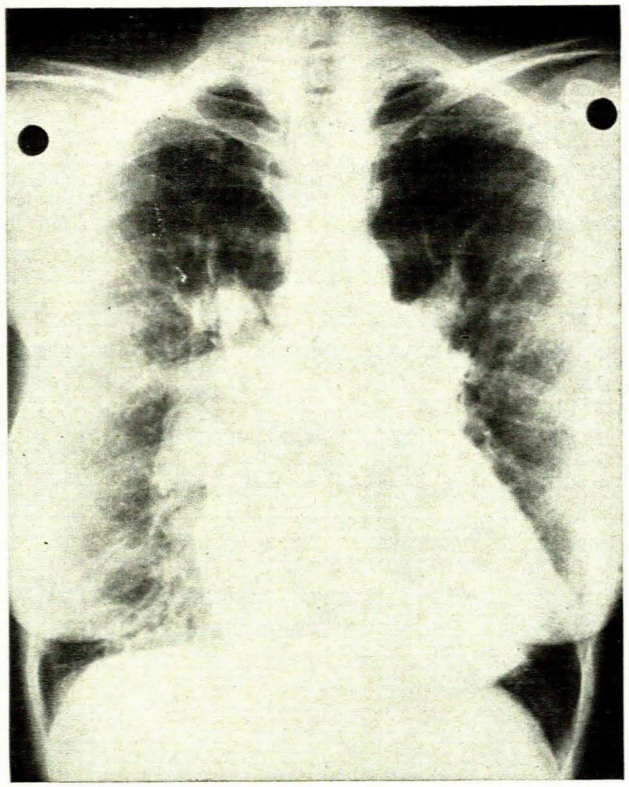

Fig. 4. Pulmonary arterial hypertension complicating an atrial septal defect. Note the abrupt change from dilated central pulmonary arteries to small peripheral pulmonary arteries.

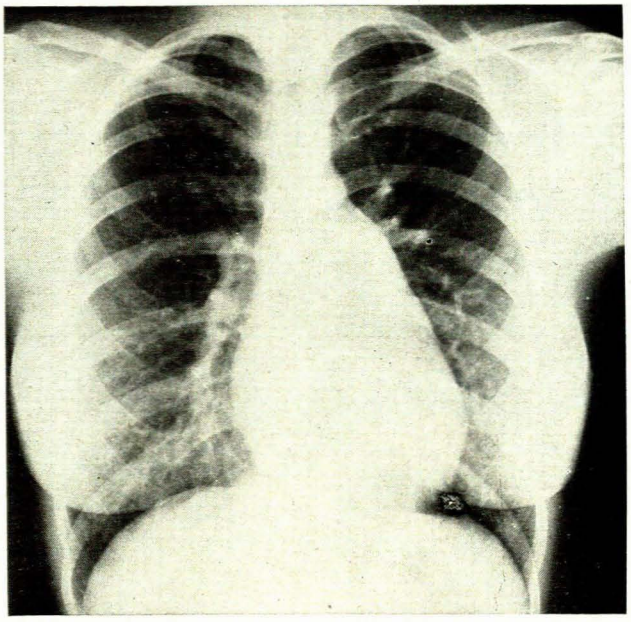

Fig. 5. Increased pulmonary blood flow in a patient with an atrial septal defect. The main pulmonary arteries and their branches, right up to the periphery of the lung fields, are increased in size and the lung fields are pleonaemic.

blood pressure remains normal until the marked ability of the lung to compensate for increased blood flow is exceeded. This relationship between pressure, flow and resistance is expressed in the formula: $P \alpha F \times R$ (P = pressure; F = flow; R = resistance) — as flow increases, resistance is lowered and pressure therefore remains normal.

In decreased pulmonary blood flow the main pulmonary arteries may be increased or decreased in size, but their branches are abnormally small and the lung fields appear hypovascular or oligaemic (Fig. 6).

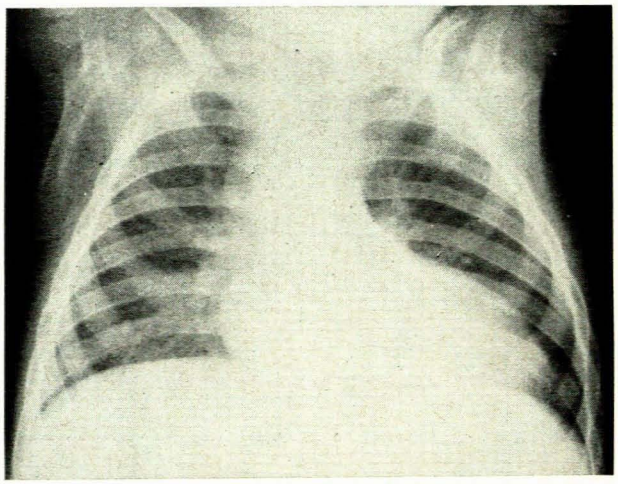

Fig. 6. Decreased pulmonary blood flow in tetralogy of Fallot. Note that the main pulmonary arteries and their branches are diminutive and that the lung fields are oligaemic. The right-sided aortic arch is a frequent finding in tetralogy of Fallot.

Although the bronchial arteries can be demonstrated by bronchial arteriography they are normally not seen on routine radiographs. When the pulmonary arterial supply is severely curtailed, for example in pulmonary atresia, there may be such a degree of compensatory enlargement of the bronchial arteries that they may become visible in the hilar regions as small opacities giving a so-called 'salt and pepper' appearance to the hilar regions.

In acute pulmonary embolism one would expect the pulmonary segment or lobe distal to the embolus to be hypovascular owing to the cut-off of the pulmonary arterial blood supply (the so-called Westermark sign), but this is only rarely seen. In most cases of pulmonary embolism the anastomoses between pulmonary and bronchial arteries distal to the embolus supply the involved segment or lobe with sufficient blood to prevent this appearance of hypovascularity and to maintain viability of the involved area. Often, however, the combination of interruption of pulmonary arterial blood supply and increased bronchial blood supply at systemic pressure causes diapedesis of red blood cells into the affected area, producing an opacity which may lead to tissue necrosis. In this way pulmonary embolism without infarction may develop and progress to pulmonary embolism with infarction.

Abnormal blood vessels may be seen and identified as such on routine radiographs of the chest in abnormal pulmonary venous drainage, pulmonary arteriovenous fistulae, pulmonary sequestration and the so-called scimitar syndrome.

Although evaluation of the pulmonary blood vessels is difficult because of the wide spectrum of normal appearances and physiological and technical factors which affect it, the pulmonary blood vessels must always be painstakingly analysed. Such an analysis gives valuable information on cardiopulmonary haemodynamics, especially as regards increased and decreased pulmonary blood flow, is often the first clue to increased pulmonary venous pressure and incipient pulmonary oedema, sometimes makes the diagnosis of abnormal pulmonary venous drainage and arteriovenous aneurysms possible, and may be the clue to the diagnosis of emphysema, pulmonary embolism, pulmonary sequestration and the scimitar syndrome.