Pulmonary capillaries

Vulnerability of pulmonary capillaries during severe exercise

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The pulmonary capillaries are vulnerable to mechanical failure during strenuous exercise

It is remarkable that it has taken so long to recognise how vulnerable the pulmonary capillaries are to mechanical failure during strenuous exercise. After all, the extreme thinness of the capillary wall was appreciated when the first electron micrographs were obtained by Low in 1952. We now know that the total area of the blood-gas barrier (BGB) in the human lung is 50–100 m^2 and that for more than half of this enormous area the thickness is only 0.2–0.3 μm.[1] The result is that during severe exercise when the pulmonary vascular pressures rise to high levels, the capillary wall stresses become extremely high approaching the breaking stress of collagen.[2] It is not surprising therefore that changes in the integrity of the BGB occur under these conditions.

Perhaps investigators were initially misled by early data from cardiac catheterisation procedures which suggested that pulmonary vascular pressures did not increase during exercise; this erroneous notion still surfaces from time to time. However, it is now known that pulmonary artery wedge pressures can rise to as high as 30 mm Hg during intense exercise, with a mean pulmonary artery pressure of as much as 37 mm Hg.[3 4] This means that the transmural pressure of some of the capillaries at the base of the lung can be as high as 40 mm Hg,[2] with the result that the extremely thin capillary wall is exposed to enormous stresses. We have shown in animal preparations that raising the capillary transmural pressure to these levels results in disruption of the capillary and alveolar epithelium, or sometimes all layers of the capillary wall.[5]

The most dramatic example of capillary stress failure during intense exercise is seen in galloping racehorses where the condition is known as exercise induced pulmonary haemorrhage. It has been known since Elizabethan times that nose bleeding is seen in some horses after a race and various explanations for this were advanced. However, now it is clear that virtually every thoroughbred in training bleeds into its lung.[6] The reason for this extraordinary situation is that the pulmonary vascular pressures are extremely high during galloping with a mean left atrial pressure of up to 70 mm Hg and a mean pulmonary artery pressure of 120 mm Hg. The reason for the high vascular pressures is that these animals have been selectively bred over hundreds of years to race, and they have enormous cardiac outputs which require very high cardiac filling pressures. It is not surprising then that these high pressures cause some capillaries to break, as we have shown directly by electron microscopy of the lungs.[7]

These findings led us to ask whether elite human athletes can alter the integrity of the BGB at high levels of exercise. Certainly there are various accounts of haemoptysis or haemorrhagic pulmonary oedema in high-performance athletes including swimmers, rowers, runners, and scuba divers, and some will remember the advice of old-time trainers to ''run until you taste blood''. We studied a group of elite cyclists who sprinted uphill for 4 km at maximal effort giving a mean heart rate of 177 beats per minute. These volunteers then underwent bronchoalveolar lavage (BAL) and the results were compared with a group of sedentary normal subjects who did not exercise.[8] The results showed that the athletes had higher concentrations of red blood cells, total protein, and leukotriene B4 in their BAL fluid than the controls. The conclusion was that brief intense exercise in these athletes altered the integrity of the BGB.

Do the same changes occur during sustained periods of submaximal exercise? To answer this question, we took another group of elite cyclists who rode at 77% of their maximal oxygen consumption for 1 h followed by BAL, and compared the results with those from normal volunteers. This time there no differences, leading to the conclusion that the integrity of the BGB is altered only at extreme levels of exercise.[9] Indeed, this is what might be expected on general evolutionary grounds.

Finally, how is it that the BGB is maintained so extremely thin for efficient gas exchange but just strong enough to withstand all but the most extreme stresses when the capillary pressure rises during intense exercise? We believe that the capillary wall senses the wall stress in some way and then regulates its structure, especially the extracellular matrix, which is responsible for its strength. Exactly how it does this is a subject of intense study at the present time.

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