To the Editor:

In a recent case report in the Journal [1], we described the adaptive mechanisms relating to gas exchange during exercise in an active young man with anatomically small lungs, the result of lung growth impairment due to pectus excavatum developed in childhood.

We found our patient to have small (total lung capacity 59% of predicted) but mechanically normal lungs. He had a normal cardiac output, a normal resting single-breath diffusing capacity (DLCOsb), and a high diffusion coefficient (Kco, 148% pred) associated with a high pulmonary capillary blood volume at rest (Vc, 131% pred). Pulmonary distensibility (K) and elastic recoil were normal. During exercise, he was unable to recruit further reserves of pulmonary capillaries, but this was not reflected in a plateau for oxygen consumption which was presumably the result of an increased pulmonary capillary blood flow rather than volume [2].

In the original report [1], we hypothesized that a period of detraining in this man might reduce his Vc, thus, allowing for DLCO recruitment during exercise; and this has in fact proved to be the case. Twelve months ago, he suffered a knee injury, which has prevented him from playing soccer since that time. He swims occasionally but has been relatively inactive compared to his soccer playing days.

He agreed to be restudied at 6, 9 and 11 months after his knee injury. All lung function parameters remained essentially unchanged after the first study at 6 months post-training, at which time both DLCOsb and Kco values averaged 14% lower than previously reported whilst active [1]. Pulmonary capillary blood volume (Vc) and maximal oxygen uptake were reduced by 22 and 21%, respectively. During steady-state exercise (fig. 1) there was a modest but reproducible recruitment of pulmonary capillaries from rest up to 25–50 W of work, whereafter a plateau of DLCO and Kco was observed. This modest recruitment is to be expected, considering his small lungs and the fact that his Vc at 90 ml was still 102% of predicted on the basis of his body surface area (BSA). Membrane diffusing capacity remained unaltered from the original values [1].

These findings confirm our predictions [1] that a period of detraining in this man might reduce his stroke volume; thus, allowing for DLCO recruitment during exercise, albeit at a lower work level. It also supports our suggestion that physical training may benefit patients with either small lungs or following pneumonectomy by recruiting available pulmonary capillaries with consequent improvement in exercise capacity.

Fig. 1 – a) Single-breath diffusing capacity (DLCOsb); and b) diffusion coefficient for carbon monoxide (Kco) at rest and during steady-state exercise (3 min) plotted against the workload in watts. The duplicate studies in the detrained state [●, ▲] were performed 2 weeks apart, 11 months after cessation of training. Run 11, from an earlier report during training [●], is plotted for comparison (▲).

References


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