Earlier study on asbestos workers, ILO scores, and oxygenation more comprehensive

The publication of “Radiographic (ILO) readings predict arterial oxygen desaturation during exercise in subjects with asbestosis” by YCG Lee et al from the Sir Charles Gardiner Hospital in Perth presents no new information and fails to reference an earlier paper on the same subject which included more patients with clinical asbestosis and four different control groups. This paper actually measured the P(A-a)O2 rather than estimating it from the oxygen saturation. Furthermore, it correlated the change in A-a O2 gradient with the amount of work performed during exercise by measuring the P(A-a)O2/VO2 ratio which was more specific and sensitive than the P(A-a)O2 gradient. The estimate of the true oxygen tension by measuring pulse oximetry is fraught with potential error well understood by most pulmonary physiologists, including motion artifact, vasodilation, hypoxia, and confusion with carboxyhaemoglobinemia, methaemoglobinemia, and anaemia. The same information contained in the paper by Lee et al is available in a more comprehensive fashion in the earlier publication by Smith and Agostoni.

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References

Authors’ reply
We read the letter from Dr Smith with interest and thank him for suggesting his paper for discussion. Dr Smith argued that (i) there was significant overlap between his study and ours, and (ii) oxygen desaturation as measured by pulse oximetry was an inappropriate measure for testing exercise desaturation. We strongly disagree with both points.

(i) Our study and that of Smith and Agostoni differed in aims, patient selection, methods, and, as a result, provided different information relevant to current clinical practice. Our study compared a validated quantitative measure of severity of chest x ray changes (ILO readings) with a readily available, quantitative measure of gas exchange during exercise (arterial oxygen desaturation). We studied 38 subjects with asbestosis diagnosed on the basis of exposure history, clinical signs of asbestosis, and typical HRCT appearances of asbestos induced interstitial fibrosis, in keeping with modern clinical practice.

The study of Smith and Agostoni did not do this. They separated 95 asbestos exposed workers into those with asbestosis and those without. The asbestosis group (n = 27) included a mixture of patients with ILO profusion >1.0 as well as subjects with unexplained reduction in lung volume or DLco, who did not have radiological evidence of asbestosis—debatable inclusion criteria rarely used elsewhere. They then used various measures of arterial oxygenation during rest and exercise to discriminate those 27 patients from asbestos exposed workers who did not have asbestosis. The best of their measures, deoxygenation on exercise, showed modest discriminatory power. They then related this variable to ILO profusion scores for their complete patient group, making no distinction between those with and without asbestosis.

Our study directly related resting lung function and a more comprehensive ILO radiographic assessment (profusion, zones affected, and pleural thickening) to deoxygenation during exercise in independently diagnosed asbestosis subjects and showed a significant relation. By multivariate analysis, we found that arterial oxygen desaturation was independently predicted by a combined use of DLco, FEV1/FVC ratio, and the number of affected zones on ILO scoring. While the profusion score correlated with oxygen desaturation on linear regression, it did not remain significant in the multivariate analysis. Smith and Agostoni only examined one ILO parameter, the profusion score, and multivariate analysis was not conducted. Hence, we emphatically disagree with Dr Smith’s suggestion that our paper “presents no new information”.

An important message from our study is that the extent of parenchymal damage (number of zones affected) is correlated with resting pulmonary function and is an independent predictor of exercise desaturation. Previous studies of ILO scores have mainly focused on the profusion and few, if any, have placed importance on the influence of the extent of asbestosis on physiological impairment. Our findings are consistent with recent studies showing that the area affected by pulmonary fibrosis as quantified on HRCT correlates well with pulmonary function.

(ii) Dr Smith went on to argue against the validity of pulse oximetry as a measure of gas exchange during exercise. Pulse oximetry is a standard methodology, in commonplace use throughout the world because of its validity and simplicity, displacing indwelling arterial lines, which are invasive and have largely fallen into disuse for clinical purposes. Hence, we strongly disagree with Dr Smith’s suggestion that oxygen desaturation measured by pulse oximetry is invalid for assessing exercise desaturation in asbestosis patients.

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