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THE INTERACTION OF AGEING AND LUNG DISEASE

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INTRODUCTION

Respiratory disease is the second commonest cause of severe disability in older adults, second only to musculoskeletal disorders (and four times more common than stroke). Atypical presentations are more common in old age, and older patients may report less breathlessness [1] than younger adults despite similar physician assessed severity [2].

There is still compelling evidence that older people are not treated purely on the basis of their physiological condition and their wishes, but on age per se. The medical profession is unduly pessimistic about the outcomes for older people. It is never more true that patients in this age group are either over treated when palliative care would have been preferable to the patient, or undertreated because of the physician's expectation of a poor outcome.

There is very good evidence regarding the futility of basing treatment decisions on the ground of age alone. In the 2007 CAOS study of prognosis for patients with COPD in intensive care units, age was only a small independent factor [3]. In this study, there was a marked deviation between physician-predicted survival and actual survival. In fact, the tenth of patients with the poorest clinician prognosis had a predicted 180 day survival of around 3% and an actual survival of around 36%. Eleven characteristics were found to have a significant independent association with increased hospital mortality in addition to age, such as the presence of severe respiratory disease, low pH, low serum albumin and multi-organ failure.

Premorbid physical and cognitive function should also be added to that list, something that is not well assessed and probably a better surrogate than age itself. The only thing that does increase with age is variability of physical activities– from the marathon runner to the bedbound!

Homeostatic instability is a hallmark of ageing – there is no homogeneous biomass called "the elderly". And so we have to distinguish between the fit and the frail...and a large number of people somewhere in between.

Reduced	**Increased**
Lung elasticity	Dynamic lung compliance
Respiratory muscle strength	Residual volume
Chest wall compliance	Oxygen uptake on exercise
Vital capacity	
Perception of bronchoconstriction	**Unchanged**
Forced expiratory volume	Total lung capacity
Ventilatory capacity	Airway resistance
Bronchial hyper-responsiveness	Pulmonary arterial resistance
Diffusing capacity	Arterial carbon dioxide levels
Arterial oxygen pressure and saturation	

Table 1. Major changes in lung physiology with age. Adapted from [4] with permission

IMPAIRMENT OF LUNG PHYSIOLOGY IN OLD AGE:

Table 1 illustrates major physiological changes that are thought to occur with respiratory system in old age. The major factors in the declining lung function with age are summarised as follows:

1. Loss of lung elasticity
2. Increasing stiffness of the chest wall
3. Reduced respiratory muscle strength

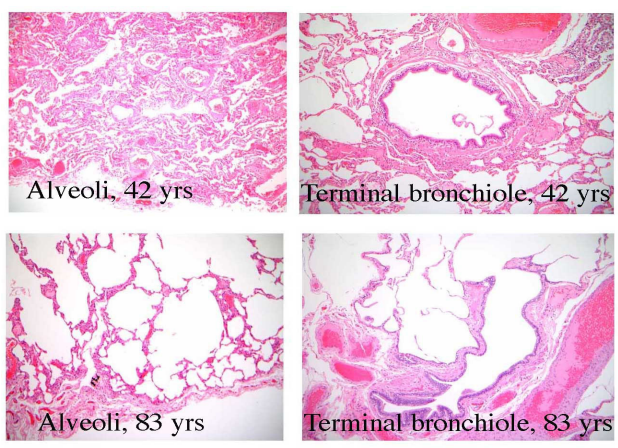

Figure 1. Section of normal lung in a young and old subject.
The top right photograph in Figure 1. is a normal terminal bronchiole from a 42 year old man. The lower right photo is an old but normal terminal bronchiole with a hugely dilated respiratory bronchiole/ alveolar duct coming off it, illustrating the dilatation of the old bronchiole. On the left we see normal visceral pleura with normal underlying lung parenchyma in the same young specimen, and beneath the older lung demonstrating marked age-related dilatation of the alveoli. The loss of lung elasticity may be due to stretch or alteration in cross linkage of elastin and/ or collagen.

4. Airspace dilatation (figure 1), increased collapsibility of small airways and a reduction in expiratory volumes. This increased tendency to collapse may be the cause of the poor reliability of bi-basal crackles as a physical sign in old age All these changes are similar to what happens in emphysema or small airways disease and sometime called "senile emphysema".

Total lung capacity remains unchanged, an important differentiation between "senile emphysema" and actual emphysema. This is because as lung elastic recoil reduces, the reduction in vital capacity is associated with an increase in residual volume, and chest wall stiffness.

5. Reduction in diffusing capacity. Gas transfer in the elderly is occasionally reduced to approximately 1/2-2/3rd of the values predicted for young adults. This fall has been attributed to two factors- a reduced efficiency of ventilation producing lower effective alveolar volume and reduced blood volume in pulmonary capillaries.

6. Gas exchange is only mildly affected at rest as oxygen requirements often meet basal metabolic rate. However, during exercise there is an increased ventilation-perfusion mismatch due to small airway closure and reduced alveolar surface area (which reduces by around 20%). This alveolar dead space affects arterial oxygen without impairing carbon dioxide elimination.

7. Both inspiratory and expiratory respiratory muscle strength decreases with age but its clinical implications have been poorly studied. This may impair cough strength, which is important for airway clearance.

8. Relative inefficiency in the monitoring and control of ventilation including decreased sensation of dyspnoea

9. Diminished ventilatory response to hypoxia and hypercapnia which make older patients more vulnerable to ventilatory failure during high demand states such as heart failure and pneumonia, and therefore to possible poorer outcomes.

These changes may explain the increased impact of lung disease with age. Concomitant factors such as under nutrition (common in older people) and those with neurological and respiratory disease will have an added effect on propensity of older people to acquire lower respiratory tract infections.

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- Occupational hazards
- Heavy alcohol consumption
- Obesity
- Respiratory infections in first year of life
- Nutritional status at birth
- Maternal or passive smoking

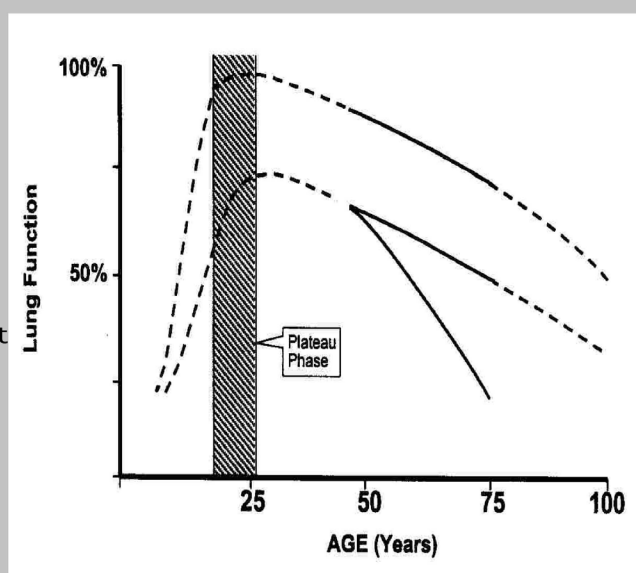

Figure 2.
Factors related to age-associated decline in FEV1. Taken from [4] with permission

Lung function will be heavily influenced by impairment of lung growth (figure 2). The lung matures by age 20–25 years, and thereafter ageing is associated with progressive decline in lung function. Many factors have been observed to influence lung growth and decline as outlined here. A notable recent research outlined childhood origin of accelerated decline in FEV1 [5]. Childhood adverse factors include maternal asthma, childhood asthma, maternal smoking and childhood respiratory infections to be associated with lower FEV1 and a more decline in FEV1.

Most lung function data is derived from cross sectional studies and there is good data available up to the age of 85. The decline in lung function with age may be non-linear with some reports suggesting acceleration in the rate of decline in FEV1 .

COPD in older adults as in all ages is underdiagnosed (please see the chapter on COPD in this issue). For older population there is an equal over-reliance in FEV1/ FVC ratio criteria which may also result in over-diagnosis. This is because FVC begins to decline later than FEV1 and at a slower rate. This results in a natural fall in the FEV1/VC ratio from about 75% to about 70% by age 70 yrs. This means that the definition of airflow obstruction on FEV1/FVC ratio of <70% is likely to be met in many more older healthy people who would incorrectly diagnosed with COPD[6].

INTERACTION OF AGEING AND LUNG DISEASE

Age itself is a risk factor for lung disease. There are higher levels of frailty, worse cognitive function and musculoskeletal changes such as kyphosis which may impair respiratory function and may adversely affect the outcome of management of lung diseases. (figure 3.)

Frailer patients may present with atypical symptoms and signs of respiratory disease and the contribution of co-morbidities is greater than younger patients. Attention to ability for patients to function at home and other social circumstances is as important as managing physical features of diseases.

Cognitive function is a vital area in the assessment of the older person. The new national dementia strategy launched in February 2010 [7] sets out several steps to improve the care for such people. As up to 70% of acute hospital beds are currently occupied by older people and up to a half of these may be people with cognitive impairment. Although 85% of elderly people can perform spirometry [8], accurate lung function measurements depend on cognitive function. Figure 4 shows the ability of 208 institutionalised elderly patients to perform spirometry versus a technically simpler respiratory impedance measurement in relation to their cognitive function [9].

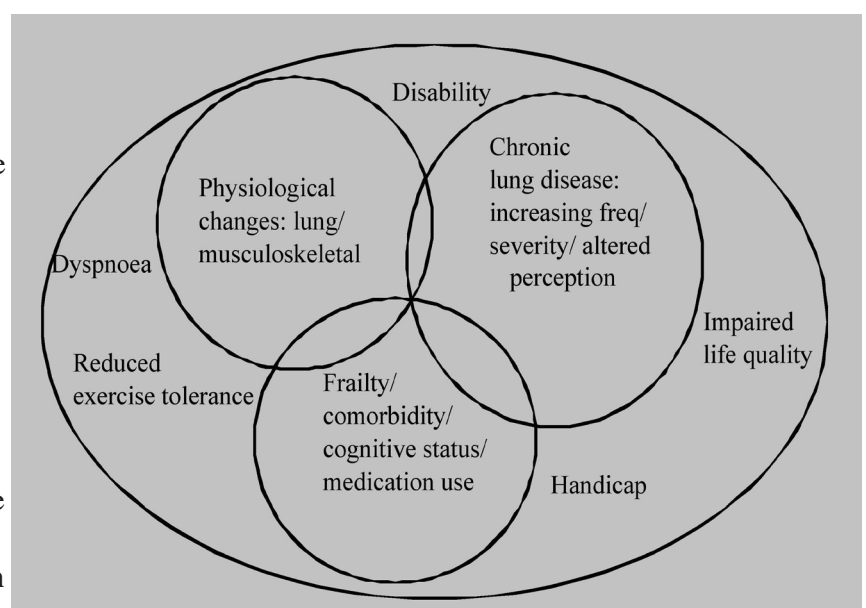

Figure 3: Interaction between old age and other clinical and physiological features.

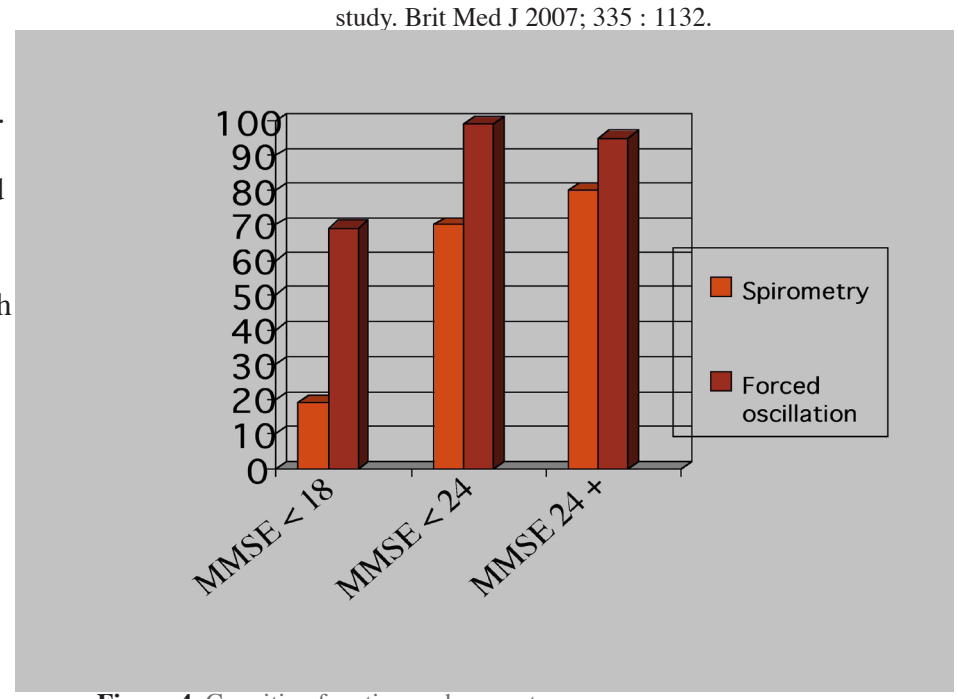

Figure 4. Cognitive function and percentage completion of spirometry and forced oscillation using the Mini-Mental State Examination Score (MMSE) in institutionalised patients

CONCLUSIONS

The ageing population presents us with many challenges and it is useful to understand the changes that occur with age in the lung. We should also be aware of the broader view in such patients, and take into account issues of frailty, dementia and physical functioning. Unfortunately some of the research conducted in this area is out of date. It would be advantageous to set new methods to better understand the consequences of aging on lung health and disease.

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