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Asthma exacerbations

In their excellent article on asthma exacerbations,[1] Aldington Beasley, ask ''…why there is such a huge discrepancy between the management of severe asthma recommended by evidence based guidelines, and that observed in clinical practice''.

Although the guidelines are in fact quite simple and straightforward, I think that non-specialist junior physicians in the emergency department are confused by the apparent complexity of, for example, fig 3 from their article reproduced from the British Thoracic Society guidelines, especially when faced with an extremely unwell patient with asthma.

For a number of years, I have taught a very simple ''6 P rule'' for the assessment of asthma:

- **P**EFR—baseline and response to first nebuliser.
- **P**ulse, >120 (it is not due to salbutamol).
- **p**O_2 (measure and then titrate oxygen against O_2 saturation).
- **P**anic (ie, ability to speak/respiratory rate).
- **P**aradox (patients cannot sustain this for long).
- **P**neumothorax (make sure the trachea is central until you can obtain a chest *x* ray; and do not allow *anyone* to put in a subclavian line).

This is the basic information needed to assess severity, and decide on management, and it is more easily taught and remembered than a complex figure.

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Competing interests: None.

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Authors' reply

We appreciated Professor Woodcock's comments and practical suggestion of the 6P rule as a quick and simple method for assessing asthma severity. We consider that the crucial issue in considering assessment tools is whether their use results in an appropriate therapeutic response. This can be achieved if assessment tools are directly linked to guidelines for management, which is the approach recommended in the British Thoracic Society algorithm (see fig 3). In this way, management is dictated by the results of the assessments made. Thus while the 6P rule is certainly quick and easy to remember, an appropriate decision will still need to be made, and the British Thoracic Society algorithm represents an ideal system to achieve this outcome.

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Innate immune activation in neutrophilic asthma

We would appreciate the opportunity to comment on the very interesting recently published paper by Simpson and colleagues,[1] putatively describing innate immune activation in a ''neutrophilic variant'' of asthma, in inhaled corticosteroid (ICS) treated patients. We feel that the paper is especially important and effective in highlighting the heterogeneity of airway cellular infiltrates in asthma, especially after exposure to corticosteroid.

We endorse the proposal that neutrophils are involved pathogenically, even in stable asthma. This is likely to be the case even when neutrophils are not grossly elevated in number, and indeed they may be at least as relevant as eosinophils across the board, as suggested in early bronchoscopic studies.[2] In these published data, neutrophil cellular activation, and also macrophage activation, were more marked than their absolute number suggested, even in patients with relatively mild, stable asthma.

Cumulative studies suggest that the role of eosinophils has perhaps been over emphasised in the airways of patients with mild, non-ICS treated asthma. Because eosinophils are so absent generally in normal control data, they give a very strong average signal in asthma. They also decrease markedly in numbers generally with ICS treatment,[3] although symptoms and bronchial hyperresponsiveness may persist. We found it interesting that in the data presented by Simpson and colleagues,[1] the actual numbers of sputum eosinophils in absolute terms in ''neutrophilic'' asthma were just as elevated as they were in their ''eosinophilic'' group. The former sputum samples were generally much more cellular and so the eosinophil percentage was found to be markedly lower. It is difficult to know if this is the more relevant end point to focus on.

Many asthmatic airways are acellular even under baseline conditions, and become even more so with ICS treatment,[3] as Simpson and colleagues[1] point out. This fact tends to get overlooked when using mean data for statistical purposes. The response to ICS therapy is also variable, and some individuals with asthma given ICS show an increase in airway neutrophils[4]; it may be this variant that Simpson and colleagues are describing.[1] Their ''paucigranulocytic'' group may reflect the more general trend to become less cellular with ICS.[3] Interestingly, we have previously found that long acting β_2 agonists had an antineutrophilic and especially an anti-interleukin 8 effect on airway inflammation,[4] which may explain some of its added value in combination with ICS.

Simpson and colleagues[1] did not find an elevation in soluble CD14 in sputum in neutophilic asthma, as an index of innate immune activation, as we have previously in bronchoalveolar lavage in post-lung transplant bronchiolitis obliterans syndrome, where bacterial infection is likely to be part of the pathogenesis.[5] We wonder whether the increase in toll-like receptor mRNA that they describe could not just reflect the corresponding increase in absolute number of neutrophils and macrophages which carry these receptors? Although Simpson and colleagues[1] raise some highly pertinent issues, many of the questions that arise from their cross sectional study will inevitably need further longitudinal interventional studies.

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Competing interests: None.

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