asthma as close to control as possible and is acceptable to both the patient and physician".

"Minimal level" of treatment and the duration of the periods where asthma control is maintained need to be specified further. An algorithm of this type may fit well with a treatment principle where control is obtained rapidly and diagnosis confirmed in the first step. In the following steps, the treatment is optimised alongside with the education of the patient.

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#### STATEMENT OF INTEREST

None declared.

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## To the Editors:

We welcome the important article delineating the difference between severity and control in asthma [1], but we wonder if, on reflection, a different nomenclature, with a broader scope, might be more useful in real life clinical practice. The authors' propose subgroups of severe asthma, where poor control for extraneous reasons (for example medication issues and/or associated co-morbidities) are included, although it was correctly pointed out that the term "severe" asthma should be reserved for those with a *requirement* for high intensity treatment. Typically, asthmatics come to a specialist clinic because of failure to respond to high dose therapy and, as discussed, there are many reasons for this.

We have proposed the umbrella term "problematic severe asthma" for these patients [2]. They may have either or both of poor baseline control and severe exacerbations. The first step is a detailed multidisciplinary assessment, if possible including a home visit, reviewing records of dispensed prescription and a psychological assessment. This initial assessment leads to about half the patients being placed in the "difficult" category [3]. Their problem (i.e. poor compliance) may not be easily resolved but they are clearly not candidates for potentially toxic therapies, such as cyclosporin or etanercept. The remainder then should have a detailed assessment of airway inflammation and an evaluation of the response to intramuscular triamcinolone (or another reliable method of administering corticosteroids which cuts out uncertainty of patient adherence) to determine steroid responsiveness. Most, but not all of this group, will turn out to be truly "severe, therapyresistant", whose exacerbations, poor baseline control or both, may need innovative therapies. Our view is that different names for specific categories will lead to a reduction in the current confusion in the literature which has been highlighted [1]. Our suggestion, shown in figure 1, emphasises the distinction between difficult-to-treat asthma and severe, therapy resistant asthma. In fact, the same concept applies at any level of asthma severity; poorly controlled asthma deserves consideration of the reason for the difficulty before costly treatment is increased.

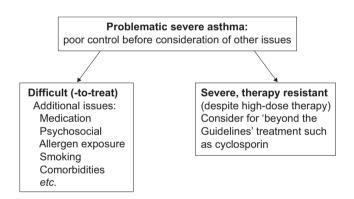


FIGURE 1. Suggested nomenclature of subgroups of problematic severe asthma

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#### STATEMENT OF INTEREST

Statements of interest for F.M. de Benedictis and G. Wennergren can be found at www.erj.ersjournals.com/misc/statements.shtml



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#### From the authors:

We are grateful to F. Madsen, and A. Bush and colleagues for their positive comments on the work of the American Thoracic Society/European Respiratory Society Task Force on Asthma Control, Severity and Exacerbations, only part of which is contained in the recently published article [1]. We await the publication of the full statement, which contains specific recommendations about the assessment of asthma control.

We agree with F. Madsen that social and cultural perspectives on the part of the patient as well as the clinician will determine the relationship between optimum asthma control and the minimum treatment required to achieve it. In the paper we state that "the patient's perspective of what constitutes "ideal" control may reflect a personal balance of priorities between clinical benefits and real or perceived risks (including side-effects and the cost of treatment)". This is consistent with F. Madsen's operational definition.

However, for reasons outlined in the article [1], it remains important that the relationship between treatment requirements and asthma severity should be considered separately. Asthma severity is defined as "the intensity of treatment required to control the patient's asthma". This is focused more on objective rather than subjective measurements, and is affected by the asthma phenotype. The distinction may seem subtle, but it is an important one which needs to be grasped so that the relationship between clinical trial evidence and individual patient treatment requirements can be more clearly understood.

We believe that A. Bush and colleagues' model for "proble-matic severe asthma" does not differ substantially from that outlined in our paper (see fig. 1 in our previously published study [1]). Identifying therapy-resistant asthma as a particular phenotype separate from other causes of difficult-to-treat asthma is an important clinical goal that we would support. However, the model of A. Bush and colleagues does not take into account patients in whom good control is achieved using high doses of therapy, in which case it is severe but not necessarily clinically problematic.

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#### STATEMENT OF INTEREST

None declared.

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# The clinical importance of rhinovirus-associated early wheezing

To the Editors:

In a recent report by a European Respiratory Society Task Force [1], human rhinovirus (HRV)-associated bronchiolitis and wheezing illness were not sufficiently discussed. Several recent observations highlight the importance of HRV infections in young wheezing children.

First, HRV is commonly associated with bronchiolitis and early-life wheezing, second only to respiratory syncytial virus (RSV). Detection rates have reached 40% in hospitalised wheezing infants [2, 3]. Moreover, HRV infection has been associated with the severity of illness [4].

Secondly, HRV infection among early wheezers is an important independent risk factor for recurrent wheezing [2, 5–8]. In population-based studies on young hospitalised children with

acute wheezing, HRV infection has been associated with recurrent wheezing ( $\geq$ 3 physician-confirmed episodes) during a 12-month follow-up period after the first episode (hazard ratio 5.1, 95% confidence interval (CI) 1.0–25, *versus* RSV-positive cases) and with the development of asthma at school-age (odds ratio 4.1, 95% CI 1.0–17, *versus* HRV-negative cases) [2, 6].

In outpatient populations with increased risk for atopic illnesses,  $\geqslant 1$  wheezing illness during infancy with HRV markedly increased the risk for third-year wheezing (odds ratio 10, 95% CI 4.1–26, *versus* HRV-positive cases with no moderate-to-severe respiratory infections) and modestly increased the risk for asthma at age 6 yrs (odds ratio 2.8, 95% CI 1.1–7.5, *versus* HRV-positive cases with no wheezing) [5, 8]. Interestingly at the third year of life, wheezing with HRV was markedly associated with asthma at age 6 yrs (odds ratio 26, 95% CI 8.2–80, *versus* those

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