Original articles

Rapid onset asthma: a severe but uncommon manifestation

J Kolbe, W Fergusson, J Garrett

Abstract

***Background*—Studies of asthma death and severe life threatening asthma (SLTA) include reports of patients who had rapid onset asthma. A study was undertaken to determine the relative frequency of rapid (<6 hours duration) and slow (≥6 hours) onset attacks in patients admitted to hospital with acute severe asthma, and to establish whether those with rapid onset asthma differ in terms of risk factors for asthma morbidity and mortality such as indices of asthma severity/control, socioeconomic factors, health care, and psychological factors.**

***Methods*—A cross sectional study was performed on 316 patients aged 15–49 years admitted with acute severe asthma and interviewed within 24–48 hours of admission.**

***Results*—Patients underestimated the duration of the index attack. Only 27 (8.5%) were classified as rapid onset. There were more men in the rapid onset group than in the slow onset group (52% versus 26%), and there was evidence of socioeconomic advantage in the patients with rapid onset attacks. The rapid onset group had more previous episodes of SLTA and were more likely to present with SLTA, but there was no difference in length of stay in hospital. The rapid onset group were less likely to have presented to a GP during the index attack and were more likely to have used ambulance services. There was no difference between the groups in any psychological or health care measure.**

***Conclusions*—Rapid onset attacks are an important but uncommon manifestation of asthma that are more likely to present with SLTA in patients who are more likely to have had previous SLTA. Male subjects are at increased risk of rapid onset attacks, and socioeconomic disadvantage, deficiencies in health care (ongoing and acute), and psychological factors are no more common in these patients than in those with attacks of slow onset. These data are consistent with the hypothesis that there is a small proportion of patients with rapid onset severe asthma who do not have the usual risk factors associated with asthma morbidity or mortality, and thus require different management strategies.**

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Keywords: acute asthma; rapid onset asthma; severe life threatening asthma; risk factors

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Studies of asthma death[1–13] and of severe life threatening asthma (SLTA)/near fatal asthma[14–16] include reports of patients who, within hours of being well and apparently free of asthma symptoms, have developed acute severe asthma, sometimes with fatal consequences. The reported incidence of such sudden severe precipitous attacks varies widely in studies of children and adults. Martin *et al*[15] reported that only 17% of children experiencing a near fatal episode of asthma in Adelaide (Australia) had sudden collapse, while the Victorian (Australia) asthma mortality study suggested that the majority (79%) of their young subjects aged 0–19 years presented with sudden collapse.[10] MacDonald *et al*[2] reported that a quarter of asthma deaths outside hospital in Cardiff occurred within 30 minutes of the onset of the attack, while only a third of patients had attacks lasting more than eight hours. In the New Zealand asthma mortality study[5 6] death occurred within three hours of the apparent onset of symptoms (a precipitous attack) in 29% of cases and in a further 11% the attack was "probably precipitous". In a more recent "district confidential enquiry" into deaths due to asthma in the UK the final attack was said to have developed rapidly (in under three hours) in 77%.[8] In the studies of asthma death the duration of the attack has been estimated from information obtained from relatives or others some time after the attack.[2–6 8–12] However, such estimates are often incomplete, inaccurate, and are subject to a variety of biases.

Our clinical experience has suggested that many patients substantially underestimate the duration of their attack. In asthma deaths (and severe life threatening asthma) severe, poorly perceived or disregarded airway obstruction, often associated with overuse of bronchodilators, may mask a more prolonged attack. Whilst classifications of asthma based on rate of onset of symptoms and/or airway narrowing have been proposed,[17] there has been little or

Table 1 Duration of index attack estimated by either a single direct question or a detailed description of symptoms

	Duration of attack (n = 316)	
	Based on single direct question	*Based on detailed description of symptoms*
<6 hours	108 (34%)	27 (8.5%)
6–24 hours	62 (20%)	38 (12%)
1–3 days	73 (23%)	93 (29%)
≥4 days	73 (23%)	158 (50%)

no scientific evidence to support rapid onset asthma as a valid clinical entity and no data on its incidence in relation to other forms of presentation.

Nevertheless, immunohistological differences have been described between cases of asthma death of slow compared with sudden onset.[11 12] Those with fatal attacks of slow onset had predominantly eosinophils in the airway mucosa while those with sudden onset fatal asthma had predominantly neutrophils; the longer the time interval between the onset of the attack and death the more eosinophils and fewer neutrophils were present.[11] While there may be two distinct pathological entities of airway inflammation resulting in distinctly different patterns of excessive airway narrowing, different phases of the same pathological process have not been completely excluded, nor has the possibility that different stimuli may result in different patterns of airway inflammation.[12]

The aims of this study were (1) to compare the estimates of duration of attack obtained by different methods; (2) to determine the relative frequency of rapid and slow onset attacks in patients admitted to hospital, including those with SLTA; (3) to determine whether those with rapid onset asthma differ from those with slow onset attacks in terms of conventional risk factors for asthma morbidity and mortality—that is, demographic characteristics, indices of asthma severity/control, socioeconomic and psychological factors.

Methods

STUDY DESIGN AND PATIENTS

A cross sectional study was undertaken of patients aged 15–49 years with acute severe asthma, normally resident in the Auckland region, admitted to the four major hospitals in the region. Patients aged ≥50 years were excluded to avoid the major inaccuracies in the diagnosis of asthma in older patients. The lower age limit is the age at which patients are transferred to adult clinics and are considered to be of sufficient maturity to assume responsibility for their own health. Patients were either admitted direct to the intensive care unit (ICU) with severe life threatening asthma[18] or to the general medical wards. Patients were excluded if the primary reason for the admission was not acute asthma or the patient was admitted primarily for a complication of asthma such as pneumothorax or pneumomediastinum, or because of the presence of other serious illnesses. All patients satisfied the criteria for reversible airflow obstruction—that is, improvement or bronchodilator response of >20% improvement in peak expiratory flow (PEF) or forced expiratory volume in one second (FEV_1).

A detailed questionnaire was administered to all patients within 24–72 hours of admission to the general medical ward (either directly or after discharge from the ICU) by a single research associate (WF). The interviewer was blinded to the specific aims of the study. All components were administered by the interviewer and included:

(1) Demographic data.

(2) Quality of previous medical care and usual asthma management including acquisition of PEF meters, action plans, availability of oral steroids, measurement of PEF, checking of metered dose inhaler technique, and accessibility of the family doctor.

(3) Details of the patients' socioeconomic status with particular reference to aspects which may influence asthma management including unemployment, financial dependence on social security benefits, financial difficulties in the last year, inability to afford doctor visits or prescription costs.

(4) Asthma morbidity in terms of number of ICU admissions (over the last 12 months), hospital admissions (over the last 12 months), Emergency Department (ED) visits (last 12 months), courses of oral corticosteroids (last 12 months), and the need for continuous oral steroids (≥7.5 mg prednisone/day for one month or longer) in the last 12 months.

(5) Assessment of practical knowledge of self management of acute asthma by the use of scenarios describing two hypothetical attacks.[19 20] One described an attack of increasing severity over seven days (slow onset) whilst the second described an attack which developed over one hour (rapid onset). Both scenarios ended with the subject "experiencing" a severe attack such that he/she was so wheezy and short of breath as to be unable to speak or rise from a chair. At three stages during each of the scenarios subjects were asked to describe what action they would normally undertake if they were actually experiencing such symptoms. The scoring system, in which scores were weighted for strategies considered most important in aborting an attack or to be potentially life saving, was based on consensus statements by the Thoracic Society of Australia and New Zealand (TSANZ)[21] and the British Thoracic Society (BTS)[22 23] on the management of asthma. The total possible score for each scenario is 25, a score of <15 being considered

Table 2 Demographic and socioeconomic indicators in those with rapid or slow onset asthma

	Rapid (n=27)	*Slow (n=289)*	*p value*
Demographics			
Mean (SD) age	26.9 (9.5)	30.2 (10.3)	NS
Sex (% male)	52%	26%	<0.005†
Current smoker	27%	32%	NS
Socioeconomic			
Paid job	67%	49%	NS
Only income of household a social security benefit	11%	34%	<0.01†
Private medical insurance	44%	28%	<0.06†
Put off going to GP because of cost	26%	42%	NS
Unable to afford prescription costs	19%	39%	<0.04

†χ^2 test.

Table 3 Circumstances of the index attack: (A) pre-hospital management, and (B) features on presentation

	Rapid (n=27)	*Slow (n=289)*	*p value*
(A) Pre-hospital management:			
During index attack			
Use of peak flow meter	26%	43%	NS
Use of action plan	37%	31%	NS
Use of nebulised bronchodilators	30%	28%	NS
Use of oral steroids (prior to admission)	26%	27%	NS
First "port of call"			
GP	19%)	48%)	
Ambulance	33%)	12%)	0.004†
Emergency department	48%)	40%)	
(B) Features on presentation:			
Presentation with SLTA[18]	67%	20%	<0.001†
Cardiopulmonary arrest	22%	6%	<0.003†
Impaired conscious level	56%	13%	<0.001†
Mechanical ventilation	19%	4.5%	<0.001†
Initial arterial blood gas tensions			
pH	7.25 (6.8–7.6) (n=25)	7.39 (6.9–7.6) (n=198)	<0.006‡
$Paco_2$ (kPa)	8.7 (2.6–34.0)	5.1 (2.2–19.7)	<0.001‡
Mean (SD) days in hospital	3.7 (2.9)	3.7 (2.6)	NS

†Fisher's exact test.
‡Wilcoxon signed rank test.

Table 4 Indices of quality of ongoing asthma specific health care, asthma severity, and previous asthma morbidity

	Rapid	*Slow*	*p value*
(A) Quality of ongoing asthma specific health care			
Very easy to obtain GP appointment	96%	76%	NS
Availability of peak flow meter	85%	81%	NS
Written action plan	59%	49%	NS
Supply of oral corticosteroids	56%	43%	NS
MDI techniques checked in last year	52%	47%	NS
Treatment			
Dose of inhaled steroids (BDP equivalence)			
0	19%	27%	
<1000 μg	48%	38%	NS
1000–2000 μg	22%	25%	
>2000 μg	11%	9%	
(B) Morbidity			
Moderate/severe interference with sleep	67%	60%	NS
Moderate/severe interference with exercise	48%	53%	NS
Previous ICU admissions (ever)	1.85 (3.1)	0.9 (1.9)	0.0001†
Hospital admissions in last year	0.8 (1.3)	1.2 (3.8)	0.0001†
Visits to emergency department in last year	1.3 (1.8)	2.6 (8.0)	0.0001†
Visits to GP in last year	5.1 (4.2)	8.4 (10.9)	0.001†
Urgent visits to GP in last year	1.0 (1.9)	1.8 (4.7)	0.001†
Courses of oral steroids in last year	2.5 (3.3)	3.3 (4.7)	NS

BDP = beclomethasone dipropionate.
†*t* test for independent samples.

representative of clinically significant inadequate self management knowledge.

(6) Patient behaviour; assessed by a very detailed history of symptoms and self management strategies undertaken before admission to hospital.[20] The interviewer determined the duration of symptoms before admission which specifically included how long the patient "had been more wheezy and short of breath than usual" (detailed questioning). This was regarded as the attack duration and the index attack was then classified as rapid (<6 hours) or slow (≥6 hours). In addition, and in a separate part of the interview, the subject was simply asked to estimate "how long the attack had been going before coming into hospital" (single direct question).

(7) Level of anxiety and depression using the Hospital Anxiety and Depression (HAD) scale,[24] which is specific for distress in physically ill subjects, and state and trait anxiety assessed on a visual analogue scale.

(8) Social support measured by a modification of the scale of O'Reilly and Thomas[25] which was designed to evaluate social support in patients with cardiac disease. This included an assessment of general support as well as disease specific support, both day to day and during acute attacks.

(9) Life events using a validated modification for New Zealand of the life event scale of Tenant and Andrews.[26]

The instruments used in (5), (6) and (7) have previously been tested and found to be feasible, acceptable, and reliable in patients attending an asthma clinic[19] and in different ethnic groups.[27]

(10) Attitudes and beliefs about asthma using a modification of the instrument developed by Sibbald *et al*.[28] This questionnaire has also been extensively modified, consensual validity tested, and trialed in different patient groups.[20 27]

All subjects gave written informed consent to participate in the study which was approved by the Auckland Healthcare ethics committee.

STATISTICAL ANALYSIS

Data are expressed as mean (SD). The modified Attitudes and Beliefs questionnaire underwent factor analysis with orthogonal transformation. Using a cut off point of eigenvalue <0.5, a four factor solution was obtained. Factor 1 contained items related to emotional (mal) adjustment to asthma, factor 2 doctor-patient relationship, factor 3 stigmatisation, and factor 4 self efficacy. Details of the factor analysis are provided in Appendix 1. After the variables representing these factors were defined, they were combined using unit weighting to provide the factor score. Those having rapid onset attacks were compared with those whose attacks were of slow onset (comparison group). Unpaired *t* tests and the Wilcoxon signed rank test were carried out on parametric and non-parametric data, respectively. Fisher's exact test was used to test the differences in proportions between the two groups. A p value of <0.05 was regarded as statistically significant.

Results

The patients' estimates of the duration of the attack (and hence whether the attack was classified as of rapid onset) were markedly underestimated in the response to a single direct question (table 1). On the basis of a detailed description of the attack, only 27 patients (8.5%) were classified as having a rapid onset attack. All aspects of the attack were of much shorter duration in those with rapid onset asthma—that is, increased difficulty with daily activities (median) 0 (range 0–2) versus 2 (range 0–30) days (p<0.001), realisation that the attack was more severe than usual 0.5 (range 0.1–2.5) versus 2.5 (range 1–115) hours (p<0.001), and difficulty speaking or getting out of a chair 0.5 (range 0–3) versus 1 (range 0–80) hours (p<0.001), respectively.

Demographic and socioeconomic details of the two groups are shown in table 2. Men comprised 52% of the rapid onset group but only 26% of the slow onset group (p<0.005). There

Table 5 Comparison of (A) psychological factors and (B) asthma self management knowledge between rapid onset and slow onset groups

	Rapid	*Slow*	*p value*
(A) Psychological factors:			
Anxiety (HAD score ⩾10)	19%	11%	NS
Depression (HAD score ⩾10)	0%	8%	NS
Psychosocial factors recorded in hospital record based on Rea *et al*[6]	19%	39%	NS
Previous emotional counselling	27%	33%	NS
Factor scores:			
I Emotional maladjustment	−26.9 (6.8)	−28.05 (7.5)	NS
II Doctor-patient relationship	14.3 (3.5)	13.3 (4.3)	NS
III Stigma/pessimism	−2.6 (2.5)	−2.9 (2.6)	NS
IV Self-efficacy	8.7 (1.8)	7.8 (2.2)	NS
Total life events (last year)	3.5 (2.5)	3.8 (2.4)	NS
Social support available:			
General	96%	96%	NS
Asthma-specific	67%	72%	NS
(B) Self management knowledge score[19 20]:			
Slow onset scenario	14.2 (4.3)	13.3 (4.7)	NS
Rapid onset scenario	16.2 (4.3)	15.8 (4.5)	NS

was no difference in the ethnic distribution of the two groups (data not shown). There were no significant differences in the rates of family history of asthma or atopy (80% versus 74%) nor in personal history of other atopic conditions including allergic rhinosinusitis (77% versus 70%). The data suggest a greater degree of socioeconomic disadvantage in the slow onset group (table 2). Management of the index attack differed little between the two groups although those with rapid onset asthma were less likely to have presented to a general practitioner and more likely to have called emergency services ($p<0.004$; table 3A). Those with attacks of rapid onset were more likely to present with SLTA[18] but the length of stay in hospital was no different (table 3B). Although those with a rapid onset attack were more likely to have had previous SLTA (mean difference 0.95, 95% CI 0.23 to 1.67), other morbidity indices were less common compared with the slow onset group (mean difference in hospital admissions in last year 0.4 (95% CI 0.18 to 0.62), mean difference in ED visits in last year 1.3 (95% CI 0.63 to 1.97), mean difference in visits to GP in last year 3.3 (95% CI 0.95 to 5.65), mean difference in urgent visits to GP in last year 0.8 (95% CI 0.27 to 1.33); table 4). Although those with rapid onset attacks were less likely to make a serious management error, to delay intervention, or to feel "frightened and panicky", these differences were not statistically significant. There were no differences between the groups in terms of any of the psychological parameters measured (with the exception of the lower rate of caseness for anxiety in the rapid onset groups), although these were generally less common or less severe in the rapid onset group. There was no difference between the groups in terms of level of knowledge of self management (table 5).

Discussion

These data show that the estimate of attack duration based on detailed questioning is generally substantially greater than that obtained from a single direct question. As a result of the strenuous efforts made in this study to determine accurately the duration of the attack, a much smaller proportion of subjects (8.5%) was classified as having a rapid onset attack than would otherwise have been the case (34%). The patients' estimates of the duration of the attack (in response to a single question) are likely to be those recorded in the medical notes. In turn, these are likely to be shorter than the duration estimated by relatives or others (as used in mortality studies), their estimate being based on the patients' distress which is only likely to be evident during the later stages of a prolonged attack. The clinical relevance of such a distinction is that attacks of slow onset are generally considered to be identifiable, preventable,[29] and manageable along conventional lines,[21–23] while for those of rapid onset a conventional management approach may be inadequate and such patients may require additional or alternative treatment such as self administered subcutaneous adrenaline and more ready access to emergency services. Although there is no "gold standard" measure of attack duration, and while the choice of <6 hours to define a rapid onset attack was arbitrary and somewhat conservative, it was considered that an attack duration of ⩾6 hours certainly would allow sufficient time for conventional intervention strategies. Thus, the results of this study are consistent with the view that most cases of severe life threatening attacks of asthma are theoretically preventable.

The patients in this study were representative of all admissions in a region, but the proportion presenting with rapid attacks (8.5%) is much less than described previously. Arnold *et al*[30] in a prospective study of cases of acute severe asthma admitted to hospital used a similar definition of duration of attack, determined from "when the patient first perceived wheezing was worse than usual", but this information was merely obtained "from the clinical history". They found that the speed of onset of an attack was rapid (defined as less than 24 hours) in 46% of episodes and was less than one hour in 13%. As well as concerns about the assessment of duration of the attack, this study included younger patients (28% less than 14 years) and rapid onset attacks occurred more frequently in their younger patients. Although we did not find an association between age and duration of attack (independent of how the duration of attack was defined), a possible explanation is that in the current study only patients of limited age range (15–50 years) were included and information was obtained directly from the patient. Wasserfallen *et al*[16] studied a different patient group, namely those requiring intubation and mechanical ventilation for severe asthma, and found that 29% had "rapid decompensation" (defined as less than three hours). Again there is concern about how the duration of the attack was established; "a history of extremely rapid deterioration, out of a clear blue sky" leading to intubation and mechanical ventilation within three hours after the onset of the first symptoms. However, a low prevalence of rapid onset attacks (17%) was found by Martin *et al*[15] in their study of near fatal asthma in children. As already outlined, more accurate, complete, and unbiased information is likely to be available for near fatal (as opposed to fatal)

attacks of asthma and this may be the explanation for the similarity between the results of Martin *et al* and ours. By the very nature of the attack those with rapid onset asthma may be more likely to present as an asthma death. During the period of this study there were three deaths from asthma which occurred out of hospital in the 15–50 year age group in Auckland. Thus, this hospital based study is unlikely to have grossly underestimated the number of severe attacks of rapid onset occurring in the community.

If patients with rapid onset asthma are more likely to present with SLTA and thus be admitted to ICU, then it is important to consider the most appropriate comparison group. In this study we elected to compare the rapid onset group with the remainder of patients admitted to hospital with a severe exacerbation of asthma. The group admitted to hospital for acute asthma more accurately reflects the population from which the rapid onset group was derived. In doing so we have also, to some extent, controlled for asthma severity/control and the effects of hospitalisation, not done in studies in which hospital outpatients were used as the comparison group. Nevertheless, those with rapid onset asthma may still represent a group with intrinsically more severe asthma.

The demographic characteristics of the two groups were not different apart from male predominance in the rapid onset group. This contrasts with the female predominance in hospital admission data. A male predominance was also found by Wasserfallen *et al*[16] in those requiring mechanical ventilation for severe asthma. Conventional measures of recent asthma severity/morbidity such as hospital admissions, ED visits, urgent and non-urgent GP visits, and number of courses of steroids in the last year were greater in those with attacks of slow onset. Nevertheless, patients with rapid onset attacks were more likely to present with SLTA—that is, they were more likely to have had an impaired conscious level, cardiopulmonary arrest, hypercapnia, and to have required mechanical ventilation during the index attack. Furthermore, those with rapid onset attacks were more likely to have previously been admitted to an ICU with SLTA; this is consistent with the results of Kelbenback *et al*[13] and Wasserfallen *et al*.[16] This suggests that these attacks may run true to form—that is, if a patient presents with a rapid onset attack then this pattern is likely to be repeated in the future and appropriate precautions need to be taken. Thus, patients with rapid onset asthma seem to be different from others who require admission to hospital for exacerbation of asthma, which suggests that they are a distinct subgroup of patients (rather than merely the extreme end of a spectrum[13]) and who, despite good ongoing medical care, may still present with SLTA. Such patients require different and specific instructions regarding the management of acute severe attacks, including subcutaneous adrenaline and the prompt summoning of ambulance services because of their increased risk of death.[18]

It has been stated that patients with rapid onset attacks often recover very quickly,[13 31] based mainly on the ability to wean such patients off mechanical ventilation early.[16 31] In this study there was no difference in the length of time in hospital between the two groups. However, no reliable and comprehensive data were available to assess accurately the rate of normalisation of physiological parameters in the two groups, and it is likely that the total length of stay in hospital in the rapid onset group was influenced by the longer stay in the ICU and the more grossly deranged physiology on presentation (table 3B).

Previous studies have suggested that atopic asthmatic subjects may be more predisposed than non-atopic subjects to the development of rapid and fatal attacks[32 33] and that aeroallergens may be important in both sporadic attacks[34 35] and in relation to epidemics.[35 36] Although skin prick sensitivity and allergen specific IgE were not routinely measured in this study, there was no evidence that those with rapid onset attacks were more likely to be atopic than those with slow onset attacks. There were no differences in terms of other allergic manifestations such as allergic rhinosinusitis and eczema nor of family history of asthma or atopy (data not presented). Similar criteria for "atopy" were used in the study conducted by the British Thoracic Society,[32] although in that study there were considerable differences between the two groups which suggested that those with atopy had more severe disease.

Determining the precipitant of an individual attack is difficult, particularly by the time of hospital presentation. Such a determination was not a principal aim of this study but very few clear cut precipitants were identified and there was no evidence of epidemic presentation of acute severe asthma. There was no history suggestive of anaphylactic reactions. Picado[17] postulated that many sporadic cases of rapid onset severe attacks result from the ingestion of a non-steroidal anti-inflammatory drug (NSAID) and lamented the lack of these data in previous studies. None of the subjects in this study were on regular NSAIDs and only three patients had used an NSAID in the period prior to admission to hospital, all of whom had slow onset attacks. Whilst it is acknowledged that some patients may develop a rapid worsening of asthma following ingestion of NSAIDs, the results of this study suggest that few admissions are directly due to NSAIDs. This low rate probably reflects widespread recognition of this phenomenon and advice given against their use, particularly in a population who have previously been admitted to hospital.

Risk factors associated with SLTA and/or asthma death include socioeconomic factors,[37–39] quality of health care,[6 40 41] and psychological factors.[6 42 43] Data from this study suggest that those with rapid onset asthma were economically advantaged when compared with those with slow onset asthma requiring admission to hospital (table 2). Indirect measures of the quality of ongoing asthma-specific health care (table 4) were not different between

the two groups, suggesting that differences in quality of ongoing health care were not operational. Furthermore, management of the index attack was similar in the two groups and the increased use of ambulance services by the rapid onset group is entirely appropriate. Thus there was no evidence to suggest that the severity of the index attack on presentation in the rapid onset group could be attributed to deficiencies in chronic or acute management.

The results of this study provide no support for the contention of Teiramao[44] that "the duration of asthmatic prodromi (sic) may be largely dependent on the nature of the psychosocial stress factors". Despite the higher rate of previous SLTA in those with rapid onset attacks, psychological problems were neither more prevalent nor more severe in this group. This contrasts with the findings of Garden and Ayers[43] who found a higher incidence of intercurrent or past psychiatric disorders, specifically anxiety and mood disorders, in subjects with "brittle" asthma than in a group of asthmatics attending the outpatient clinic. However, this difference may have merely been due to differences in baseline asthma severity between the two groups. In our study, by comparing the rapid onset group with a group admitted to hospital with severe acute asthma, we have to a large extent controlled for "severity"[45 46]—that is, the lack of difference in psychological factors demonstrated in this study may be due to the comparison of two groups of similar "severity" and the different patterns of presentation are not important in this regard. Also, it is possible that those with "brittle" (as defined by Garden and Ayers[43]) asthma and those with rapid onset asthma represent two distinct but different subgroups of asthmatic subjects. Sibbald *et al*[28] found that patients with the highest morbidity from asthma have the greatest feelings of stigma, less positive attitudes to their doctor, and less self confidence in managing asthma attacks (self efficacy). In this study, although those with rapid onset asthma were more likely to have sustained previous SLTA and thus might be expected to be more pessimistic, have higher emotional maladaptation, and less positive attitudes to their doctor in view of a perceived lack of success in preventing severe attacks, this was not found to be the case. Campbell *et al*[47] in a cohort study of near fatal asthma reported a correlation between denial score and mode of presentation. They postulated that high levels of denial mitigate against successful management of severe asthma. However, in our study there was no evidence that those presenting with rapid onset asthma had inferior management of the index attack; indeed the trend was in the opposite direction. It is possible that the denial measured by Campbell *et al* was manifest as neglect of the early symptoms of an attack and thus led to gross underestimation of the duration of the attack.

It was not possible to determine the extent to which the rapid and slow onset groups defined in this study corresponded to the different histological patterns of mucosal inflammation,[11 12] but it would clearly be of considerable clinical benefit to be able to identify reliably those with a pattern of rapid onset asthma and at risk of subsequent SLTA in order to intervene appropriately.

Rapid onset asthma is a distinct but uncommon clinical entity with a male predominance. The clinical importance of this entity relates to the higher likelihood of SLTA and the need for alternative management strategies for acute attacks. Risk factors generally associated with asthma morbidity and mortality are no more prevalent in patients admitted to hospital with rapid onset asthma than in those with asthma of slower onset.

Appendix 1

Factor analysis:		
S10	I am worried that my asthma may interfere with the lives of the people closest to me	0.73
S8	Being asthmatic often makes me feel depressed	0.71
S15	Being asthmatic often makes me feel angry	0.69
S11	My asthma is severe	0.66
S16	I can't enjoy a full life because of my asthma	0.65
S14	I worry that I might die from asthma	0.64
S5	I feel different from other people because I am an asthmatic	0.63
S1	I worry about the long term effects of asthma on my health	0.62
S3	Even when I feel well, I worry about getting an attack of asthma	0.56
S13	Asthma makes me physically less attractive	0.51
(S17	The people who are closest to me seem overly protective of me because of my asthma	0.48)
Factor 2. Doctor-patient relationship		
S30	My doctor tells me everything I want to know about my asthma	0.82
S22	I feel understood by my doctor	0.77
S7	I have confidence in my doctor's management of my asthma	0.75
S28	My doctor has helped to make my asthma better	0.71
S18	My doctor told me what the medicines he/she prescribed would do for me	0.63
S4	I wish that my doctor talked more to me about my asthma	0.61
Factor 3. Stigma/pessimism		
S27	I avoid other people who know I am an asthmatic	0.66
S12	It embarrasses me to use asthma inhalers or other asthma medicines in public	0.61
S29	Looking towards the future, I feel certain that my asthma will get better	0.50
(S25	I feel somehow to blame for being an asthmatic	0.49)
Factor 4. Self-efficacy		
S21	I know when an asthma attack is beginning	0.65
S20	I have confidence in my ability to cope with an asthma attack	0.59
S19	I don't do anything about my asthma until it gets bad	0.50
(S6	I know what things start my asthma attacks	0.48)

Each question was scored using a 5 point Leikert scale

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