hay fever[4] and for raised IgE for common inhalation allergens at age 11.[5] It would therefore be most informative if the authors could provide additional data on the differences between the neonatal anthropometric data of the children with and without ceasarean section, and on the indications for caesarean sections themselves. This issue is of significant importance and of clinical relevance because, if indeed a causal relationship exists between mode of delivery and development of asthma, this would certainly make an argument against elective caesarean section for non-medical reasons. It would seem that there is currently insufficient evidence to infer a causal relationship, but it certainly seems worthwhile sorting this out.

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Caesarean section and asthma: alternative explanations?

In their detailed analysis of almost 3000 children followed from birth until the age of 8 years, Roduit *et al*[1] showed that children born by caesarean section have a higher risk of asthma than those born by vaginal delivery. Surprisingly, the authors offer only one explanation for this finding—namely, delayed microbial colonisation—whereas we believe other mechanisms cannot be excluded.

As an alternative hypothesis we propose to investigate the possibility of confounding by factors already present at/before birth. This hypothesis is supported by studies showing that immunological parameters in cord blood are different between children born by vaginal delivery and those born by caesarean section.[2] One such factor could be head circumference which has been repeatedly found to be related to increased IgE and the development of asthma and related disorders,[3–5] and babies born by caesarean section probably have relatively high values.

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Presence of MBL in airways: is it a disease severity marker or an additional host defence mechanism?

We welcome the paper by Fidler and colleagues reporting the presence of mannose-binding lectin (MBL) in infected airways.[1] MBL is an important acute phase protein with pro- and anti-inflammatory immunomodulatory functions.[2] The collectin family comprises surfactant protein (SP)-A, SP-D and MBL, of which the latter is mostly present in peripheral blood while the other two are mostly located in the lung.[3] We agree with Fidler *et al* that MBL might contribute to lung host defence by acting locally at the airway surface because of its similar structure to lung collectins and its presence at a physiological level in the lung. It is possible, however, that the presence of MBL in the bronchoalveolar lavage (BAL) fluid of infected children might just be a marker of lung infection or disease severity. The data of Fidler *et al* clearly show a trend suggesting that MBL was more consistently detectable in acute than in chronic diseases; this may simply be a correlate of alveolar epithelial permeability. A similar study performed by our group on HIV-infected adults showed that the levels of MBL in BAL fluid were undetectable even when present in serum. The levels of SP-D in the same study were not significantly different in lung fluid from HIV-uninfected and HIV-infected individuals with a high CD4 count (>200), but were raised in HIV-infected individuals with a low CD4 count.[4] We tested the hypothesis that levels of SP-D or MBL in HIV-infected individuals would be lower than in HIV-uninfected individuals, but this was not the case. The phenomenon that levels of defence factors are poorly associated with protection has also been shown with other defence factors such as antibodies.[5]

In conclusion, we totally agree with Fidler *et al* that future studies should focus on measuring the functional aspect of collectins. Functional assays will help to determine whether the presence of MBL in the lung acts as an additional host defence or whether it is just a marker of disease severity.

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Thoracic ultrasound: an important skill for respiratory physicians

We read with interest the article by Qureshi and colleagues describing thoracic ultrasound (TUS) characteristics for the detection of malignant pleural effusions.[1] This relatively simple bedside technique has been routinely performed by the respiratory physicians in our department in a busy general hospital for the last 4 years, resulting

Table 1 Findings on thoracic ultrasound (TUS)

Finding on TUS	No (n = 102)
Simple effusion	77
Small	31
Moderate	29
Large	17
Septae in effusion	9
Collapse/consolidation	14
Pleural thickening	4
Other findings	11

The size of the effusion on TUS was estimated to be small if <1 cm in depth, moderate if 1–3 cm in depth and large if >3 cm in depth. Other findings included mass lesions, consolidation and a raised hemidiaphragm. Some patients had more than one abnormality on TUS.

in a gradual reduction in the number of radiology departmental procedures from 63 to 17 per annum.

A recent audit of our activity showed that over a period of 18 months, 102 bedside TUS procedures were performed (table 1). The main indications for TUS included confirmation of the presence of a small pleural effusion, and guidance for pleural procedures. In 71 cases TUS confirmed the radiological findings, with discordant findings in the remaining 31 cases. The TUS findings were crucial in 30 cases, showing either an absent or a very small pleural effusion, not suitable for an invasive pleural procedure. In a further 10 patients TUS facilitated case monitoring. TUS provided guidance in all cases requiring a pleural procedure; 48 and 8 for chest drain insertion and needle aspiration, respectively. None of the subsequent procedures had any associated complications.

Use of TUS decreases the complications associated with pleural procedures,[2] which may result in serious harm or even death.[3] Recent British Thoracic Society advice on chest drain insertion has advocated the use of ultrasound image guidance.[4] Our experience confirms that TUS can be employed by respiratory physicians both as a diagnostic aid and for guiding procedures, and not just in major centres. Cost benefits also accrue from a decrease in the number of scans performed in the radiology department, and in the time involved in waiting for them. Therefore, we would support increased use of this technique by respiratory physicians, after appropriate training, for which guidance already exists.[5] The recognition of the characteristics of malignant pleural effusion as described by Qureshi and colleagues may, however, require a higher level of expertise in the interpretation of TUS findings, as well as the use of high frequency ultrasound probes.[2] This may have training implications for respiratory physicians if we wish to maximise the potential benefits of TUS in diagnosing malignant pleural effusions, rather than limit the technique to the support of diagnosis of pleural effusions and safer invasive procedures.

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Authors' reply

We would like to thank J A Kastelik *et al* for their letter in response to our recent *Thorax* publication.[1] As the letter describes, we entirely agree that thoracic ultrasound is a technique which is becoming more commonly practised by chest physicians, and there is good and increasing data in support of this technique as a sensitive diagnostic test for pleural effusion, and as the safest mode of guiding pleural intervention. We further agree with the data presented from their local audit that this chest physician-based activity may result in a decrease in radiology department activity for pleural procedures and thoracic ultrasound. This does have training implications, not only for chest physicians hoping to accrue the necessary skills to perform thoracic ultrasound as part of routine practice, but also for trainees in radiology who may no longer have the opportunity to conduct pleural procedures under ultrasound guidance.

The findings of our study suggest that thoracic ultrasound is a sensitive and specific diagnostic test in a population of patients with suspected malignant pleural effusion. It is important to note that these scans were conducted by radiologists (NQ and FVG). We believe that conducting an extensive thoracic ultrasound scan for the diagnosis of malignant pleural disease is likely to be beyond the remit of level 1-trained thoracic ultrasound practitioners.[2] Most chest physicians will use thoracic ultrasound for detection of pleural effusion and guided procedures (level 1). The features we described in our article (assessment of parietal pleural thickening, visceral thickening, nodularity and diaphragm anatomy abnormalities) may be subtle and require some experience to recognise. It is therefore likely that only radiologists and chest physicians with a high degree of experience will be in the position to assess similar criteria in the clinical situation. It is also likely that the majority of chest physicians will train to level 1 competence in thoracic ultrasound, with a fewer number (eg, those with an interest in pleural disease in specialist centres) training to level 2 competence.[2]

Although Kastelik *et al* have suggested that higher frequency ultrasound probes may be necessary to detect these abnormalities, in our study all features were visible using a 3–5 Hz curvilinear abdominal probe.[1] We initially used a higher frequency probe to assess for subtle thickening and nodularity, but in fact abandoned its use for the study as it led to no increase in diagnostic yield and substantially increased scanning time.

Once again, we would like to thank the authors of the letter for their interest in our study.

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