apposition of the cords can occur normally during quiet sleep in young lambs.³ Briefly, this 'braking' of expiratory airflow by the larynx generates positive subglottic pressure of up to 10 cm water and is an important mechanism in preventing lung collapse in the young until a noncollapsible chest wall is established. If this expiratory laryngeal pressure is prevented, such as by a tracheotomy or endotracheal intubation, respiratory frequency falls and respiratory drive is greatly diminished especially during quiet sleep. Equally, it can be reasoned, if an overdistensible trachea exists or develops, respiratory drive could diminish, and failure or 'unexplained death' occur during respiratory challenge—such as with an apparently trivial infection or upper airways obstruction during sleep. That is to say, positive expiratory pressure could not be developed if the upper airways simply dilated with glottic adduction.

What subglottic pressures are generated in respiratory disease in the young appears to be uncertain. From our studies in early postnatal life we would expect tracheal distension during the expiratory cycle (rather than obstruction) in quiet sleep but such airway collapse could occur in rapid eye movement sleep when upper airway expiratory resistance is slight.

Thus a hypothesis to be tested could be an association between vocal cord necrosis, indicative of chronic laryngeal adduction in expiration, and a distended trachea in these cases. The information from this comprehensive series of necropsies could provide valuable evidence which could point the way to a functional evaluation in such infants possibly averting later morbidity and mortality. Surely a morbid anatomist's dream!

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- 3 Johnson P. Comparative aspects of the control of breathing during development. In: von Euler C, Lagercrantz H, eds. *Central nervous control mechanisms in breathing*. Oxford: Pergamon Press, 1979: 337–52.

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Professor Emery and Dr Wailoo comment:

We were very well aware of most of the points raised by Paul Johnson but did not wish to speculate too far beyond our evidence.

The possibility of spasmodic glottic closure being a major factor in unexpected death is one that we have been studying for many years but unfortunately, as with so many things related to cot death, these vocal cord lesions also occur in children who die in hospital from apparently clinically justifiable disease.¹ It would be possible, and

nice, to do the anatomical correlations that Johnson suggests but the nonspecificity of the vocal cord lesions would be likely to lead to an inconclusive result.

Our own interest has progressed from the general statistical conclusions to the possibility that there is a particular area of tracheomalacia at the carina which enables the trachea to 'block off' at this point and that infection is either absent or present at the laryngeal conus.

It would be of interest to us if Paul Johnson could give his findings regarding the presence or absence of laryngeal lesions indicating spasm in his lambs!

Reference

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Gluten intolerance, gluten enteropathy, and coeliac disease

Sir,

I read with interest the article by Dodge.¹ His enlarged concept of gluten-induced 'disease' is attractive, original, and may be correct, but his proposal for omitting the second of the present three biopsies recommended by the European Society for Paediatric Gastroenterology and Nutrition (ESPGAN)² seems inopportune and open to criticism.

Most coeliac children put on a gluten-free diet will present a normal jejunal mucosa from as soon as a few months up to 1–2 years after introduction of the diet and if they do not one suspects incomplete gluten withdrawal.³ Though some biochemical, haematological, or immunological data can be of help in the follow-up of coeliac children as well as in the assessment of the degree of adherence to the gluten-free diet, a normal jejunal mucosa is the most reliable finding. So, omitting the second biopsy not only fails to confirm the second step to a final diagnosis of gluten-induced enteropathy, but also loses a valuable way of assessing the strictness of dietary treatment.

The criteria of Interlaken,² including 'the 2-year rule', may be too rigid, but nevertheless they fit with most coeliac children (95%)⁴ and in our experience they contribute to an increased accuracy in the diagnosis of coeliac disease.

Until a more complete knowledge is reached concerning permanent, transient (?),⁵ or latent (?)⁶ gluten enteropathy or gluten intolerance any escape from the ESPGAN criteria seems undesirable and will confuse rather than help to clarify the diagnosis and management of children with coeliac disease.