Professor Whitehead replies as follows:

It is not easy to comment on the letter of Drs T Cooke and J Matthews as they do not give a reference to their work. They apparently used a method for a study of adenomatous colonic polyps by harvesting cells by cytological brushing, which is not comparable with our work. They showed that 18% were aneuploid and expressed "surprise" that we, in our study, did not find aneuploidy in 16 adenomatous polyps. In our discussion it is made quite plain that the conclusion that aneuploidy in polyps does not occur had only a 75–90% chance of being correct. Furthermore, Drs Cooke and Matthews do not seem to be aware that even frank carcinomas may be diploid. They also assume that the degree of dysplasia in polyps must parallel the degree of cell division activity. This is not necessarily true. The fact that adenomatous polyps and carcinomas may have a profile indicating minimal cell division activity bears this out. The reference to differences in our findings to those of Cuvelier *et al* is irrelevant.

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Eosinophilic gastroenteritis

I read with interest the review article by Blackshaw and Levison on eosinophilic gastroenteritis,¹ but I am unhappy about the coverage of parasitic infections, which I think is misleading.

Concerning anisakiasis as a cause of eosinophilic gastroenteritis, the designation of the parasite as "*Eustoma rotundatum*" was abandoned long ago,² and the invasive larvae belong to the genus *Anisakis*. Semantics apart, the purported British cases of 1964³ were not associated with any helminths, and thus there is no evidence to suggest that either of them were, indeed, anisakiasis. The first autochthonous British cases were reported in 1979⁴ and 1985.⁵ At the moment, anisakiasis is regarded as the result of actual tissue invasion by larvae rather than the passage through the gastrointestinal tract of the larvae, and therefore a diagnosis of anisakiasis requires the detection of larva or fragments thereof. In the future serodiagnosis may be of help.⁶

I am surprised that giardiasis is mentioned as a cause of prominent gut eosinophilia.

This is not my experience when *Giardia lamblia* is the sole parasite present, and it is not described in standard texts or in the description of purported invasive giardiasis.⁷

One protozoon that is, however, associated with eosinophilic enteritis is *Sarcocystis* spp.⁸ This comes from eating meat containing *Sarcocystis* cysts, whereby man can become the definitive host to the sexual phase in the intestinal mucosa. This may produce an acute enteritis with eosinophilia of the lamina propria. How common the condition is remains uncertain, partly because the parasites are so small they can be readily overlooked.

Hookworm infection may reasonably be diagnosed histologically only if the worms are seen rather than from "erosions or circular channels." Notably, recorded cases of invasive ankylostomiasis are rare.⁹

Other helminths not mentioned in the review are well described as producing eosinophilic gastroenteritis, including oesophagostomiasis and angiostrongyliasis.^{10,11} In the United Kingdom invasive enterobiasis happens occasionally, with eosinophilic abscesses in the large bowel wall and serosa (personal observations). More important is the omission of strongyloidiasis as infection with *Strongyloides stercoralis* may be life threatening. Some cases are diagnosed from intestinal biopsy, where a dense mucosal eosinophilia associated with invasive larvae is seen.¹⁰

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⁹ Piza JE, Biagi F. Localisation of *Ancylostoma duodenale* within the intestinal wall. *Parasitologia* 1963;5:189–92.

¹⁰ Marcial-Rojas RA. *Pathology of protozoal and helminthic diseases*. Baltimore: Williams and Wilkins, 1971.

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Doctors Levison and Blackshaw reply as follows:

We welcome the letter: it reflects Dr Lucas's unique experience in the United Kingdom in parasitology through the London School of Hygiene and Tropical Medicine, and adds considerably to what we said about parasitic infestations.¹ Our review was written principally for pathologists practising in the "West" and we therefore opted to emphasise and contrast in the first part of the article "inflammatory fibroid polyp" and "eosinophilic gastroenteritis" and to devote the second part of the article to other gut lesions with eosinophilia. In the second part of the article we devoted proportionately more space to lymphomas and histiocytosis X than some other lesions, as we have some special experience in these areas, and perhaps our coverage of parasitic lesions was consequently sketchier than might have been.

We are grateful to Dr Lucas for his update on the terminology of *Anisakis* infections, for providing references to the "first autochthonous British cases," and for referencing other parasitic causes of eosinophilic enteritis—several of which we freely admit to never having seen personally. As Dr Lucas does rather strongly reprimand our omission of *Strongyloides stercoralis* we should like to add to his comment that we have seen several examples of this parasite in the bowel without prominent mucosal eosinophilia. We agree that *Giardia lamblia* is not often associated with prominent gut eosinophilia, but such eosinophilia is recorded in what we consider to be a "standard text".² Also, "erosions or circular channels" are considered by the same author to be helpful pointers towards the diagnosis of hookworm infection.²

Apart from these two minor matters of disagreement, we fully accept the points made by Dr Lucas and are very pleased with such a constructive response, broadening, it does, the originally intended scope of the article.

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New method or new application of a method?

In a thorough evaluation of a morphometric technique for the assessment of pulmonary arteries Fernie and Lamb claim that the method they use entailing a computer assisted planimeter is new.¹

The concept of using the internal elastic lamina as a reference for the true size of an artery was pioneered in Britain by Cook and Yates in 1972.² Work, which extended this concept to evaluate the intima and also used computer assisted planimetry, was presented by myself at the summer meeting of the Pathological Society of Great Britain and Ireland in Edinburgh in 1983.³ A further detailed description of the method was presented at the winter meeting of the society in January 1984⁴ and was subsequently published in the *Journal of Clinical Pathology*.⁵

I would suggest that the paper of Fernie and Lamb represents the application of an established method to a specific problem. While the methodology may be new to the authors working on pulmonary vessels, its advantages and ease of use come as no surprise to those who have already used it to evaluate arteries in other sites.

When the authors presented their work at the Pathological Society summer meeting in 1984 I expressed this point of view and suggested to them that the method was not new. I feel that the word "new" when applied to a method should mean what it says.

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Drs Fernie and Lamb reply as follows:

We do not claim any originality with regard to the concept behind our measuring technique, which is the use of the length of the internal elastic lamina as an indicator of artery size. In the first¹ of our series of papers we refer to the work of Cook and Yates,² who pioneered this technique in Britain, following description of the technique by Furuyama.³

On the issue of computer assisted morphometry, such measuring techniques are new to those engaged in studies of the pulmonary vasculature as we pointed out to Dr Lowe when we presented our work at the summer meeting of the Pathological Society of Great Britain and Ireland in 1984.⁴ Furthermore, we consider that a measuring technique is incomplete if it is not applied to a range of subjects with a view to determining how the data should be analysed and how subjects should be compared. As such an approach has not been adopted in previous studies of the pulmonary vasculature we feel that our paper⁵ makes a new and useful contribution in this area.

Lastly, with regard to Dr Lowe's suggestion that "the paper of Fernie and Lamb represents the application of an established method to a specific problem" we would question whether such methods are truly established. It is interesting that our paper⁵ was rejected by a reputable British pathology Journal in July 1984 on the grounds that there were "queries concerning the fundamental validity of the technique."

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Fine needle aspiration of thyroid: confusion v. subsequent histology

The review article published in your January 1985 issue¹ has stimulated us to report some additional data of fine needle aspiration of thyroid performed in the department of pathology at Athens University.

During the past few years Greek physicians have increasingly accepted percutaneous, fine needle aspiration in the diagnosis of thyroid nodules. According to many authors, fine needle aspiration is the best procedure currently available for selecting and directing treatment for thyroid nodules.²⁻⁶ In our institution 320 thyroid nodules (1.5-4 cm in diameter) have been studied by fine needle aspiration since October 1984. Of these, 12 cases were identified as papillary carcinoma, seven as follicular tumours, two as medullary carcinoma, five as unclassified tumours, two as Hurthle cell tumours, one as lymphoma, and 17 as "suspected neoplasia." All of the above cases underwent operative treatment by means of the cytology; in addition, 35 patients were clinically selected for surgical biopsy of the nodule despite the fact that the cytological diagnosis of fine needle aspiration was negative.

The needling was performed with a 22 gauge needle and occasionally with a 20 gauge needle. When no adequate material had been obtained by the first needling, the procedure was repeated. Double sampling was also performed if the nodule was greater than 2.5 cm in diameter. The procedure was well accepted by most patients, and no appreciable complications or side effects were observed, as in other series.^{3,7} Miller, however, noted that an intranodular hematoma can occur, which may be palpated by the physician.

When we examined histologically the 55 surgical specimens of biopsied nodules most showed intranodular bleeding. In two of these cases it was so extensive that we could not precisely define the histological pattern of the lesion. Moreover, it was impossible to