

Letters to the Editor

An analysis of blood specimen container leakage

Since the study which we recently reported (*Journal of Clinical Pathology*, 1978, **31**, 888) we have modified the fluorescein test for leakage from closed containers in order to take account of the rheological differences between aqueous fluid and blood. The reagent that we now use consists of 10 volumes of blood in 1.5 volumes of ACD, to which are added 25 g/l of sodium fluorescein (uranine). A sufficient volume of this is pipetted into the bottom of the container in order to cover the capped section when it is inverted. After capping securely, the container is immersed upside down into a tube containing water, and it is left at 37°C for two hours and then at room temperature (20 ± 2°C) for a further two hours. The container is removed from the water, which is then examined by ultraviolet light for any traces of fluoresceinate as an indication of leakage.

This modified test is more relevant than the aqueous leakage test for blood specimen containers. Using this modification, we have found no leakage in batches of containers of several different types which we had previously reported as unsatisfactory. This does not, of course, affect the observations on spontaneous discharge, which remains a serious problem with all types of container.

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Histological comparison of adenomatous and hyperplastic parathyroid glands

We were interested to read the two papers by Dr Lawrence (*Journal of Clinical Pathology*, **31**, 626, 1978 and *British Medical Journal*, **II**, 92, 1978). In the first paper he argues against the concept of tertiary hyperparathyroidism and says that 'it is doubtful if a clear cut distinction between secondary and tertiary hyperparathyroidism can be made'. A figure (Fig. 4) is used to support this argument and is labelled as secondary hyperparathyroidism. An apparently identical figure was used to illustrate a case of tertiary hyperparathyroidism in the *British Medi-*

cal Journal. The comment in that paper says that 'the histological features of the parathyroid glands supported the diagnosis of the true tertiary hyperparathyroidism'. Thus it appears that the whole concept of tertiary hyperparathyroidism is in a muddle yet again.

St. Goar (1963), who introduced the term tertiary hyperparathyroidism, believes it should be abandoned (St. Goar, 1978). He classes secondary hyperparathyroidism as A and B. A is mild or reversible where there is hyperplasia of four glands; B is severe with hyperplasia of four glands with or without a superimposed adenoma. It seems a logical classification but it does acknowledge the presence of adenomas superimposed on hyperplastic parathyroids.

We feel the problem could be solved by going back to first principles. An adenoma is a benign tumour, which is usually round and well circumscribed, has a fibrous capsule, and produces pressure atrophy in the adjacent parenchyma. The presence of pressure atrophy in the surrounding epithelium is a particularly important feature. We have seen several patients in whom all these criteria are fulfilled.

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References

- St. Goar, W. T. (1963). In Massachusetts General Hospital, Case records, 29-1963. *New England Journal of Medicine*, **268**, 949 and 953.
- St. Goar, W. T. (1978). In Massachusetts General Hospital, Case records, 4-1978. *New England Journal of Medicine*, **298**, 271.

The author comments as follows:

Certainly the same case is discussed in both papers. However, although Fig. 4 is labelled secondary hyperparathyroidism in the *J. Clin. Path.* paper the discussion that goes with it indicates that it was tempting to suggest that this was an autonomous adenoma developing in an established case of secondary hyperparathyroidism (ie, tertiary hyperparathyroidism). The more clinically orientated

paper in the *British Medical Journal* is more definite about the role of histology in differentiating secondary and tertiary hyperparathyroidism. I agree that this is open to criticism and that the last sentence of that paper is somewhat contentious. There is probably no such entity as a histologically proved adenoma.

Regarding the use of first principles in diagnosing an adenoma, I believe it is clear from my analysis and from the reports of others that, in practice, it is very difficult to apply these criteria successfully. When stating 'we have seen several patients in whom all these criteria are fulfilled'—are Hasleton *et al.* referring to adenomas in 'tertiary' hyperparathyroidism? If so, they should publish their findings.

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Effect of bacterial flora on staphylococcal colonisation of the newborn

In reference to the article by Speck *et al.* (*Journal of Clinical Pathology*, 1978, **31**, 153-155) it was 'noted that the concept of seeding with non-pathogens to prevent colonisation with disease-causing microorganisms is not new'. 'The present data suggest that in the newborn there is interference between *Staph. aureus* and *Staph. epidermidis* as well as between either staphylococcus and Gram-negative bacteria. These findings lead one to consider the attractive possibility of using the natural body flora to control colonisation and combat infection. . . However, the present data indicate that this concept should be applied to a new site.' Before one embarks on bacterial interference with 502A staphylococcus, one should be familiar with the complications that can occur. These complications were outlined in a review article (Houck, P. W., Nelson, J. D., and Kay, J. L. (1972). Fatal septicemia due to *Staphylococcus aureus* 502A; a report of a case and review of the infectious complications of bacterial interference programs. *American Journal of Diseases of Children*, **123**, 45).

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