Fine needle biopsy: effect of needle size.

Sir,—In their recent leading article, Hall-Craggs and Lees¹ stated that no single study comparing the diagnostic yield of different needle sizes was available. This is no longer correct.

In a study of percutaneous pancreatic biopsy, Dickey et al.² reported on the sensitivity rate in pancreatic carcinoma using 22 and 20 gauge needles. The respective rates were 71.9% and 86.7%. The overall diagnostic rates for all types of pancreatic lesions were 61.8% and 73.3%. A 19 gauge sheathed needle was used for lesions with fluid or necrotic debris where a pseudocyst was a possibility. The overall diagnostic accuracy of the 19 gauge needle was 81.8%. No case of pancreatic carcinoma was biopsied using the 19 needle.

Furthermore, several studies have attested to the value of combining cytology with histology in the diagnosis of hepatic malignancy.³⁻⁶ These reports have indicated that in 12–15% of cases the cytological specimen alone is positive.

Fine needle aspiration is of particular value in the identification of hepatic metastases. Large needle biopsy has a success rate in metastatic liver disease ranging from 23–78% but the higher percentages apply only to cases with extensive liver involvement.⁷ In a study by Conn and Yesner, where involvement by hepatic metastases was ‘minimal’, double liver biopsy using a Vim-Silverman needle yielded positive results in only 20% of cases. In a small personal series of cases of metastatic liver carcinoma corresponding to Conn and Yesner’s ‘minimal’ involvement group, ultrasound guided fine needle aspiration yielded positive results in 72% of cases (unpublished observations). The vast majority of false negatives occurred at the very beginning of the series. There were no false positives.

Clearly fine needle aspiration has a definite role to play in the diagnosis of malignancy, either alone, or in combination with large needle biopsy.

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References
2 Dickey JE, Haaga JR, Stellato TA, Schutz CL, Hau T.

Effect of sulphasalazine and its metabolites on the generation of reactive oxygen species

Sir,—I read with interest the above paper by Miyachi et al.,¹ and I applaud the authors on their thoroughness, but I felt the paper overcomplicated the issue. Neutrophils only produce one reactive oxygen species and that is superoxide anion which is formed via the action of an oxidase enzyme which is present within their cell surface membrane.² Hydrogen peroxide and the hydroxyl radical are not produced directly by the neutrophil, but as a result of further reactions which superoxide may participate in once generated (dismutation, Fenton and the Haber-Weiss reaction).³

Therefore, logically it would be expected that any agent which inhibits neutrophil superoxide production also inhibits hydroxyl and hydrogen peroxide production as they are dependent on the former. I therefore find it surprising that the authors show sulphapyridine as inhibiting neutrophil superoxide production but not affecting hydrogen peroxide or hydroxyl radical production, and likewise that 5 amino-salicylic acid (5-ASA) inhibits hydrogen peroxide and hydroxyl radical generation but does not affect superoxide production. Although the latter could be explained by postulating that 5-ASA interferes with the reactions that occur after superoxide release.

It has recently been shown that sulphasalazine at a concentration of 1 mM will inhibit the formyl peptide (F-Met-Leu-Phe) induced stimulation of neutrophil superoxide release, but not 4 b-N-horbol 12 myristate 13-acetate (PMA) induced stimulation of superoxide production.⁴ Implying that sulphasalazine is exerting its inhibitory effect at the level of the neutrophil.

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surface membrane as FMLP interacts with a surface receptor but PMA interacts with an intracellular receptor to activate protein kinase C which stimulates the oxidase enzyme.

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References


Reply

sir,—I wish to thank Dr Andrew Williams for his interesting and constructive comments on my recent paper. I agree with his opinion that the agent that inhibits neutrophil derived superoxide production also logically inhibits hydroxyl radical and hydrogen peroxide generation. In our experiments, SP mildly inhibited superoxide generation in the neutrophil system and also showed inhibitory trends in the xanthine-xanthine oxidase system, although the latter was insignificant. If SP had an SOD like activity, it will raise the level of hydrogen peroxide as we have previously reported. In this case, it is not unlikely that SP inhibits superoxide production slightly without apparently affecting hydrogen peroxide or hydroxyl radical levels, because the expected slight reduction in these levels induced by the mild suppression of superoxide production may be masked. Another possible explanation for this dissociation is a time gap between each assay being done, although this is less likely.

As mentioned in the paper, these agents seem to affect the oxygen metabolism of neutrophils with somewhat different and probably multiple modes of action, which makes the issue complicated.

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Teeth and benign oesophageal stricture

sir,—Maxton et al (*Gut* 1987; 28: 61–3) have found that benign oesophageal strictures occur more frequently in a group of edentulous patients attending for endoscopy than in a control group with normal dentition. They suggest that edentulous patients eat less solid food than controls and the lower oesophagus is therefore subject to less dilatation with a greater tendency to stricture.

An alternative explanation, however, is that the edentulous patients because of poor masticatory function, chew solid food less efficiently and therefore swallow larger boluses which are more likely to obstruct their strictures. In this way they have more symptoms of dysphagia and thus present earlier for dilatation. The authors have assessed neither the dietary intake or masticatory abilities of their patient groups, and whilst other papers have suggested that edentulous patients eat less solid food than ‘normal’, these factors have not been established in this study.

We would agree that patients with strictures should be urged to use dentures, not primarily to reduce stricture formation but to encourage better chewing and therefore less bolus impaction and dysphagia.

The discrepancy between severe symptomatic oesophagitis and the relatively symptom free patients who appear to develop stricture does not invalidate oesophagitis as a cause of stricture formation. It is possible that those patients who are symptom free have the most severe reflux oesophagitis precisely because they are unaware of their oesophageal damage and do not present until a stricture has formed.

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Reply

sir,—Drs Tait and McKinlay add another interesting suggestion for the cause of the increased prevalence of benign oesophageal strictures in the elderly with few or no natural teeth.

Because of their less efficient chewing, however, edentulous patients tend to choose softer, less solid food rather than eating larger poorly masticated boluses of more solid food. Thus, nuts and tough meat are rarely eaten by edentulous patients. We are