Letters to the Editor

Failure to demonstrate specificity of the morphological and histochemical changes in mucosa adjacent to colonic carcinoma

The failure of Isaacson and Attwood (1979) is to appreciate that transitional mucosa has been defined by us as a specific combination of histochemical and morphological changes. We, too, have found sialomucins in solitary ulcers, but the morphology is quite different from that of transitional mucosa, as their illustration shows. We, too, have found transitional mucosa with squamous and melanotic carcinomas of the anal canal (Greaves et al., unpublished observations) but this in no way invalidates our hypothesis that transitional mucosa represents prepolypoid adenomatous neoplasia. On the contrary, it fortifies the concept since the anal canal is simply developing its own characteristic neoplasms. Furthermore, when we find transitional mucosa in a colostomy we expect to learn that the patient has had a rectal carcinoma removed, yet Isaacson and Attwood do not tell us about their cases. They would not be surprised to find an adenomatous polyp developing in the remaining bowel after a cancer had been excised, and we regard the anal canal equally part of the same field of action of putative carcinogens. Moreover, the secretory changes in transitional mucosa are not only quantitative, with increased sialomucins, but qualitative, with variable proportions of different types of sialic acids showing values between normal controls and tumours (Dawson et al., 1978; Rogers et al., 1978).

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The authors have commented as follows:
Contrary to the assertion of Branfoot and Felipe, we fully appreciate their definition of transitional mucosa, and our 'failure' remains that of demonstrating its specificity as a premalignant change in colorectal mucosa. Mucosal morphology in solitary ulcer syndrome is quite variable, and our initial illustration of this was chosen to show the similarity to transitional mucosa (Figure). The editors of 'The Journal', however, requested the substitution of a more characteristic illustration. Both are from the same section. We are gratified to learn that Branfoot and Felipe have noted similar histochemical changes in solitary ulcer syndrome and have confirmed our findings with respect to anal melanomas and squamous cell carcinomas. The suggestion that the association of transitional mucosa with these anal tumours (and colonic lymphoma) reinforces the concept that it is a premalignant change is an original one but is not borne out by any statistical relationship between these various tumours, nor by analogy with other well-defined precancerous conditions of the colon, such as polyposis coli or ulcerative colitis. We cannot comment on the qualitative secretory changes adduced to by Branfoot and Felipe since these have not been sought in transitional mucosa not related to adenocarcinoma.

Our suggestion that transitional mucosa represents a secondary phenomenon, perhaps due to mechanical factors, is further supported by the recent paper of Rhatigan and Saffos (1979), who describe transitional mucosa in association with diverticular disease of the colon. Until more substantive evidence is produced we remain unconvinced that the changes characterised as transitional mucosa are premalignant, and still less that they represent 'adenomatous neoplasia'.

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References


Figure Mucosa from a case of solitary ulcer syndrome showing deepened branching crypts with enlarged goblet cells. Haematoxylin and eosin × 40.

Interpretation of serum total calcium

I have been involved previously, albeit in a very minor role, in the continuing saga of the 'corrected', or as it is now known 'adjusted' calcium (Sanderson, 1974). Once again, as a district hospital biochemist I find it difficult to know just who to believe. This time my dilemma concerns the precision of serum total calcium determinations required for diagnostic purposes.

Dr Payne and his colleagues, in their latest contribution to the subject (Payne

References