LETTERS TO
THE EDITOR

Radioisotope determination of regional colonic transit in severe constipation

EDITOR.—We were interested to see the article by van der Sijp et al (Gut 1993; 34: 402–8) on a scintigraphic method of regional colonic transit measurement compared with the conventional radiological method. There are, however, several points that we would like to raise. They have dismissed the method using methacrylate coated capsules as unnecessary and resulting in inaccuracy when using a capsule. We were particularly interested to view the activity in the small and large bowel in the fasting state. It is of limited value, however, if there is fast gastric emptying. Although this is not important in patients with severe chronic constipation, it is a problem when dealing with patients with normal or fast transit (such as some patients with inflammatory bowel disease). The method of methacrylate coating of the capsule, however, is important and in our preliminary studies using capsules with several coatings, there was a delay of release in two of 10 patients. As we have modified the method of coating by reducing the number of coatings to two only in one of 130 patients studied so far has the capsule failed to open before reaching the ascending colon. One should aim for capsule release somewhere in the small intestine, having passed intact through the stomach, as activity tends to collect in the terminal ileum before its release into the colon. In our experiment we focused on obtaining activity before transit in the terminal ileum.

A fundamental problem arises in the use of ‘centre of mass’ (COM) to describe colonic transit and to compare the two methods of studying transit. COM is useful for examining groups of patients with colonic disorders. The radiologic and scintigraphic methods seem to be different from each other in this study as only the COM is used to compare groups for both methods. It is of limited value, however, in reporting and treating individual patients, especially when a full range of colonic motility disorders is under study. COM ignores all detailed information obtained by the scintigraphic method, which is essential for correct classification of different patterns of colonic movement. This is best achieved using parametric images.

A final point is the activity given for these investigations. We have found on a four day study sufficient to identify all patterns of colonic motility disorders even in those with severe constipation. The shorter study allows us to use a smaller 111In activity (2 MBq) and still obtain good image quality, reducing the whole body irradiation even further than that described by van der Sijp.

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Reply

EDITORS.—I am interested in the comments by Dr Notghi and his colleagues about our paper on colonic transit. Even though about half of our severely constipated patients have slow gastric emptying we did not find overlap of the small and large intestine to be a problem (in constipated patients or healthy controls).

I believe that a pH sensitive capsule is satisfactory if the goal is only to study colonic transit. Many patients with disturbances of colonic motility, however, have a disorder that affects much of the gut. The use of a coated capsule precludes the gathering of detailed information about upper gut motility. The use of a radiolabelled meal, however, allows the assessment of upper and lower gut transit during the same study.

The ‘centre of mass’ measurement was just one of the methods we used to describe colonic transit. It provides a useful single numerical guide to the effectiveness of transit. We also described in detail, however, the quantitation of transit through each colonic region.

In some patients with delayed colonic transit we have found a four day study inadequate for assessing transit through the left colon. Any reduction in radiolabelled dose, however, without the loss of information should be welcomed.

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Polysaturated fatty acid pattern and fish oil treatment in inflammatory bowel disease

EDITOR.—We read with great interest the paper by M Estève-Comas et al (Gut 1992; 33: 1365–9). The authors claim that patients with inflammatory bowel disease have different plasma fatty acid patterns by comparison with controls, mainly characterised by an increase in n3 series and a decrease in some n6 series (20:3 n6) and that these differences are more pronounced, in an inversely proportional manner, accordingly to the severity of the disease. These data suggest an increased polysaturated fatty acid biosynthesis and consumption in active inflammatory bowel disease, especially of the n3 series, raising some doubts on the use of high doses of fish oil in the treatment of acute inflammatory bowel disease. These results, however, need to be carefully considered. The authors did not separate the different lipid fractions containing the plasma fatty acids (phospholipids, triacylglycerols and free fatty acids) by thin layer chromatography. This would have been of great benefit as the main source for the eicosanoids’ synthesis is the fatty acid stored in the phospholipids and also because each fraction has different representation of every single fatty acid. Moreover, V Schacky et al have shown that the variability in the plasma fatty acid pattern is enormous and that the alimentary habit and the quality of lipid intake can modify the pattern in a few hours. For this reason an overnight fast is certainly insufficient to guarantee the stability of the plasma fatty acid pattern.

We recently quoted the plasma phospholipid fatty acid profiles in a group of Crohn’s disease patients in comparison with a group of healthy controls and we did not find any significant differences. On the contrary we studied, in the same groups, the phospholipid fatty acid profiles in red blood cell membranes and we found remarkable differences: a significant decrease in all polysaturated fatty acids and a significant increase in the main saturated fatty acids (palmitic and stearic acids). Actually, the fatty acid profile in red blood cell phospholipid membranes is much more stable; six to eight weeks of a very high dose of n3 fatty acid supplementation (fish oil) is needed to modify its composition and occurs during cell formation and not in the same inflammatory bowel disease, it should be noted that the incorporation of n3 fatty acid in the neutrophil membranes occurs by the replacing of arachidonic acid, the main source for LTB4 production and one of the most powerful inflammatory mediators. Our findings would suggest that the authors’ results could have different interpretations.

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Polyunsaturated fatty acid pattern and fish oil treatment in inflammatory bowel disease

EDITOR.—We read the interesting comments by Dr Belluzzi et al regarding our paper on polyunsaturated fatty acids in inflammatory bowel disease. In the main source for the eicosanoids’ synthesis is the fatty acid stored in the phospholipids and also because each fraction has different representation of every single fatty acid. Moreover, V Schacky et al have shown that the variability in the plasma fatty acid pattern is enormous and that the alimentary habit and the quality of lipid intake can modify the pattern in a few hours. For this reason an overnight fast is certainly insufficient to guarantee the stability of the plasma fatty acid pattern.

We recently quoted the plasma phospholipid fatty acid profiles in a group of Crohn’s disease patients in comparison with a group of healthy controls and we did not find any significant differences. On the contrary we studied, in the same groups, the phospholipid fatty acid profiles in red blood cell membranes and we found remarkable differences: a significant decrease in all polysaturated fatty acids and a significant increase in the main saturated fatty acids (palmitic and stearic acids). Actually, the fatty acid profile in red blood cell phospholipid membranes is much more stable; six to eight weeks of a very high dose of n3 fatty acid supplementation (fish oil) is needed to modify its composition and occurs during cell formation.

Regarding the use of fish oil in active inflammatory bowel disease, it should be noted that the incorporation of n3 fatty acid in the neutrophil membranes occurs by the replacing of arachidonic acid, the main source for LTB4 production and one of the most powerful inflammatory mediators. Our findings would suggest that the authors’ results could have different interpretations.