similar. Moreover, it has been known for several years that these bacterial protein toxins are also structurally similar to ricin and abrin. These plant toxins, which are, respectively, present in seeds of the castor bean plant (Ricinus communis) and the jequirity bean (Abrus precatorius), are not closely related to each other. There is compelling evidence that the toxic effect of all these toxins is mediated by an identical mechanism.

The toxin molecules consist of an A subunit and one or more B subunits. After binding of the B subunit(s) to a cell surface receptor, the A subunit enters the cell, where it catalyses an adenine residue from ribosomal RNA molecules. The end result is that protein synthesis stops and the affected cells die.

This mechanism almost certainly explains the diarrhoea (with or without rectal bleeding) that follows ingestion of castor or jequirity beans, the colonic inflammation that occurs in S dysenteriae type 1 and E coli O157:H7 infections, and the endothelial cell damage that accompanies the haemolytic uraemic syndrome. The traditional equation of bacterial haemorrhagic colitis with bacterial invasion of the mucosa is clearly an over-simplification. At least in the case of E coli O157:H7 and S dysenteriae type 1 the important issue is the production of a cytotoxic toxin, irrespective of whether the bacteria invade the mucosa.

To date ricin achieved its greatest notoriety in the 1978 'umbrella murder' of the Bulgarian broadcaster Georgi Markov. It is intriguing that the systemic effect of ricin in this unfortunate episode can be directly compared with the pathophysiology of bacterial colitis.

Finally, polymerase chain reaction amplification of E coli O157:H7 DNA encoding part of the cytotoxic toxin molecule may facilitate the detection of this bacterium in clinical specimens and in contaminated food.

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Unethical research relating to Helicobacter pylori

EDITOR.—We are writing concerning the paper by Vincent et al (Gut 1994; 35: 313-6) regarding the prevalence of Helicobacter pylori infection in cohabiting children. In that study, the authors examined the prevalence of H pylori infection among mentally retarded children residing in an institution. H pylori infection was confirmed by gastroscope. 'Informed consent' was obtained from the families.

We believe that this study raises important ethical questions and concerns. Research involving children always raises the 'ethical flag' and the use of invasive procedures requires a very critical look at the risk versus benefit obtained. In most instances invasive procedures will be deemed to be unjustified in asymptomatic children. The problem is somewhat easier if you are dealing with a population of symptomatic children, but even in that group the motivation and clinical practices of a group reporting the results of invasive procedures for diagnosis must be questioned.

This study describes endoscopic investigations in asymptomatic mentally retarded children. It is extremely difficult to imagine any benefit that the children could have gained from participation in the study. We are hard pressed to come up with any possible justification for doing the study. Hammerschmidt and Gross note that when a journal accepts a paper for publication after peer review that 'acceptance constitutes at least a subtle, if not overt, [no objection] of the nihil obstat [official licence to print]. Authority of the journal has been placed behind the paper announcing that it has been subjected to scrutiny and has been found to be without fault in its truth, novelty, and potential importance.' There are two questions: firstly, should the study have been done at all and secondly should it have been accepted for publication?

We recognise that these concerns are serious, but they must be considered and considered now as the role of H pylori in the paediatric population is coming under increasing scrutiny.

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Reply

EDITOR.—Ethical aspects regarding our paper (Gut 1994; 35: 313-6) were asked by one of the reviewers, and the necessary answers were given. Study protocol, diagnostic test, and the results were detailed in the first version of our paper, but was shortened afterwards according to the reviewers' recommendations. Finally, only epidemiological and microbiological aspects were described in this article.

We agree that such a study should not be done only for epidemiological purposes. Our study took place in a clinical context with therapeutic concerns at the first instance, three cases of H pylori endoscopic gastritis were found in this institution and then treated with clinical ameliorations. These three children had endoscopy because of suspected oesophagitis and macroscopic nodules were seen in the antrum. This led us to perform biopsies, which showed the presence of H pylori gastritis. Specific H pylori treatment was given because we could not distinguish the potential role of the infection in the upper gastrointestinal complaints of these patients.

In severely neurologically impaired children, gastro-oesophageal reflux is common. In our experience, 20-50% of these children suffer from gastro-oesophageal reflux. If not controlled in time, the disease can evolve and lead to severe complications such as oesophageal stenosis or Barrett's oesophagus. In these children, gastro-oesophageal reflux is often associated with other disorders (nutritional, metabolic, respiratory, urological, or digestive diseases). It is widely admitted that gastro-oesophageal reflux is difficult to recognise on clinical grounds in mentally retarded children.
Unethical research relating to Helicobacter pylori?

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