interference with the potassium method on the DT60 analyzer but a 38% decrease was seen with lipaemic serum.

The exact reason for the difference between interference by Intralipid and lipaemic sewn samples is unknown; differences in light scattering properties of the lipids in the two different matrices could account for the observed differences.

Thus the standard way of assessing lipaemic interference (that is, the addition of Intralipid to serum) may not be appropriate for assessing lipaemic interference in all cases. We are not aware of any previous studies addressing this important issue; it is clear that further studies evaluating the different effects of lipaemic serum samples and Intralipid on other analysers are necessary.

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Campylobacter colonisation, duodenal ulceration, and changes in gastric mucosa

There is an increasing amount of evidence to support the association between *Campylobacter pylori* and antral gastritis in patients with or without duodenal ulceration.[1] So far, however, we have seen no reports of a comparative study of the oxyntic mucosa in these two conditions. We therefore collected biopsy specimens from 20 patients with and 30 patients without duodenal ulceration from the fundus, the greater curvature of the body, the lesser curvature of the antrum and the prepyloric area of the stomach for histological examination and culture. Culture was carried out as described previously.[2] *C pylori* was isolated from the fundus, the body and the antral mucosa of the 20 patients with duodenal ulceration, and of 20 of the patients with gastritis but without duodenal ulceration. *C pylori* was not isolated from the other 10 patients (tables 1 and 2). *C pylori* was associated with severe gastritis in 96% of specimens from the antral and prepyloric mucosa, and 40% of specimens from the body and fundal regions in patients with and without duodenal ulceration. In most of the patients with duodenal ulceration the histology of the oxyntic mucosa was normal, or only showed mild gastritis. In the 20 patients with gastritis, *C pylori*, but without duodenal ulceration, however, oxyntic mucosa showed gastritis in 64% of the biopsy specimens from the body and the fundus ($\chi^2 = 18{\cdot}66$; $p < 0{\cdot}001$). Furthermore, the antral mucosa of patients with and without duodenal ulceration showed similar histological changes ($\chi^2 = 3{\cdot}40$). In conclusion, the observed differences may be due to colonisation by different strains of *C pylori*, or they may be the consequence of different mechanisms of host parasite interactions, or both.

Table 1 *Histological findings in oxyntic mucosa of patients with Campylobacter pylori with and without duodenal ulceration*

Histology of oxyntic mucosa	Patients with C pylori			
	With duodenal ulceration		Without duodenal ulceration	
	Body	Fundus	Body	Fundus
Chronic gastritis	2	4	14	11
Mild gastritis	7	6	2	1
Normal	9	10	4	7
Total	18*	20	20	19*

*Three superficial sections could not be classified.

Table 2 *Histological findings in antral mucosa of patients with Campylobacter pylori with and without duodenal ulceration*

Histology of antral mucosa	Patients with C pylori			
	With duodenal ulceration		Without duodenal ulceration	
	Prepyloric region	Antrum	Prepyloric region	Antrum
Chronic gastritis	20	20	18	19
Borderline gastritis	0	0	2	1
Normal	0	0	0	0
Total	20	20	20	20

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Laboratory infection with parvovirus B19

A survey of clinical laboratory staff[1] has implicated occupational exposure as a probable cause of infection with hepatitis B, tuberculosis, shigella, salmonella, pseudocholera, and streptococcus. We have observed seven probable laboratory infections with human parvovirus B19 (table) and wish to draw attention to this hazard. Though it is impossible to say conclusively that these