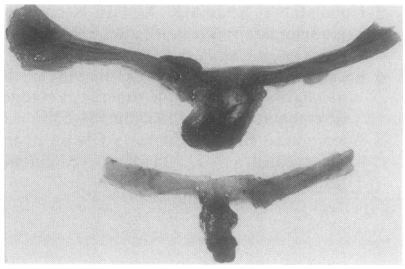

Figure *Diverticula from right colon.*

patient who had a subtotal colectomy was mucosal ulceration in at least one diverticulum; blood had, in fact, been noted in some of the diverticula when blocks were taken. This must also be the explanation in the other case even though no definite ulceration was seen in the diverticula sampled.

Perry and Morson[1] suggested that diverticular disease confined to the right colon should be regarded as a distinct form of diverticulosis, to be differentiated from classic left sided disease, from the more extensive disease that also affects the transverse colon, and from solitary diverticula. The findings in these two cases support this view, and it is noteworthy that one of the two cases described by Perry and Morson presented with profuse rectal haemorrhage. Right sided diverticular disease should not be overlooked as a possible cause of unexplained bowel haemorrhage.

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Reference

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Lipaemic interference: effects of lipaemic serum and intralipid

Lipaemia interferes with a variety of clinical chemistry methods.[1] Most studies evaluating the effect of lipaemia use Intralipid 20% to simulate the turbidity effects of increased triglyceride concentrations. In reality, any interference with testing is likely to be the result of chylomicron accumulation in serum. Whether the interference from Intralipid is comparable with the interference seen in lipaemic serum samples is unknown. We carried out a study to compare the interfering effects of lipaemic serum samples and lipaemia simulated by Intralipid on various common tests on desk top analysers.

The instruments evaluated were the Abbott Vision (Abbott Laboratories, North Chicago, Illinois, USA), the Reflotron analyzer (Boehringer Mannheim Canada, Inc, Dorval, Quebec), the Kodak DT60 analyzer (Eastman Kodak Co, Rochester, New York, USA) and the Ames Seralyzer (Ames Division, Miles Laboratories Elkhart).

The Vision uses individual test packs into which capillary tubes with whole blood, plasma, or serum are inserted. The blood is spun down inside the analyser and mixed with reagents. The absorbance is read by a photometer and the result calculated and printed. Tests evaluated were glucose, urate, alkaline phosphatase, urea nitrogen, and cholesterol.

The Reflotron analyser is a photometer controlled by a microprocessor that uses test strips. The strip—which accepts blood, serum, or plasma—is inserted into the analyser and the coloured product of the reaction is measured by the photometer. The tests evaluated were glucose, urea nitrogen, cholesterol and δ-glutamyltransferase.

The Kodak DT60 analyser uses special slides for performance of tests on serum or plasma. All reactions take place within the multilayered elements of the slide. The bar code reader in the analyser automatically identifies the test to be performed; all processes are controlled by a selfcontained microprocessor. The tests evaluated were glucose, urate, potassium, bilirubin, creatinine, and amylase.

The Seralyzer is a reflectance photometer and uses test strips. For each test a different test module is inserted into the analyser. This module indentifies the test measurements, processes the reflectance signals from the test strip, and calculates the result. The test strips use serum or plasma. The tests evaluated were glucose, creatinine, aspartate aminotransferase, theophylline, phenytoin, and phenobarbital.

Simulation of turbidity was done by adding 10% Intralipid (Cutter Laboratories, Berkeley, California, USA) to pooled sera with known concentrations of anlayte. A triglyceride concentration of 3·4 mmol/l was obtained. Concentrations of all analytes tested on each of the instruments were measured.

To test the effect of lipaemic sera, blood was drawn from normal volunteers one hour after ingestion of 600 ml of 18% butterfat ice cream. Serum was separated and used for studies testing interference by "lipaemic serum". To obtain the same target concentration of triglycerides (3·4 mmol/l) for the lipaemic serum, it was appropriately diluted with aqueous assayed material that had known concentrations of analytes to be tested. Concentrations of all analytes were then measured on the above mixture. To assess the degree of interference, lipaemic serum samples and Intralipid were then added to plasma based quality control material with known concentrations of analyte. The final concentration of triglycerides was 3·4 mmol/l. Interference was considered to be important when the apparent change in the concentration of analyte exceeded the daily imprecision of the particular method for each anlayser.[2-5]

The results are summarised in the table. Each test was done in duplicate. It is clear from these results that at a given triglyceride concentration (3·4 mmol/l in this case) the interference for some tests can be different depending on whether the triglyceride is present in Intralipid or in lipaemic serum samples. For example, Intralipid showed no

Table *Percentage change in analyte concentrations with intralipid and lipaemic serum at a triglyceride concentration of 3·4 mmol/l.*

Instrument and tests	*Intralipid*	*Lipaemic serum samples*
Vision:		
Glucose	NI	NI
Urate	+43%	+16%
Alkaline phosphatase	NI	NI
Urea nitrogen	+15%	NI
Cholesterol	NI	NI
Reflotron:		
Glucose	NI	NI
Urea nitrogen	+15%	+12%
Cholesterol	NI	NI
γ-Glutamyltransferase	NI	NI
DT-60:		
Potassium	NI	−38%
Glucose	NI	NI
Amylase	−20%	−48%
Urate	NI	−9%
Bilirubin	NI	NI
Creatinine	NI	NI
Ames:		
Glucose	NI	−13%
Creatinine	NI	NI
Aspartate aminotransferase	NI	NI
Theophylline	NI	NI
Phenytoin	NI	NI
Phenobarbital	NI	+11%

NI—no interference.

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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

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