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

Table *Laboratory infection with parvovirus B19*

Laboratory	*Age*	*Sex*	*Grade*	*Seroconversion*	*IgM*	*Clinical details*
London, England	30	Female	Scientist	Yes	Yes	Malaise and myalgia for two weeks starting four weeks after first laboratory exposure, then transient rash (< 12 hours) and arthralgia. Myalgia and malaise persisted for a further four weeks
Paris, France[2]	31	Female	Technician	Yes	Yes	Vascular purpura on legs; 8 weeks pregnant—miscarriage
	23	Female	Technician	Yes	Yes	No symptoms
	26	Female	Technician	Yes	Yes	Transient rash
Munich, FRG[3]	26	Female	Technician	Yes	Yes	Malaise and myalgia for two weeks then rash and arthralgia for three weeks, and headaches for four weeks starting after the first week of the rash
Fukuoka, Japan	31	Male	Medical	Non tested	Yes	Fever (38·2°C) two weeks after first laboratory exposure, followed by rash (three weeks) and arthralgia (four weeks)
Washington, USA	30	Male	Medical	Yes	Yes	B19 DNA in serum, fever, arthralgia of knees, ankles, wrists, and lower back lasting three to four days. Neutropenia, reticulocytopenia

infections were occupationally acquired the timing of the illnesses strongly suggests this. We propose three measures that might reduce the occupational risk from B19 virus.

Firstly, inactivate the antigen. Diagnostic tests for B19 virus infection are at present done with antigen prepared from the plasma of viraemic patients and this material has not been inactivated until now.[4] Initial studies done by one of us (BJC) indicate that doses of up to 2·4 Mrads gamma irradiation do not destroy the antigenicity of B19 virus. This treatment destroyed the infectivity of 30 viruses.[5] Inactivation of current supplies of B19 antigen by irradiation is therefore probably feasible. In the near future it is likely that a non-infectious B19 antigen will be provided by recombinant DNA technology.[6]

Secondly, keep aerosols in a safety cabinet. B19 virus has been transmitted experimentally by the respiratory route,[7] and it seems likely that this was the route of spread for the probable laboratory infections reported here. Infectious aerosols could have been generated by centrifugation, during the resuspension of virus pellets, or in washing stages of solid phase immunoassays. Where possible such procedures should be done in an exhaust protective cabinet.

Thirdly, find out if the laboratory personnel are immune. It may not be necessary to restrict work with parvovirus B19 to those known to be immune, but a strong case can be made for finding out if staff are immune, and excluding pregnant women. Exposure to B19 virus during pregnancy may carry an increased risk of abortion (Hall SM, Anderson MJ, Caul EO, *et al.* Paper presented at the joint meeting of the European Association against Virus Diseases, Switzerland, 1987).

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Sarcoma-like nodules in cystic ovarian tumours

We read with interest the recent report concerning sarcoma-like mural nodules in cystic serous ovarian tumours, and noted particularly the second case where undifferentiate sarcoma was found arising within serous cystadenocarcinoma.[1] We have recently encountered a similar tumour which occurred in a 44 year old premenopausal multiparous woman with a history of neurofibromatosis.

The patient presented with a few months' history of abdominal discomfort. At laparotomy she was found to have bilateral ovarian masses, and widespread pelvic and abdominal metastases with ascites. Hysterectomy and bilateral salpingo-oophorectomy were carried out and the patient started on chemotherapy.

Each ovary was replaced by a cyst, the larger measuring 23 cm in diameter. On histological examination these showed moderately differentiated serous cystadeno-