The protective effect of urea, which has free access through the outer membrane, is probably due to the fact that the urease of *H pylori* is localised intracellularly as well as in the outer membrane, and is not greatly affected by the morphological changes of the outer membrane.9 The susceptibility of the pleomorphic four day cultures to acid can not be attributed to any single bacterial form. Nevertheless, as a group, these cultures were unusually susceptible to hostile conditions.7

In summary, we found that the morphological changes which occur with longer incubation of *H pylori* have an inverse relation with the organism’s resistance to an acidic environment. This observation suggests that older bacteria are less resistant to certain adverse conditions. The finding that the addition of urea to the medium almost reversed this phenomenon emphasises again the central role of urea in the survival of *H pylori* in acidic conditions. It also suggests that the morphological changes which occur with longer incubation are not associated with obvious changes in the urease activity of the bacteria.


**Bacteraemia caused by *Campylobacter* spp**

C Ladrón de Guevara, J Gonzalez, P Peña

**Abstract**

The genus *Campylobacter* has become increasingly recognised as the cause of various infections. *Campylobacter jejuni* and *C coli* cause acute gastroenteritis in man all over the world. *C jejuni* enteritis can lead to bacteraemia, but its actual incidence remains unknown.

Seven cases of bacteraemia caused by *C jejuni* or *C coli* are reported, from the blood of seven patients: five immune deficient adults; a newborn baby; and a patient who had had abdominal surgery. Patients who develop diarrhoea as a result of *Campylobacter* infection are at risk of bacteraemia thereafter.

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**Case reports**

**CASE 1**

A 2 day old baby with jaundice caused by an isoimmunisation with the isoantibody anti-A had diarrhoea with mucus and blood. The clinical picture was of septicaemia, but examination yielded normal results. *C coli* was recovered from stool and blood cultures after 48 hours of incubation. Erythromycin (1000 mg orally a day) was given, and the patient’s symptoms resolved.

**CASE 2**

A 41 year old man with a long history of alcohol related disease, including jaundice and other features of chronic liver disease, was admitted with diarrhoea and fever. Two of three blood cultures grew *C jejuni* after 72 hours of incubation. Fecal culture was also positive for this organism. Erythromycin, to which the bacterium was sensitive, was given and the patient was apprized after 24 hours. Thereafter, he had only mild diarrhoea which finished on the fourth day.

**CASE 3**

A 33 year old man was admitted to hospital with abdominal pain, fever, and increased
leucocytes, but there was no diarrhoea. Appendicitis was diagnosed. Surgery was required, and this was complicated by enteric fistula. After blood samples had been taken for culture gentamicin and clindamycin were given. One of three blood cultures grew C jejuni after 72 hours of incubation. Faecal culture was not carried out. After four weeks of treatment the patient was discharged.

CASE 4
A 61 year old woman, diagnosed as having colonic adenocarcinoma, was admitted to hospital after five days of fever, but without diarrhoea. Three blood cultures were taken in the emergency room. Three days later, C coli was identified in the three blood cultures. Ciprofloxacin was given intravenously.

CASE 5
A 15 year old haemophilic boy was admitted to hospital because of fever and abdominal pain associated with diarrhoea. A faecal sample and four blood samples were cultured. One of the four blood cultures grew C jejuni after 24 hours of incubation, and the faecal culture was also positive for this organism. The patient responded well to 12 days' treatment with erythromycin given orally.

CASES 6 AND 7
Two men of 29 and 36 years old, each with a history of intravenous drug misuse and HIV infection with severe immunosuppression, were admitted to hospital. Seven and 10 days beforehand, they had had fever and diarrhoea respectively. Blood cultures grew C jejuni, and faecal cultures were negative in one patient.

An automatic BACTEC 660 system was used. Blood was cultured in NR16B and NR17A bottles and examined daily. When a positive BACTEC signal (threshold 30) was obtained, the samples were cultured on tryptic soy agar with 5% sheep blood and chocolate agar IsoVitaleX (Becton Dickinson) at 35°C under microaerophilic conditions (5% CO₂, 95% air, 53% humidity). The strains were isolated from 16B aerobic bottles, but not from 17A anaerobic bottles. All strains were observed between 24 and 72 hours incubation directly from the blood culture bottle using Gram stain. They were Gram negative, motile, spiral or curved rods.

Faecal samples were cultured by methods that included culture for Campylobacter spp on Skirrow's blood agar, which contains vancomycin (10 mg/l), trimethoprim (5 mg/l), and polymyxin B (2500 IU/l) (Oxoid); and Preston campylobacter blood free medium with cefoperazone (32 mg/l) (Oxoid). The media were incubated at 43°C for 24 hours in a microaerophilic atmosphere (5% O₂, 10% CO₂) provided by microaerophilic systems (Campy Pak Plus BBL).

The suspected strains were examined for oxidase reaction, and tested for in vitro sensitivity to nalidixic acid and hippurate. In vitro, these strains were susceptible to erythromycin, cephaporphins, and trimethoprim sulphamethoxazole. C jejuni is now recognised as a major cause of both endemic and epidemic gastroenteritis. Unlike the closely related organism Campylobacter fetus, C jejuni is not frequently associated with bacteraemia. C jejuni enteritis may lead to bacteraemia, but its actual incidence remains unknown. C fetus intestinalis is an opportunistic organism that infects adults, often elderly, debilitated patients, causing systemic illness, including bacteraemia. Similar cases of localised infections due to C fetus subspecies intestinalis secondary to bacteraemia, have been reported: patients with arthritis; pericarditis; and receiving continuous ambulatory peritoneal dialysis.

Strains of C fetus are highly resistant to the bactericidal activity of normal human serum. In contrast, C jejuni and C coli are said to be susceptible to the antibacterial activity of serum.

In a study by Tee et al only 0·7% of patients with C jejuni diarrhoea had bacteraemia. This may have been due to the fact that many strains of C jejuni appear to be serum susceptible. This susceptibility seems to require normal concentrations of complement and preformed antibody in the serum. Immunocompromised patients who lack the capacity to respond with the production of antibody specific for C jejuni are susceptible to infection with this organism.

The strains of Campylobacter isolated from the blood of the patient with appendicitis complicated by enteric fistula, and the patient with colonic adenocarcinoma probably follow on from transmural migration of intestinal bacteria after changes in the intestinal wall. C jejuni and bacteraemia can occur in immune deficient patients and patients with changes in the intestinal wall with a minimum of symptoms. Five out of seven of our cases with bacteraemia caused by Campylobacter spp had previous or concomitant diarrhoea attributable to the same micro-organism. This suggests that Campylobacter spp penetrate through the intestinal mucosa in the absence of changes in the intestinal wall.

Patients who develop diarrhoea as a result of Campylobacter spp infection are at risk of bacteraemia from this organism.

Bacteraemia caused by Campylobacter spp.

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