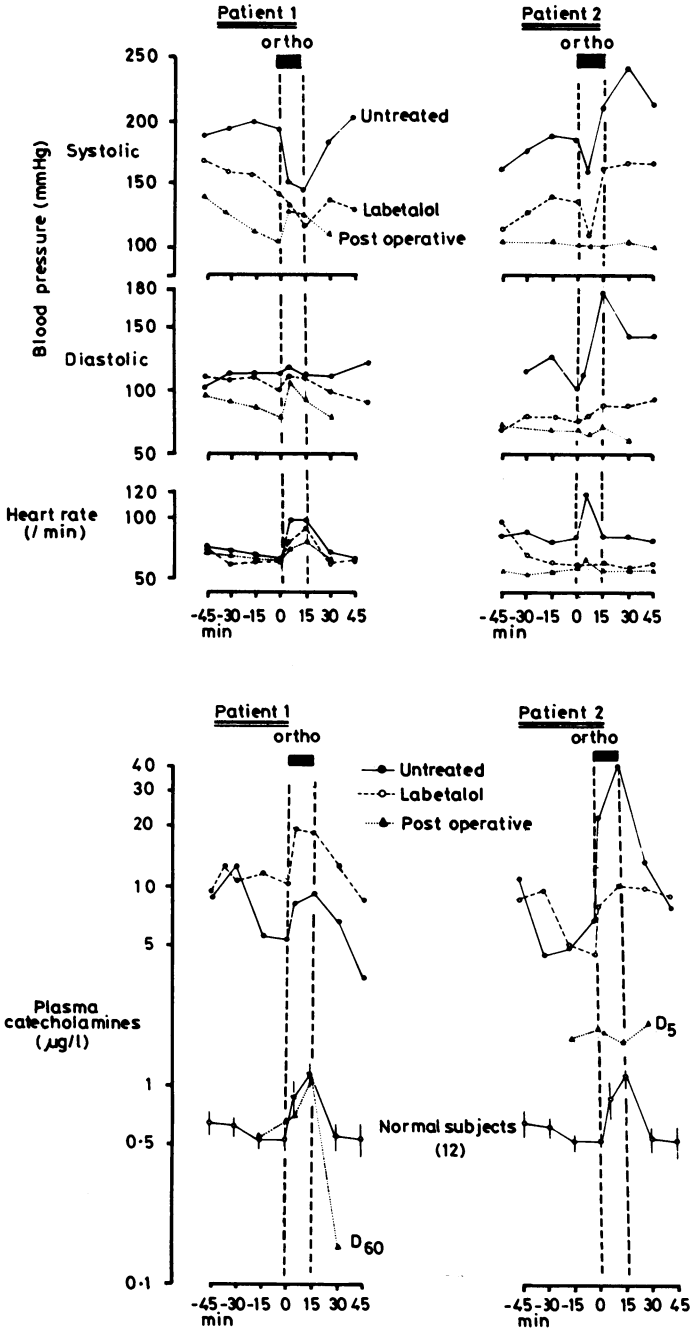
patient 2 labetalol 50 mg did not improve hypertension: on the contrary, the blood pressure rose transiently. The next day it fell to normal and remained there for four hours after labetalol 100 mg. During the next eight days the blood pressure was kept at normal with labetalol 100 mg six times a day. The average blood pressure was 144/98 mm Hg. In patient 3 intravenous infusion of labetalol 50 mg over three hours rapidly brought the blood pressure to normal. Labetalol 100 mg by mouth was given one hour before stopping the infusion. The blood pressure was then restored to normal for six days with only 100 mg labetalol twice a day, the average blood pressure during that time was 134/82 mm Hg.

Orthostatic systolic hypotension was of a similar magnitude in patients 1 and 2 before and during treatment (figure). Labetalol also lowered the acceleration of heart rate during orthostasis. It had no effect on plasma catecholamine concentrations, which remained about 10 times higher than in the controls. This shows that the effect of labetalol on blood pressure was not a chance one.



Blood pressure, heart rate, and plasma catecholamine concentrations in two patients (1 and 2) in recumbency and during orthostatic tests (ortho) when untreated (o ——— o), when taking labetalol (o ——— o), and postoperatively (△ ——— △). Plasma catecholamine concentrations (mean ± SEM) in 12 healthy controls also shown.

Conversion: Traditional to SI units—
Catecholamines (as adrenaline): 1 µg/l ≈ 5·0 µmol/l.

Comment

None of our patients had a sustained hypertensive response to labetalol, as has been reported in one case.[2] Only in patient 2 was a 50-mg dose followed by transient hypertension. A subsequent 100-mg dose lowered the blood pressure. This sequence would be consistent with the hypothesis that an inadequate alpha-adrenergic blockade by too low a dose of the drug aggravates hypertension. Labetalol is indeed much more potent as a beta- than an alpha-adrenoreceptor blocking agent.[4] Finally, the blood pressure was restored to normal with small doses of the drug. In all the patients headaches and sweating disappeared, there were no side effects, and orthostatic hypotension was not aggravated. We therefore think that labetalol could be useful in the preoperative management of patients with phaeochromocytoma provided the blood pressure was carefully monitored, especially when starting treatment.

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Campylobacter jejuni/coli meningitis in a neonate

Campylobacter fetus is a rare cause of various human infections, including, occasionally, meningitis.[1] Campylobacters of the *jejuni/coli* group have been recognised recently as an important cause of gastroenteritis.[2] There have been no reports so far, however, of meningitis caused by these organisms, although earlier confusion about the taxonomy of the genus may mean that a few of the cases attributed to *C fetus* were in fact *C jejuni/coli* infections. We describe the first clearly defined case.

Case report

A 12-day-old boy presented with a 20-hour history of lethargy and refusing feeds. Gestation had been uncomplicated, vaginal delivery at term was normal, and birth weight was 3·3 kg. The baby had been discharged home, breast-feeding well, after 48 hours. On readmission he was drowsy, hypotonic, mildly dehydrated, jaundiced (serum bilirubin 200 µmol/l (12 mg/100 ml)), and febrile (37·8°C). His anterior fontanelle was normal. The peripheral blood showed a leucocytosis of $41{\cdot}6 \times 10^9$/l (41 600/mm³) with 95% neutrophils. Lumbar puncture yielded turbid cerebrospinal fluid (CSF) containing leucocytes $1{\cdot}092 \times 10^9$/l (1092/mm³) with 64% lymphocytes and 36% neutrophils, and raised protein (>2 g/l) and low glucose concentrations (0·1 mmol/l (1·8 mg/100 ml)). Blood glucose was 5·3 mmol/l (95·4 mg/100 ml). No organisms were seen in the Gram-stained deposit. Penicillin G (200 mg/kg/day), chloramphenicol (50 mg/kg/day), and cloxacillin (100 mg/kg/day) were given intravenously with clinical response. Culture of the CSF yielded a scanty growth of spiral and S-shaped Gram-negative bacilli resembling campylobacters 24 hours later. Treatment was then modified to chloramphenicol (50 mg/kg/day for 13 days) and gentamicin (6 mg/kg/day for seven days) intravenously. A blood culture taken on admission subsequently proved to be sterile. The baby remained well and was discharged seven days after all antibiotic treatment had been discontinued.

The organism was microaerophilic and grew well on campylobacter medium at 43°C and 37°C but not at 25°C. It was oxidase positive and catalase positive, reduced nitrate to nitrite, did not ferment sugars or produce

hydrogen sulphide, and was Voges-Proskaeur negative. On disc diffusion testing it was sensitive to nalidixic acid, gentamicin, tetracycline, chloramphenicol, erythromycin, and metronidazole and resistant to trimethoprim, sulphamethoxazole, cephradine, penicillin, colistin, and vancomycin. It was consistently non-motile from first isolation. Electron microscopy revealed a structure similar to that described for *C jejuni/coli*[3] except that no flagella were seen. The organism was confirmed as *C jejuni/coli*.

Although the baby had never had diarrhoea, on specific questionning both his mother and a sister were found to have had an episode lasting two to three days six weeks before the delivery. They were not investigated bacteriologically at that time. Campylobacters were not isolated from two faecal specimens taken from the patient within three days of the onset of his illness, or from single samples taken during the same period from his parents and twin sisters (aged 4 years). Cultures of his mother's milk were also negative for campylobacters. Serum taken four weeks after the onset of the baby's symptoms showed indirect fluorescent antibody titres[4] of 1/32 (baby) and 1/256 (mother) against the patient's organism. Testing the antibody against specific anti-human immunoglobulins indicated that it was predominantly IgG. The baby's total serum immunoglobulin concentrations at this time were: IgG 10·41 g/l (normal range at this age[5] 2·50–8·50 g/l), IgA $0{\cdot}94 \times 10^{-2}$ g/l (normal range $5\text{-}30 \times 10^{-2}$ g/l), and IgM $4{\cdot}45 \times 10^{-2}$ g/l (normal range $20\text{-}70 \times 10^{-2}$ g/l).

Comment

This case illustrates some unusual aspects of *C jejuni/coli* infection—namely, the absence of diarrhoea in the baby, the localisation of infection in the meninges, and the non-flagellated nature of the isolate. The absence of bottle feeding excludes proprietary milk as a source of infection. The mother's high antibody titre suggests that her diarrhoea was caused by *C jejuni/coli* and that she may have been the source. We cannot define the nature and timing of transmission—whether transplacental, during delivery, in breast milk, or otherwise. Total IgA and IgM concentrations were lower than normal at six weeks. The slightly raised IgG probably reflects maternal antibody. Perhaps a degree of immune deficiency allowed invasive infection to develop while passively transferred specific IgG was responsible for its mild and localised nature.

We thank Dr Martin Skirrow for confirming the identity of the isolate.

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Significance of nocturnal abdominal pain: a prospective study

Standard medical texts, journals, and volumes on gastrointestinal disease either imply or indicate that abdominal pain that wakes the sufferer at night is a strong indication of organic as distinct from functional gastrointestinal disease.[1–3] This statement appears not to be based on scientific studies, but it is perpetuated in both undergraduate and postgraduate medical teaching. Hislop[4] found that 32 out of 64 patients with colonic spasm had nocturnal pain but he does not state if they were woken by it. Horrocks and De Dombal,[5] in a computer-aided study, found that 32% of persons with functional bowel disease were woken by pain compared with 70% of sufferers from duodenal ulcer. We did a prospective study to determine the significance of nocturnal, waking abdominal pain as a marker of organic or non-organic gastrointestinal disease.

Patients, methods, and results

One hundred and one outpatients presenting with undiagnosed abdominal pain were questioned at personal interview by a physician. Their answers were entered on a designed protocol which included a detailed analysis of nature of the pain, sleep pattern, incidence of nocturnal pain, and other relevant symptoms. The completed protocols were detached from the case records before investigations were begun and were analysed at the end of the study. Knowledge of a specific disease on presentation excluded patients from the study. Investigations in all patients included blood picture, erythrocyte sedimentation rate, serum biochemistry, and contrast radiographs of the upper and lower gastrointestinal tract. Upper gastrointestinal tract endoscopy was performed in 63% of cases. At completion of the study patients were divided into those with organic disease (group 1) and those with functional disease (group 2). Observations from the two groups were tabulated into two-dimensional contingency tables and analysed using the chi-squared test. The frequency of night pain and of pain waking the patient were analysed by the Mann-Whitney U test.

Group 1 comprised 22 men and 17 women (mean age 48 years) with predominantly pyloroduodenal ulceration (26) and gastric ulceration (8). Other conditions included cholelithiasis (2), Crohn's ileitis (1), viral enteritis (1), and chronic pancreatitis (1). Group 2 comprised 62 patients (36 men, 26 women, mean age 41 years) with functional disease diagnosed by excluding organic disease. The distribution of pain was almost exclusively upper abdominal in group 1. In group 2 it was upper abdominal pain in two-thirds and lower abdominal in one-third. Duration of symptoms and episodes of pain, marital status, and country of origin were similar in both groups. Nocturnal pain waking the sufferer was recorded in 71% of patients with organic disease but also in 58% of those with functional disease (table). The difference was not statistically significant. There was no significant difference between the two groups in the number of times the pain woke the patient each night. The number of nights of nocturnal pain per week was also similar. Careful analysis of sleep pattern showed no significant difference between the two groups in usual time of retiring, hours before falling asleep, hours of sleep, and hour of final awakening. Abdominal pain was more closely associated with food in group 1 and with bowel habit in group 2 ($p<0{\cdot}01$). Response to antacids was favourable in group 1 ($p<0{\cdot}01$).

Differences in incidence and nature of abdominal pain in patients with organic disease (group 1) and patients with functional disease (group 2)

Pain	Group 1 (n = 39)	Group 2 (n = 62)	Significance of difference (χ^2 test)
Nocturnal	28 (71%)	36 (58%)	NS*
Relation to bowel habit:			
Before	2	22	
After	1	2	$p<0{\cdot}01$
Nil	36	38	
Response to antacids:			
Good	23	13	
Mild	1	3	$p<0{\cdot}01$
Poor	3	14	
Relation to food:			
Before	4	4	
After	22	18	$p<0{\cdot}01$
Nil	8	35	

*Not significant.

Comment

Abdominal pain that woke the patient was more common with organic disease than functional disease, but the difference was not statistically significant. Hence nocturnal pain which wakes the patient is not, by itself, a distinguishing feature of organic disease.

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