hypertension admitted at the same time because the latter tended to be admitted for other reasons, including vascular complications of hypertension, which might be related to smoking. The lack of association between hypertension and cigarette smoking is well documented,[21] but inclusion of control cases with end organ damage might have shown a different trend. The small reduction in cigarette smoking in the United Kingdom over the past 10 years cannot account for the observed excess of smoking in patients with malignant hypertension, which has been confirmed elsewhere.[14-16] We were unable to confirm the link of smoking with grades III and IIIa retinopathy, although smoking was slightly more common than in control patients. Other authors usually combined their patients with grade III and IV retinopathy in similar analyses.

The prognosis of malignant hypertension, if left untreated, is worse than any form of cancer, with up to 90% of patients dead in one year.[12] Controlling blood pressure prolongs life,[22] and long term follow up studies have reported a dramatic reduction in mortality.[10] The results of treatment in our patients were disappointing, but this may be because our cases dated back to 1960, when effective antihypertensive treatment was not so enthusiastically prescribed. Furthermore, this series includes all patients seen in a district general hospital, some of whom were acutely ill and died within hours or days of admission.

The Keith Wagener classification with its gradation of retinopathy from one to four suggests a gradation of risk that is not borne out by our findings. The clinical features of hypertension in patients with grades III and IV retinopathy are similar and quite distinct from those seen in patients without retinopathy. Although the severity of hypertension and quality of control of blood pressure with antihypertensive drugs are both potent predictors of risk,[23] malignant hypertension seems to carry a particularly bad prognosis, probably related to renal damage due to fibrinoid necrosis of intrarenal arterioles. If control of blood pressure had been better mortality might have been further reduced. The Keith Wagener classification of hypertensive retinopathy, however, seems particularly unhelpful as grade III and IV do not differ, so we suggest that is should be abandoned. Malignant and accelerated hypertension are one and the same disease, which should be regarded as a medical emergency requiring accurate long term control of blood pressure.

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SHORT REPORTS

Diclofenac sodium versus pethidine in acute renal colic

Experimental work suggests that prostaglandins play an important part in producing pain in renal colic, [1 2] and studies have suggested that anti-prostaglandins may be of value in the management of this condition.[3] We designed this trial to compare the efficacy of and tolerance to diclofenac sodium, a potent inhibitor of prostaglandin synthetase, with those of the commonly prescribed narcotic pethidine in the management of acute renal colic.

Patients, methods, and results

Patients presenting to the accident and emergency department with severe pain and thought to have acute renal colic were entered into this double blind study, being randomised to receive an intramuscular injection of either pethidine 100 mg or diclofenac sodium 75 mg. We excluded those already taking non-steroidal anti-inflammatory agents; those with a history of allergies, asthma, peptic ulceration, or renal insufficiency; and those who had been given strong analgesics by their general practitioner before their admission.

On entering the study the patient assessed his pain on a scale from 0 to 5 (0=no pain at all; 5=the most severe pain imaginable). The pain score was recorded every 15 minutes for the next hour and then at hourly intervals. Treatment was regarded as successful if the pain score improved by three points or more. A second injection of the same drug was offered after 30 minutes if the first had not been successful or if pain had returned. If pain persisted at one hour or returned thereafter patients were given pethidine 100 mg intramuscularly. Side effects attributable to the treatment were recorded by the patient or the medical staff. An urgent intravenous urogram was obtained in all patients and was normal in 12, who were therefore withdrawn from the study.

Altogether 58 patients were evaluated (41 men, 17 women), their ages ranging from 19 to 85 years (mean 46 years). Thirty received diclofenac sodium and 28 pethidine. Both groups were comparable for weight, sex, age, and the site and size of the stone. Of the 30 patients who received diclofenac sodium, 28 (93%) obtained satisfactory relief of pain after a single injection, compared with only 18 of the 28 (65%) who received pethidine ($p<0.05$, Fisher's exact test) (table).

Pain relief after single injection (figures are numbers of patients)

	Satisfactory (≥3 point improvement)	Mild (1-2 point improvement)	None
Diclofenac sodium	28	2	
Pethidine	18	5	5

Thus 12 patients required a second injection 30 minutes later. All had severe pain at that time, though seven reported transient moderate relief of pain after the first injection. No patient required further analgesia at one hour.

Side effects occurred in 14 patients receiving pethidine (50%) but in only five receiving diclofenac sodium (17%). They included nausea, vomiting, drowsiness, and blurred vision but were transient and not severe.

Comment

Traditionally the pain of renal colic has been relieved by administration of narcotic analgesics, sometimes combined with a spasmolytic agent. This study confirms the findings of Lundstam *et al* and Naveh that diclofenac sodium 75 mg intramuscularly is effective in relieving the pain of acute renal colic.[4 5]

Because of the addictive properties of opiate drugs their storage and use cause several legal and practical problems. Substitution of an effective non-narcotic agent would alleviate these problems, both for accident and emergency departments and for general practitioners, who may be called to see patients with renal colic at home.

We conclude that diclofenac sodium 75 mg intramuscularly is more effective than pethidine 100 mg intramuscularly in the management of acute renal colic and has fewer side effects.

The diclofenac sodium (Voltarol) used in this study was kindly supplied by Geigy Pharmaceuticals.

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Diarrhoea due to Clostridium difficile associated with antibiotic treatment in patients receiving dialysis: the role of cross infection

Diarrhoea due to *Clostridium difficile* associated with treatment with antibiotics has been described among patients receiving peritoneal dialysis,[1] and cross infection is thought to be important.[2] We describe an outbreak of diarrhoea associated with *C difficile* in patients undergoing haemodialysis and continuous ambulatory peritoneal dialysis in which a "fingerprinting" technique of typing strains was used to investigate the possibility of person to person spread.

Patients, methods, and results

The table gives details of 18 patients from whom *C difficile* was isolated on stool culture. All developed diarrhoea while inpatients in the medical renal unit, Royal Infirmary, Edinburgh, between July 1983 and April 1984. *C difficile* had been isolated from only one patient with renal disease in the previous six months.

C difficile was cultured and identified as previously described[3]; strains were identified by the fingerprinting method of Poxton *et al*, using SDS-polyacrylamide gel electrophoresis of surface proteins extracted with edetic acid followed by Coomassie blue staining and an immunoblot probe using rabbit antiserum to cells of *C difficile* NCTC 11223 killed with ultraviolet light.[4] When *C difficile* was isolated patients were given oral vancomycin (500 mg every six hours) and other antibiotics were withdrawn if possible. Diarrhoea resolved in 12 patients. Four patients died during or shortly after treatment; all were severely debilitated by pre-existing medical conditions. The fingerprinting technique identified 13 different strains of *C difficile*. One strain occurred in five subjects (cases 12, 13, 14, 15, and 18) and one strain in two (cases 7 and 11); the 11 other strains occurred in only one patient each.

Comment

Cross infection with *C difficile* in hospitals has been clearly shown previously,[4] and seemed likely in this series of cases among our patients receiving dialysis; all had been inpatients in the medical renal unit, with considerable overlap in their periods of stay in hospital, and the rate of isolation of *C difficile* increased abruptly over 10 months. Standard measures to prevent spread of the organism were taken—namely, isolation when feasible, use of gown and gloves when working with patients, and careful attention to personal hygiene.

Isolation of patients was limited by lack of space and the specialised nursing that dialysis requires. The five patients from whom the same strain was isolated were probably cross infected; all were nursed in one of two adjacent cubicles, the first four within one month. The isolation of 13 different strains of *C difficile* appears, however, to exclude cross infection as the major mechanisms by which organisms were acquired during this outbreak. Among patients undergoing dialysis who have uraemia the frequent use of broad spectrum antibiotics, defective immunity, abnormal nutrition, and perhaps other changes in gut flora or mucosal defence mechanisms might combine to permit acquisition of *C difficile* or to promote its selective growth.[5] After this outbreak we tried to give as narrow a range of antibiotic treatment as possible and avoided oral antibiotics, particularly oral cephalosporins; the incidence of isolation of *C difficile* and related clinical disease returned to a low level.

We recommend early selective faecal culture for *C difficile* in any patients undergoing dialysis who have diarrhoea. Our findings suggest that cross infection with *C difficile* may occur in patients receiving dialysis, although it is not always the major mechanism of acquisition of this organism. It would be unwise to abandon standard measures against cross contamination, and it should be appreciated that patients undergoing dialysis may be particularly prone to infection with *C difficile*.

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Details of patients from whom C difficile was isolated

Case No	Age (years)	Sex	Type of dialysis	Type of infection	Antimicrobials given	Month when strain isolated	Outcome
1	61	F	CAPD	Peritonitis	None	July 1983	Resolved
2	60	F	CAPD	Peritonitis	Cephradine, flucloxacillin, tobramycin	July 1983	Died
3	48	F	Haemodialysis	None	None	July 1983	Remained well
4	15	F	CAPD	Peritonitis	Cephradine, tobramycin	August 1983	Died
5	56	F	Haemodialysis	Wound	Cefuroxime, metronidazole	August 1983	Diarrhoea continued
6	59	F	Haemodialysis	Arteriovenous fistula	Flucloxacillin, benzylpenicillin	August 1983	Resolved
7	61	M	CAPD	Peritonitis	Flucloxacillin, metronidazole, ticarcillin	August 1983	Resolved
8	69	F	CAPD	Peritonitis	Cephradine	September 1983	Resolved
9	59	M	Haemodialysis	Mastoid	Flucloxacillin, benzylpenicillin	October 1983	Resolved
10	50	M	Haemodialysis	Pericolic abscess	Cephradine, cefuroxime, metronidazole	October 1983	Resolved
11	68	F	CAPD	Peritonitis	Tobramycin	November 1983	Resolved
12	71	F	CRF	None	None	January 1984	Resolved
13	73	M	Haemodialysis (acute)	Pneumonia	Ampicillin, cefuroxime, erythromycin, metronidazole, gentamicin, benzylpenicillin	January 1984	Resolved
14	33	F	Haemodialysis	Urinary tract	Co-trimoxazole	February 1984	Resolved
15	63	F	Haemodialysis (acute)	Ischaemic bowel	Cefuroxime, metronidazole, tobramycin	February 1984	Died
16	64	F	CAPD	Peritonitis	Flucloxacillin	February 1984	Resolved
17	60	M	CAPD	Peritonitis	Flucloxacillin	March 1984	Resolved
18	66	F	Haemodialysis	Arteriovenous fistula	Cephradine, cefuroxime, tobramycin	March 1984	Died

CAPD = Continuous ambulatory peritoneal dialysis. CRF = End stage chronic renal failure.