cutaneous sphincterotomy is the treatment of choice for patients with chronic anal fissure. Both procedures are easily and safely performed in the outpatient department under local anaesthesia, thereby avoiding the costs and disruption of admission to hospital.

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SHORT REPORTS

Current practice of diagnostic lumbar puncture

About 30% of patients have a headache after lumbar puncture,¹ and many studies have examined its cause and prevention. As some of these studies have been imprecise or contradictory we surveyed current practice for diagnostic lumbar puncture.

Method and results

We contacted a junior doctor from each of the neurology departments of 15 London teaching or postgraduate institutions and 13 provincial university hospitals. These 28 doctors, most of whom worked for more than one neurologist, completed a telephone questionnaire. All 28 had performed a lumbar puncture: six estimated that they had performed fewer than 10, four from 10 to 20, three from 20 to 30, six from 30 to 40, five from 40 to 50, and four more than 50. Twenty four of the doctors had forewarned their patients of the possibility of headache. Seventeen used 18 or 19 standard wire gauge (SWG) needles, six 20 SWG needles, and five 22 SWG needles. The amount of cerebrospinal fluid removed was: 1.5 ml (one doctor), 2-3 ml (one), 3-5 ml (10), 6-10 ml (14), and 15 ml (two). To help prevent headache three doctors allowed immediate mobilisation, one advised half an hour of bedrest, one advised two hours, two advised three hours, two advised four hours, seven advised six hours, one advised eight hours, three advised 12 hours, and eight advised 24 hours. Ten doctors advised drinking plenty of water.

All the doctors agreed on bedrest and analgesia when the patient had an established headache. In addition, one doctor used the head down position and three prescribed steroids. Epidural blood patching was not used. Only seven doctors claimed to have received any instruction on policy from senior colleagues (five from registrars and two from consultants).

Comment

The cause of headache after lumbar puncture is persistent leakage of cerebrospinal fluid through the dural puncture hole made by the spinal needle. This produces cerebrospinal fluid hypotension, intracranial venous dilatation, and headache due to stimulation of pain sensitive nerve endings. There is no difference in the incidence of headache whether dural puncture is made alone or up to 25 ml fluid is removed, emphasising the magnitude of the leak.² Surprisingly, 12 doctors of the 28 said that they took only 5 ml or less cerebrospinal fluid for diagnostic purposes, a small quantity for adequate analysis. Warning patients of the possibility of headaches increases their incidence, so there appears to be no merit in doing this.

Theoretically, the smaller the needle the smaller the dural hole and fluid leak. A reduced incidence of headache has been shown with a 26 SWG needle, but this is technically difficult to use.³ It is doubtful, however, whether many patients would be spared a headache by use of a very small gauge needle, though a 20 or 22 SWG needle may be recommended after limited experience with an 18 or 19 SWG size.

Bed rest for 24 hours is still arbitrarily recommended as a means of preventing headache but does not do so,¹ for the dural hole will take longer than 24 hours to heal. Clearly, however, an established headache is relieved by increasing the cerebrospinal fluid pressure with bedrest. Early mobilisation is practised exclusively by some doctors in both inpatients and outpatients, though the use of small needles has added to their confidence in doing so. There is a theoretical possibility of subdural haematoma complicating spinal puncture, though

patients reported on had had spinal anaesthetics and some had predisposing causes.⁵ This has not been reported as a complication of routine diagnostic lumbar puncture despite many severe headaches, which suggests that it is rare. The value of increasing hydration has not been adequately assessed.

Our practice is to explain the purpose and procedure to the patient and tell outpatients that should they develop a headache they should be recumbent and be reassured that it is self limiting. We use 19-22 SWG needles and collect 10-15 ml cerebrospinal fluid, after which the patient is allowed up; should a headache develop then strict bedrest is instituted for the duration of the pain, with regular simple analgesia if required.

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Oral contraceptives and stroke: findings in a large prospective study

The suggestion that use of oral contraceptives might increase the risk of stroke was first made over 20 years ago, but controlled studies of this problem are few. We have therefore reviewed the data on stroke that have accumulated in the Oxford Family Planning Association contraceptive study.

Subjects, methods, and results

The methods used in the Oxford Family Planning Association study have been described.¹ The investigation includes over 17 000 women who, when recruited between 1968 and 1974, were all married and aged 25-39. Fifty six per cent were using oral contraceptives, 25% a diaphragm, and 19% an intrauterine device. During follow up, among other items, data are recorded about changes in contraceptive methods, hospital referrals, and deaths. Discharge summaries are obtained for inpatient spells and as much information as possible is collected about fatal illnesses.

At the end of January 1984 data accumulated during 192 000 woman years

of observation were available for analysis. In total, 46 women had been diagnosed during the follow up period by the consultant responsible for their care as suffering from a first stroke (ICD (8th revision) codes 430-438). Thirteen had suffered a subarachnoid haemorrhage, of whom four died; eight had an aneurysm or arteriovenous malformation detected by angiography or at necropsy. Two other women (who are not considered further here) suffered definite intracranial haemorrhage (a fatal intracerebral haemorrhage in one and a chronic bilateral subdural haematoma in the other). The remaining 31 women developed strokes of thrombotic, embolic, or unknown pathogenesis (hereafter referred to as "non-haemorrhagic" strokes). Of these 31, only two died, while so far as could be ascertained from the available records another nine were left with an important disability. Arteriography or brain scanning was undertaken in 17 women and an abnormality was found in nine. The diagnosis of stroke in the non-haemorrhagic group was thus usually on the basis of the clinical picture.

We analysed the data concerning subarachnoid haemorrhage and non-haemorrhagic stroke separately, taking into account the woman's age, whether or not she had been referred to hospital for management of hypertension before the stroke occurred, her smoking habits, and her use of oral contraceptives. The table summarises the results. Numbers were small, but the data suggest that hypertension and smoking were strongly related and that pill use was weakly related to the risk of subarachnoid haemorrhage.

Incidence of subarachnoid haemorrhage and "non-haemorrhagic" stroke in relation to age, history of hospital referral for hypertension, cigarette smoking, and oral contraceptive use. (Data given for each variable adjusted for effects of all other variables by indirect standardisation)

Variable	Subarachnoid haemorrhage		"Non-haemorrhagic" stroke	
	No of women	Incidence rate per 1000 woman years	No of women	Incidence rate per 1000 woman years
Age (years):				
25-34	3	0.04	6	0.07
35-39	5	0.08	9	0.15
40-44	5	0.08	9	0.24
≥45			7	0.51
Past hospital referral for hypertension:				
No	10	0.05	31	0.17
Yes	3	1.08	0	0
Cigarette smoking:				
Never smoked	4	0.04	15	0.14
Ex-smoker	2	0.09	4	0.18
Current smoker	7	0.12	12	0.19
Oral contraceptive use:				
Never	3	0.05	10	0.13
Ex-user	7	0.09	7	0.10
Current user—up to 36 months	3	0.06	6	0.41 ⁺
Current user—over 36 months			8	0.30 ⁺

*In addition there was evidence in the records that two patients with subarachnoid haemorrhage and four patients with "non-haemorrhagic" stroke had hypertension for which they had not been referred to hospital.

⁺"Non-haemorrhagic" stroke: current users v never and ex-users $\chi^2_1 = 8.7$ ($p < 0.01$).

The pattern seemed to be quite different for non-haemorrhagic stroke; there was little indication of an adverse effect of hypertension or smoking, but current oral contraceptive use emerged as a clear risk factor, duration of use appearing to be unimportant. We also examined the risk of non-haemorrhagic stroke in current oral contraceptive users in relation to the type of pill used. No strokes were observed during 9100 years of observation in women using the modern pills containing less than 50 µg oestrogen, while 13 strokes were observed during 39 400 years of observation in women using pills containing a higher dose of oestrogen. These data are encouraging but too few to be conclusive.

Comment

The findings in the Oxford Family Planning Association study agree with those of the other major studies in that they suggest that any increase in the risk of subarachnoid haemorrhage in oral contraceptive users is modest, probably not more than about 1.5-fold to 2-fold.²⁻⁵ They also suggest that the increase in the risk of non-haemorrhagic stroke is greater and is confined to current users. This conclusion supports the results of the collaborative group study² conducted in the United States from 1969 to 1971, which remains by far the largest investigation concerned with this particular question. That we have yet to observe a stroke among women using modern low oestrogen pills is encouraging but must be interpreted cautiously.

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Polymicrobial septicaemia due to *Clostridium difficile* and *Bacteroides fragilis*

We report a case of polymicrobial septicaemia due to *Clostridium difficile* and *Bacteroides fragilis*, which has not been reported previously.

Case report

A man aged 65 was admitted for urgent arteriography complaining of claudication at 100 m and a cold, painful, swollen left foot. No pulses were palpable below the right femoral artery, and the left femoral artery was not palpable at all. A gangrenous area was present on the left great toe. Arteriography showed an aortoiliac block, and a left axillofemoral bypass graft was performed. Cefuroxime 750 mg three times daily was started before the operation and continued because of suspected postoperative infection. Blood taken at this time and cultured with 0.1% glucose broth (two bottles), fastidious anaerobe broth (FAB, Lab M), and a pour plate was sterile.

On the sixth postoperative day diarrhoea developed. Pseudomembranous colitis was diagnosed presumptively, cefuroxime stopped, and oral vancomycin 125 mg four times a day started. Sigmoidoscopy was performed, a rectal biopsy specimen taken, and faecal samples analysed for the isolation of *C difficile* and its toxin. Examination for the presence of *C difficile* was by standard methods¹ using selective medium (Oxoid *C difficile* agar plus 7% defibrinated horse blood and Oxoid antibiotic supplement) and alcohol at a final concentration of 50% to select out clostridial spores. Toxin was investigated using a HEP₂ cell line (Flow Laboratories). *C difficile* and toxin were not detected. Sigmoidoscopy showed normal mucosa with liquid faeces coming from above. Histological examination of the rectal mucosa showed some congestion and extravasation of erythrocytes in the lamina propria. There was a small area without superficial epithelium, but specific features of ischaemic or pseudomembranous colitis were absent.

The diarrhoea gradually decreased until on the 10th postoperative day septicaemia developed, which was treated blindly with cefuroxime and metronidazole. A blood culture set taken before treatment with antimicrobials originally yielded *B fragilis*. On subsequent subculture three days later, however, fastidious anaerobe broth yielded a strain of *C difficile* that produced toxin. Formal identification of *C difficile* was made by volatile fatty acid profiles shown by gas-liquid chromatography in conjunction with a short set of biochemical tests.¹ Specificity of the cytopathic effect in HEP₂ cells was confirmed by neutralisation with *C sordelli* antitoxin (Wellcome Laboratories).

The patient recovered from this septicaemic episode without any additional treatment or recurrence of diarrhoea. On the 21st postoperative day a bed sore was sloughed and a subcutaneous abscess found that yielded a multi-resistant strain of *Staphylococcus aureus* sensitive only to vancomycin. Despite appropriate treatment the patient's general condition deteriorated and he died 46 days after operation.

Comment

C difficile is now firmly established as a leading cause of pseudomembranous colitis, and examination for the organism and production of toxin in faeces is a well established laboratory investigation. On this occasion Oxoid CCFA medium (cycloserine, cefoxitin, fructose,