There were no significant differences between performances on any occasion at the $p<0.05$ level of confidence (two-tailed Student's correlated t-test) except on digit span backwards, when performances on DDAVP were significantly worse than those preceding or following it. This is presumed to be an artefact.

EEGs were done before and at the end of treatment but no changes in the pattern were observed (Dr M. Schwartz). These same patients were subsequently given lysine-vasopressin intranasally four times daily for a further period of 6 weeks but again no significant changes in memory were found.

The failure to obtain benefit from DDAVP or lysine-vasopressin on memory deficit in this study differs from previous favourable reports, although it must be borne in mind that our patients had all had severe head injuries. The dosage of the drugs must also be considered. Although the amount of desmopressin and vasopressin was the maximum dosage usually given for the treatment of diabetes insipidus, it is far smaller than that used for experiments on memory in animals, and there is evidence from our laboratory that very little desmopressin and vasopressin crosses the blood-brain barrier. It seems likely that if consistent effects of neurohypophyseal hormones on brain function in man are to be obtained much higher doses (with the attendant risks of water intoxication) or active analogues with a better penetration into the brain will be required.

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PERITONEAL DRAINAGE

SIR,—Your Nov. 3 editorial on peritoneal drainage was excellent and provocative. One clinical suggestion may be helpful to your readers—namely, always place the external drains, if you must use them, into an ileostomy bag or a similar sterile adherent, transparent, collection device. This simple method minimises infection, permits accurate measurement of the drainage, and is convenient and clean for the nurses. There is no excuse for soaked and filthy drainage-stained bandages. A randomised study of drainage in patients with uncomplicated cholecystectomy at this centre provided no evidence of efficacy.[1]

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CERVICAL NEUROFIBROMA AND GENERALISED SPINAL STENOSIS IN VON RECKLINGHAUSEN DISEASE

SIR,—A woman of 28 was admitted with a history of neck pain, a feeling of pressure in the chest for 4 months, paræsthesiæ in both hands, and diminished strength in her arms and legs. For many years she had had an uncoordinated gait, which had got worse in the months before admission. In 1965 signs of neurofibromatosis were noted on her skin. She was twice operated on for subcutaneous neurofibromas. There was no family history of neurofibromatosis but her father and his brothers and sister all had severe kyphoscoliosis. On physical examination she had all the characteristic skin signs of neurofibromatosis, slight scoliosis, and pes cavus. Paræsthesiæ in the hands could not be induced by forced neck movements, and there was no neck rigidity. Sensibility was disturbed in all extremities and pyramidal dysfunction signs were present. Plain radiographs and conventional tomographs of the spine were normal except for a generalised stenosis of the neurocanal extending from C3 to the lumbar region. In the cervical region the anteroposterior diameter of the neurocanal, as read from the plain films, was narrow (C2 17 mm, C3 13 mm, C4 10 mm, C5 10 mm, C6 11 mm). On lumbar puncture there was a positive Queckenstedt during anti-flexion of the neck, and the cerebrospinal fluid (CSF) showed raised protein levels (0·75 g/l). Myelography, with air and metrizamide via a lateral puncture between C2 and C3, revealed at C0/C1/C2 an oval, well-circumscribed space-occupying lesion (35×12 mm) compressing and displacing the cord posteriorly (figs. 1 and 2). A computerised tomographic scan with metrizamide enhancement revealed an erosion in the left posterior margin of the foramen magnum related to the mass seen on myelography; there were no supratentorial masses. A neurofibroma was removed with complete recovery from the tetraplegia that had developed.

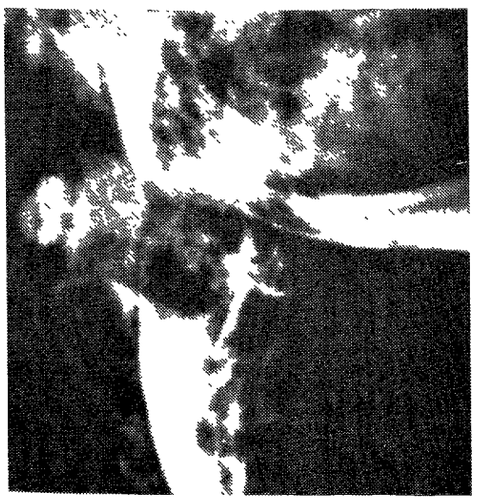

Fig. 1—Double-contrast myelography.
Visualisation of tumour at C0/C1/C2.

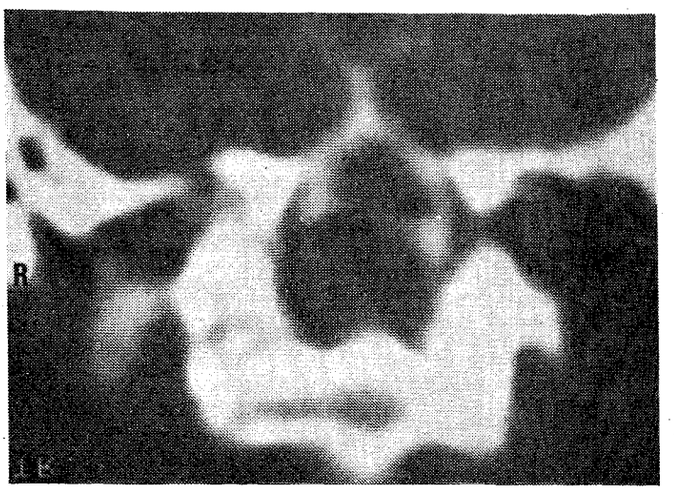

Fig. 2—Axial CT-scan through C0/C1/C2.
Cord outlined by contrast, compressed, and displaced posteriorly by tumour mass.

von Recklinghausen disease is an autosomal dominant disease consisting of multiple widespread tumours in the fully fledged form but it can often have discreet manifestations. Neurofibromatosis can also be complicated by other conditions without evidence of local tumour growth. Radicular pain syn-

1. Goldberg IM, Goldberg JP, Liechty RD, Buerk C, Eiseman B, Norton L. Cholecystectomy with and without surgical drainage. *Am J Surg* 1975; **130:** 29–32.

drome, kyphoscoliosis, spina bifida, and intrathoracic neuromas may be part of the clinical picture. Our patient had a rare combination of neurofibroma in the proximal cervical region and diffuse spinal canal stenosis. Double contrast myelography and CT scanning were needed to bring out the association of the two conditions. Both examinations determined the site of the lesion precisely, but CT scanning also demonstrate a defect in the foramen magnum caused by pressure from the neurofibroma and excluded supratentorial manifestations.

Cervical myelography with single (metrizamide) or double contrast (air and metrizamide) is easier, more comfortable for the patient and less risky when done via lateral puncture at C2/C3 than when done by the suboccipital route. This is now our usual technique for cervical myelography. CT scanning of the cervical spine with or without intrathecal metrizamide is now a major method of diagnosis of spinal and neurocanal lesions; furthermore it can, as here, show whether the lesion has extended beyond the neurocanal.

We thank Prof. H. Kemp, and Dr M. C. Kaiser for their helpful criticism, Dr R. de Graaf for his help with the myelography, Ms H. J. Vis and Miss Rowena H. Veiga-Pires for their help in the preparation of this letter and Mr I. de Groot for the illustrations.

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FATAL ASTHMA

SIR,—Dr Herxheimer (Nov. 17, p. 1084) says: "Isoprenaline cannot be an important cause [of fatal asthma] because it has been replaced worldwide by other aerosols with different properties, but asthma mortality does not seem to have declined".

ASTHMA DEATHS PER 100 000 PERSONS AGED 5–34: ENGLAND AND WALES, 1959–78

Year	Rate	Year	Rate
1959	0·66	1969	0·89
1960	0·68	1970	0·85
1961	0·89	1971	0·85
1962	1·00	1972	0·80
1963	1·43	1973	0·65
1964	1·76	1974	0·52
1965	2·01	1975	0·55
1966	2·18	1976	0·50
1967	1·77	1977	0·59
1968	1·14	1978	0·66

Source: O.P.C.S.

Among persons aged 5–34 years in England and Wales asthma mortality per 100 000 per year increased from 0·74 in 1959–61 to a peak 2·18 in 1966.[1,2] Thereafter, following a general alert and a warning on aerosol canisters, mortality quickly declined to its level in 1959–61 where it had stood, more or less, for over a hundred years (see table). Mortality has remained below its 1959–61 level since about 1973, so it is not clear why Herxheimer thinks it ought to have declined after isoprenaline was replaced. Abuse of isoprenaline, which was implicated, presumably stopped after the warning in 1967, before isoprenaline was withdrawn.

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1. Speizer FE, Doll R. A century of asthma deaths in young people. *Br Med J* 1968; iii: 245–46.
2. Inman WHW, Adelstein AM. Rise and fall of asthma mortality in England and Wales in relation to use of pressurised aerosols. *Lancet* 1969; ii: 279–85.

RISK OF ECTOPIC PREGNANCY

SIR,—Professor Vessey and his colleagues (Sept. 8, p. 501) reported on the risk of ectopic pregnancy in women using an intrauterine device (IUD) and its relation to duration of IUD use. We noted[1] that the incidence of ectopic pregnancy had increased in England and Wales since the late 1950s—particularly since 1970—and suggested that part of the increase might have been related to the increasing prevalence of IUD use. At that time information up to 1972 was available, but we now have Hospital In-patient Enquiry (HIPE)[2] data up to 1976. The accompanying table shows the estimated numbers

ESTIMATED TOTAL HOSPITAL DISCHARGES DUE TO ECTOPIC PREGNANCY BY AGE: ENGLAND AND WALES, 1966–76*

	Age								
	15–24			25–34			35–44		
Year	i	ii	iii	i	ii	iii	i	ii	iii
1966	780	22·5	2·1	1414	49·0	3·7	479	15·6	5·7
1967	801	22·8	2·1	1416	48·9	3·8	560	18·7	7·2
1968	825	23·4	2·1	1529	52·2	4·1	495	16·7	6·6
1969	777	22·1	2·0	1523	51·5	4·1	496	17·0	6·7
1970	830	23·6	2·1	1715	56·7	4·4	476	16·5	6·5
1971	1240	35·5	3·2	1683	55·5	4·3	626	21·8	8·9
1972	902	26·3	2·5	1997	63·1	5·3	548	19·4	8·6
1973	998	29·6	3·0	2039	62·8	5·6	542	19·3	9·5
1974	702	20·8	2·2	1712	52·0	4·8	647	23·1	12·2
1975	832	24·5	2·8	1816	54·3	5·2	411	14·8	8·4
1976	865	25·2	3·1	2126	62·3	6·2	331	12·0	7·2

*Column i=estimated number of hospital discharges due to ectopic pregnancy; column ii=estimated hospital discharge rate per 100 000 women; column iii=estimated ratio of ectopic pregnancies per 1000 conceptions (i.e., per 1000 therapeutic abortions plus births six months later).

of hospital discharges attributed to ectopic pregnancy (ICD 631, excluding 631·0 and 631·4) for the years 1966 to 1976, the annual age specific hospital discharge rates per 100 000 women, and the estimated proportion of fertilised ova implanting into ectopic sites, expressed as a ratio per 1000 conceptions. Data on legal abortions[3] and births[4] were used to estimate conceptions, the denominator being the total number of legal abortions performed during that year plus the total births occurring six months later. The ratios are slightly different from those reported in our earlier paper,[1] as these figures have been adjusted for the sampling fraction of HIPE data.

The most recent data reveal that, although there are irregularities, the hospital discharge rates for ectopic pregnancy have remained high since 1970. An exception may be at ages 35–44 years where rates have fallen since 1974 but more data are required before this trend can be assessed. When ectopic pregnancies are expressed as a ratio of all estimated conceptions these ratios have generally continued to increase throughout the 1970s. The all-ages ratio has increased from 3·2 per 1000 estimated conceptions in 1966 to 5·0 in 1976.

Erkkola and Liukko noted a similar trend in Turku, Finland.[5] They found that the increase in ectopic pregnancy incidence could be accounted for largely by the increasing number of ectopics associated with an in situ IUD: from 1966 to 1970,

1. Beral V. An epidemiological study of recent trends in ectopic pregnancy. *Br J Obstet Gynæcol* 1975; **82**: 775–82.
2. Office of Population Censuses and Surveys. Reports of Hospital-In-patient Enquiry. HM Stationery Office.
3. Registrar General's Statistical Review of England and Wales (1968–1976): supplement on abortion. HM Stationery Office.
4. Registrar General's Statistical Review of England and Wales (1966–1976). HM Stationery Office.
5. Erkkola R. Liukko P. Intrauterine device and ectopic pregnancy. *Contraception* 1977; **16**: 569–74.