

rates are accelerated in smokers.<sup>6</sup> In this study multiple regression analyses on a large population of patients found a positive correlation of antipyrine clearance with smoking and age. Since this study showed smoking capacity to decrease with age, however, this shortening of antipyrine half life in smokers may be partly due to age differences. Our study was designed to overcome such defects.

Compounds present in cigarette smoke include 3, 4-benzpyrene, 3-methylcholanthrene, and many related polycyclic hydrocarbons.<sup>11</sup> In-vivo animal studies and results from rat fetal liver cell cultures<sup>26</sup> have shown a direct inducing effect of these hydrocarbons on the hepatic microsomal enzyme oxidising systems. But in our study correlation of changes in antipyrine half life with the number and type of cigarettes smoked and their calculated nicotine and tar contents did not enable us to identify the component of cigarette smoke responsible. This was largely because nicotine levels and tar levels in cigarettes tend to parallel each other thus making it difficult to discriminate between these two components. Variability in the number of puffs per cigarette and the extent of cigarette smoke inhalation may also be important.

The mean increase of 23% of antipyrine half life on stopping smoking is greater than the 12% estimated by Vestal from multiple regression analyses<sup>6</sup>; he also found that age contributed 3% to interindividual variability while coffee and tea consumption contributed less than 1%. The mean decrease of antipyrine half life was found to be 40% in insecticide workers,<sup>27</sup> 22% in those with a heavy alcohol intake,<sup>4</sup> 51% after 14 days' administration of the hypnotic glutethimide,<sup>18</sup> and 34% after 140 mg phenobarbital daily for two weeks.<sup>9</sup> Since the 23% change in cigarette smoking is comparable with these other well-known inducing agents, we conclude that cigarette smoking is an important factor contributing to the large variation in rates of drug metabolism seen in man.

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## SHORT REPORTS

### Computerised axial tomography findings in patients with migrainous headaches

The demonstration of cerebral oedema by computerised axial tomography (CAT) in patients with migraine was drawn to our attention by Drs J Ambrose and J Gawler of London. The subsequent unexpected finding of a significant degree of cerebral atrophy in a migraine sufferer further prompted our interest in the CAT changes in these patients. The findings in 46 patients with migrainous headaches were therefore reviewed.

#### Patients, methods, and results

Thirty women and 16 men with a history of recurrent migrainous headaches for up to 18 years were examined on the EMI scanner, usually because of a change in headache pattern.

The findings are summarised in the table. Small areas of infarction were found in six cases. In four with fixed visual field defects these were in the medial occipital cortex and were bilateral in two; in the other two small clinically unsuspected areas of infarction were found in one temporal lobe. Two patients without known intellectual dysfunction showed a significant degree of cerebral atrophy. One was a 35-year-old woman who had had classical migraine for several years, and the other was a 36-year-old woman who had had mitral stenosis and intractable migrainous headaches for 10 months. Six other patients showed appearances suggestive of very early

cerebral atrophy. Twenty-one patients showed appearances compatible with mild cerebral oedema—namely, increased blackening on the Polaroid photograph and scattered matrix cells with  $\mu$  values of two or more less than contiguous cells. These appearances were most common in the frontal white matter, being bilateral in 15, unilateral in four, and more extensive throughout the hemisphere on the side of the headache and contralateral to the sensory aura or signs, or both, in two (see table). Seven of these patients were scanned during a migraine attack. Clinically unsuspected gliomas were found in two patients, one with common migraine and one with a classical visual aura.

#### Focal neurological symptoms and signs in patients with normal and abnormal CAT findings

CAT findings:	Normal	Infarcts	Oedema	Atrophy	Tumour
No of patients	9	6	21	8	2
No with visual or other sensory aura		4	6	2	1
No with focal signs*		4	8	1	1

\*Visual field defect or lateralised motor, sensory, or reflex changes.

#### Discussion

While the demonstration of areas of infarction in the visual cortex in patients with fixed visual field defects was not unexpected, the finding of small clinically silent areas of infarction in the temporal lobe in two patients does suggest that focal cerebral damage due to migrainous vasospasm may occur more often than is appreciated.

Two possible explanations for the appearances of oedema need to be considered. The first is that the low  $\mu$  numbers reflect an increased fluid content of brain tissue—that is, true oedema—perhaps related to regional cerebral ischaemia due to vasospasm or to arteriovenous shunting with bypassing of the microcirculation.<sup>1</sup> Alternatively, a reduction in  $\mu$  numbers and increased blackening on the Polaroid film might be due to a decreased content of blood per matrix cell if regional cerebral blood flow were decreased at the time of scanning. Further observations with both CAT and cerebral blood flow techniques in the prodromal and headache phases of migraine are required to throw further light on the significance of this finding.

The significance of the mild cerebral atrophy found in some patients is uncertain. In view of the lack of data from normal volunteers,<sup>2,3</sup> the possibility that the mild widening of the subarachnoid space and ventricular system in these patients may in fact represent one end of the spectrum of normality rather than being truly indicative of early cerebral atrophy cannot be excluded. Nevertheless, in view of the fact that a greater degree of atrophy was found in two cases, it is tempting to consider the possibility that some degree of cerebral atrophy may result from the effects of recurrent episodes of regional cerebral ischaemia in migraine.

We thank our radiological, neurological, and neurosurgical colleagues for their co-operation.

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<sup>2</sup> Gawler, J, *et al*, *Journal of Neurology, Neurosurgery and Psychiatry*, 1975, **38**, 948.

<sup>3</sup> Cala, L A, and Mastaglia, F L, *Proceedings of the 24th Annual Scientific Meeting of the Australian Association of Neurologists*, Auckland, New Zealand February 1976. In press.

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## Pilonidal sinus of the prepuce

There have been many reports of pilonidal sinus at various sites since the first description of a case in 1847 by Anderson. We report here a case of pilonidal sinus of the prepuce, which has not previously been recorded.

### Case report

A single 40-year-old bottle packer presented to the casualty department on 28 June 1974 complaining of swelling and soreness of the penis. On examination he was a fit man of White race with light brown hair. His penis was grossly swollen, particularly around the prepuce, so that it could not be retracted. On 12 July his penis was not painful, but there was some induration of the skin on the left side of the shaft. The prepuce was retracted and three groups of hairs were seen emerging from the inner surface of the prepuce, just distal to its junction with the shaft (see figure). One group of hairs came out very easily. There was no urethral discharge or lymphadenopathy.



Hairs arising from inner surface of prepuce.

He underwent circumcision on 23 September 1974, which showed multiple sinuses just proximal to the glans. The area of indurated tissue, impregnated with hair, was excised and the skin was closed with cat gut.

The macroscopic appearance of the hairs from the pilonidal sinus was different from that of the rest of the

hairs on his pubic region. They were therefore sent for electron microscopy, which showed them to be of human origin.

### Discussion

Since Anderson first described pilonidal sinus, which commonly occurs in the upper part of the natal cleft, there have been many reports of similar pathology in other sites, including the supra-pubic region, clitoris, upper eyelid, left brow, intermammary region, cervical cutaneous subarachnoid region, ear, nipple, anal canal, scalp, perineum, and mons pubis.

Pilonidal sinus of the shaft of the penis was described independently by Bervar and Manojlovic<sup>1</sup> and Yates-Bell<sup>2</sup> in 1968. Bervar and Manojlovic in fact coined the name "pilonidal sinus penis" for this condition.

Since Patey and Scarff<sup>3</sup> described pilonidal sinus of barber's hand in 1946 the opinion has swung towards the view that it has an acquired aetiology.<sup>4</sup> Ghosh and Sanyal<sup>5</sup> emphasise this theory by stating that pilonidal sinus is a peculiarity of occupation and poor personal hygiene; men are affected four times more than women, and the subjects are usually hirsutes.

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<sup>3</sup> Patey, D H, and Scarff, R W, *Lancet*, 1946, **2**, 484.

<sup>4</sup> Patey, D H, and Scarff, R W, *Lancet*, 1955, **1**, 772.

<sup>5</sup> Ghosh, D P, and Sanyal, J, *Journal of the Indian Medical Association*, 1965, **44**, 309.

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## Carbenoxolone-induced hypokalaemia simulating Guillain-Barré syndrome

We report the cases of three patients who after carbenoxolone treatment developed weakness and areflexia mimicking a profound Guillain-Barré syndrome.

### Case reports

*Case 1*—A man aged 67 had received, unknown to us, carbenoxolone 150 mg daily for eight weeks. During the 10 days before his admission he had noticed progressive weakness of his arms and legs, difficulty in swallowing, and headaches and constipation. Examination showed a symmetrical proximal weakness of the limbs with weak sternomastoids. Tendon reflexes were diminished or absent. Sensation was normal. The provisional diagnosis was Guillain-Barré syndrome and he was treated with steroids. The cerebrospinal fluid (CSF) protein was, however, normal, and the serum potassium concentration was 1.6 mmol (mEq)/l. ECG showed flat T and U waves. The steroids were discontinued and he was given potassium supplements 80 mmol (mEq) day. After 48 hours he walked normally with a serum potassium of only 2.1 mmol/l, and two weeks later his strength and serum potassium level were normal.

*Case 2*—A 54-year-old mechanic was admitted to hospital unable to stand after four weeks of progressive weakness and paraesthesiae of the limbs. Inquiries showed that he had been taking carbenoxolone 300 mg daily. His speech was slurred and he had weakness of the proximal arm and leg muscles with distal paraesthesiae and diminished reflexes. The CSF protein was raised at 0.92 g/l with normal cells. Serum potassium was 1.6 mmol/l and he was treated with oral and intravenous potassium. At 72 hours he was clinically normal. Although he was receiving 100 mmol potassium daily, it took nine days for his serum potassium to return to normal.

*Case 3*—A 50-year-old clerk was admitted to hospital after collapsing with a suspected haemorrhage from a known gastric ulcer for which he was taking carbenoxolone 150 mg daily. On admission he had weak muscles, paraesthesiae, and difficulty with speech, swallowing, and respiration. He required intubation and assisted respiration for hypoventilatory hypoxaemia. His