mg bendrofluazide daily, found fewer adverse effects in the low dose group,¹⁶ as did a long term trial of the 5 mg dose.⁸ We found that the number of adverse effects in the group given 1.25 mg was the same as that in the group given placebo, although the effects were different. Patients' symptoms are known to change when they receive antihypertensive treatment. We conclude that in low doses thiazides are one of the most effective and well tolerated treatments for arterial hypertension.

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Managing seizures in the casualty department

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Epilepsy has a prevalence of 1 in 200¹: roughly 5% of the population can expect to have a seizure at some time.² Anyone experiencing a first fit is likely to be referred as an emergency. There have been studies in the United States of how such patients are managed,³ but none have been reported from the United Kingdom. We report on the management of patients who presented over two years at a casualty department after a seizure.

Patients, methods, and results

We obtained the names of patients recorded as having had a "seizure" or a "blackout" in 1985 and 1986 from the casualty register of this hospital, a 600 bed teaching hospital serving a population of 325 000. One or more of the following criteria were required: (a) description by an independent witness; (b) appropriate history with postictal clinical signs; (c) a further witnessed seizure in the casualty department; (d) loss of consciousness in a patient known to have epilepsy. We obtained the hospital case sheet for each patient and completed a standard form from the data available during the year after the initial presentation.

From a total of 50 000 referrals in 1985 and 1986 we identified seizures in 597 patients (370 (62%) men, 227 (38%) women; mean age 41 years, range 12-88 years). Of these, 260 (44%) patients (mean age 44) had had their first fit. The other 337 patients (mean age 38) were known to have epilepsy, and the presenting event was regarded as a "recurrent" seizure.

Of the seizures, 498 (83.5%) were generalised tonic-clonic fits. Sixty seven (11.3%) of the patients pre-

Possible predisposing factors in 317 patients presenting to an emergency department after a seizure

	First seizure (n=155)	Recurrent seizure (n=162)	Total (%) (n=317)
Alcohol abuse	96	97	193 (60)
Head injury	17	31	48 (15)
Vascular accident	13	9	22 (7)
Neoplasm	8	5	13 (4)
Infection	1	9	10 (3)
Atrophy	8	2	10 (3)
Drug related	7	2	9 (3)
Metabolic abnormality	5	3	8 (3)
Arteriovenous malformation	0	4	4 (2)

sented with a complex partial seizure, and 15 (2.5%) had multiple episodes. Only 11 patients were admitted in status epilepticus. One reported a simple partial seizure, and a further four fits remained unclassified. The table lists possible predisposing factors in 317 (53%) patients.

After referral 331 (55%) patients were being treated with one or more anticonvulsant drugs (20% who had a first seizure, 80% who had recurrent seizures). Of these, 251 (76%) took one drug (carbamazepine 93, phenytoin 83, sodium valproate 45, phenobarbitone 23, primidone 7), 70 (21%) took two drugs, and 10 (3%) took three drugs. More patients with an "abnormal" electroencephalogram received anticonvulsant drugs (n=303, 62% abnormal v 39% normal, χ^2 test: $p<0.005$). This was not the case with positive results of computed tomography (n=106, 62% abnormal v 60% normal).

Of the 260 patients with a first seizure, 46 (18%) were discharged immediately and 28 given a follow up appointment. The other 214 patients were admitted to hospital: 137 (64%) spent under 48 hours in a short stay facility, and 46 (18%) were investigated in a general medical ward. Overall, of the 597 patients 371 (62%) (167 with a first seizure; 204 with recurrent seizures) subsequently attended an outpatient clinic. Most (208; 56%) were seen by a general physician (127 (76%) first seizure, 79 (39%) recurrent seizure); 93 (36%) patients with a first seizure and 133 (40%) with recurrent seizures did not attend for follow up.

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Comment

In each of two years 1.2% of all patients referred as emergencies had had a seizure. One fifth of the patients who had just experienced their first fit were treated with an anticonvulsant drug. Current practice recommends starting treatment only after two or more seizures have been witnessed by someone else.⁴ Almost one fifth of the patients with first seizures were discharged immediately, some without an outpatient appointment. Just over half spent less than 48 hours in a short stay facility; a third were subsequently lost to follow up. Three fourths of those attending as outpatients were seen in a general medical clinic.

The immediate care of patients who have a first seizure is important because over half can be expected to have a further seizure within five years.⁵ Early referral to a specialist will avoid a premature diagnosis of epilepsy and unnecessary treatment with anti-

convulsant drugs. Regular review will prepare the patient for the diagnosis and ensure that suitable treatment is started. Patients who present to our casualty department with a first fit are now reviewed urgently at our epilepsy clinic.

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Proliferative retinopathy in a woman with growth hormone deficiency and diabetes mellitus

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There have been several reports of the influence of pituitary function on diabetic retinopathy emphasising the role of growth hormone secretion and high concentrations of insulin like growth factor I in the vitreous.¹ The results of a study on growth retarded patients with growth hormone deficiency and diabetes have provided evidence of the relation between retinopathy and growth hormone as the patients were free of retinal lesions. Such observations have led to treatments to arrest or reduce pituitary function, but conflicting results and side effects have limited the use of these treatments. We report on a woman who developed severe proliferative retinopathy despite total growth hormone deficiency.

Case report

Diabetes mellitus was diagnosed in a 24 year old woman. She required a daily injection of insulin after treatment with oral insulin failed. She did not follow dietary advice, weighed 87 kg, and had a body mass index of 34 kg/m². Her first pregnancy in 1981 was complicated by haemorrhagic shock during delivery. The results of endocrinological tests showed partial pituitary deficiencies of adrenocorticotrophic hormone and thyroid stimulating hormone. Concentrations of prolactin were barely detectable in both basal and stimulated states. Severe growth hormone deficiency was confirmed between 1981 and 1989 (table) by the absence of a reaction to stimulation with thyrotrophin releasing hormone, insulin induced hypoglycaemia, glucagon-propranolol (1 mg injection of glucagon, 40 mg oral propranolol), and intravenous growth hormone releasing factor (80 µg). In 1989 the concentration of insulin like growth factor I was 1.23 U/ml (normal 0.4-2 U/ml) as measured by a specific radioimmunoassay after acid-ethanol extraction of serum.

The retina was normal in 1981, but a nucleated cortical cataract was found in the right lens and in 1985 in the left lens. Examination of the fundus of the retina showed bilateral exudative lesions. Fluorescein leaks were noted in perimacular areas during angiography. A cataract was removed from the right eye. Ocular lesions progressed rapidly thereafter. In 1988 many

haemorrhagic lesions with microaneurysms and areas of capillary non-perfusion were noted. Neo-vascularisation was detected in the nasal pole of the right eye. Despite the progression of cataract in the left lens there was cystoid macular oedema and neo-vascularisation was suspected. Laser photocoagulation was started on the right eye. After cataract surgery of the left eye proliferative lesions were also noted. Panretinal photocoagulation was therefore carried out with partial success.

*Growth hormone concentrations (ng/l) stimulated by thyrotrophin releasing hormone, insulin, growth hormone releasing factor, and glucagon-propranolol in diabetic patient with growth hormone deficiency**

	Time (min)								
	-10	0	10	15	30	45	60	120	150
<i>Thyrotrophin releasing hormone</i>									
1981	<1	<1			<1		<1	<1	
1985	1.3	1.1	1.4		1.3		1.5	1	
1989	1.7	1.2	1.8		1.5		1.3	1.1	
<i>Insulin</i>									
1982		<1			<1		<1	<1	
<i>Growth hormone releasing factor</i>									
1989	0.9	0.8		1	1.6	1.4	1.3	0.9	
<i>Glucagon-propranolol</i>									
1983	0.9	<0.9			<0.9		<0.9	1.5	1.1
1989	1.1	1.0					1.3	2.1	1.4

*Background retinopathy was noted in 1985 and proliferative lesions in 1988.

Comment

In our patient proliferative retinopathy developed despite total growth hormone deficiency and partial panhypopituitarism of pituitary origin. This is of interest as growth hormone is thought to have an important role in diabetic retinopathy and the response of growth hormone to growth hormone releasing factor and thyrotrophin releasing hormone is usually more pronounced in diabetic patients with retinopathy. Two other patients with hypopituitarism and diabetic retinopathy have been described. One developed only background retinopathy,² while the other had severe retinopathy but transient diabetes mellitus and no growth hormone deficiency.³ The concentrations of insulin like growth factor I in our patient were normal despite growth hormone deficiency. Normally, synthesis of insulin like growth factor I is stimulated by growth hormone. In the absence of growth hormone high concentrations of prolactin and insulin may be responsible for the normal concentrations of growth factor.⁴ Our patient, however, did not have raised concentrations of prolactin. Abnormal weight and